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Review

Lead, Mercury and Cadmium in Fish and Shellfish from the Indian Ocean and Red Sea (African Countries): Public Health Challenges

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Abstract: The main aim of this review was to assess the incidence of Pb, Hg and Cd in seafood from African countries on the Indian and the Red Sea coasts and the level of their monitoring and control, where the direct consumption of seafood without quality control are frequently due to the poverty in many African countries. Some seafood from African Indian and the Red Sea coasts such as mollusks and fishes have presented Cd, Pb and Hg concentrations higher than permitted limit by FAOUN/EU regulations, indicating a possible threat to public health. Thus, the operationalization of the heavy metals (HM) monitoring and control is strongly recommended since these countries have laboratories with minimal conditions for HM analysis.

Keywords: heavy metals; seafood monitoring; Indian Ocean; Red Sea; lead; mercury; cadmium; public health

1. Introduction

Seafood (fish and shellfish) is one of the main food and international economic sources in many coastal countries [1,2]. Additionally, it contains an essential nutritional composition which is crucial to human diet such as vitamins D, B12, A and E (for bones fortification [3]; nucleic acids synthesis, red blood cell and neurological function [4]; maintenance of vision and respiratory tract [5]; and for antioxidant defense [5], respectively), minerals [Selenium (for antioxidant defense and thyroid function regulation) [6], calcium (for maintenance of bones and teeth) [7], Zinc (for enzymatic catalysis for human metabolism and immune system functioning) [5], Iron (for oxygen transportation in the blood) [8], proteins (for enzymatic composition) [9], lipids [ω-3 polyunsaturated fatty acids, especially eicosapentaenoic acid and docosahexaenoic acid (for prevention the of heart disease due to low cholesterol content) [10]], iodine (for the production and functioning of the thyroid) [8] and others.

On the other hand, seafood is one of the vectors of heavy metals (HM) occurring naturally or introduced in the marine environment by different human activities, mainly, port activities, mineral resources extraction in marine and fluvial environments or untreated discharges of chemical industries or domestic wastes [11–25]. Reported HM that are environmentally relevant due to their effects on the species found in the marine environment, including seafood, which constitute a threat for public

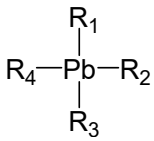
health, include: lead (Pb), mercury (Hg) and cadmium (Cd) [11–25], due to their involvement in the activation of enzymes and biochemicals, constituting a base for human organism functioning.

The presence of HM in seafood is one of the main threats to public health, and its monitoring and control has to be done rigorously in African countries of the Indian Ocean and the Red Sea, where most of the seafood from marine environments are consumed directly without quality control due to poverty [20,26,27]. Thus, the main objective of this review was to assess the incidence of Pb, Hg and Cd in seafood from African Indian and the Red Sea coasts and the level of their monitoring and control; some recommendations will also be presented. In this review, the term "HM" is used to refer to Pb, Hg and Cd and "seafood" to refer to fish and shellfish.

2. Heavy Metals and Their Effects on Humans

The occurrence of HM in seafood is one of the main threats to public health since they cause several negative effects in humans, of which the most reported induce oxidative stress [28–36] and inactivation of crucial molecules in the human organism, such as sulfhydryl groups [metallothionein (MT) [37,38] and glutathione (GSH)] [39,40] for Cd, Hg and delta-aminolevulinic acid dehydratase, delta-aminolevulinic acid synthase and ferrochelatase for Pb [41]. Thus, the control and monitoring of HM in seafood is most important to minimize associated seafood poisoning. The monitoring of HM in seafood requires active organization because there are a lot of aspects that have to be taken into consideration, from the sampling process to data interpretation. HM occurs in seafood in the form of different species distributed in a heterogeneous manner in different seafood tissues. Different monitoring programs of HM (Table 1) specify the edibles tissues (generally muscle) and seafood species to be monitored and recommend the permitted HM limit for each tissue and species (Table 1). The analytical methods that are used for HM monitoring, the different human biomarkers and the methods of their detection are described in Table 1. The most reliable HM biomarker is their presence in essential biological fluids, such as urine and blood, since the biochemical biomarkers seem similar for many HMs [20,26,27,42–44]. HM poisoning treatments consist generally of the administration of chelating agents that complex the HM and that are later excreted in urine.

Table 1. Lead compounds toxicologically relevant. TtEL—tetraethyllead, TEL—trimethyl lead, TML—trimethyl lead, X -halide (Cl).

Structure	R_1	R_2	R_3	R_4	Name
	CH_3CH_2	CH_3CH_2	CH_3CH_2	CH_3CH_2	TtEL
	X	CH_3CH_2	CH_3CH_2	CH_3CH_2	TEL
	X	CH_3	CH_3	CH_3	TML
$PbSO_4$	-	-	-	-	Lead sulphate
$Pb_3(PO_4)_2$	-	-	-	-	Lead phosphate
PbS	-	-	-	-	Lead sulfide
$PbCO_3$	-	-	-	-	Lead carbonate

2.1. Cadmium

Cadmium usually occurs in the environment in the ionic form Cd^{2+} (Cadmium oxide—CdO_2, cadmium chloride—$CdCl_2$ or cadmium sulfate—$CdSO_4$ [42]. Cd is considered the seventh most toxic nonessential heavy metal [44] and enters the environment by natural sources, such as volcanism [45], and anthropogenic sources; the latter is considered the most important. Anthropogenic activities, include smelting, mining nonferrous metals, production of nonferrous metals, iron and steel and the production and disposal of Cadmium-containing materials (electroplating, pigments, stabilizers and Ni-Cd batteries) [46], use phosphate fertilizers [47], arsenic pesticides, herbicides, fungicides, plastic stabilizers, wood preservatives and others [48]. Cd can be found in all environmental compartments

(air, soil, water and food) [46], with drinking water and food as the main human exposure routes for non-smokers and non-occupational workers [49]. Tobacco is another additional source for smokers (active and passive) [50–52]. In marine environments, Cd normally occurs in lower concentrations than in open oceans water [53] and in higher concentrations in coastal and estuarine environments due to the intensive industrial discharge, port activity and mining activity in rivers [14,19,54], distributed in the sediment, particulate matter, water and marine organisms [55]. Sediments, particulate matter and water have an important role for Cd availability for marine organism bioaccumulation, and it depends on several factors, such as temperature, salinity (ionic strength—chloride and metals ions), size of particle, organic matter content (polyphenols, amino acids, humates, proteins), sediment ion-exchange capacity and chemical Cd form, among others [55,56]. These factors have to be taken into consideration in the sampling process of the marine organism for HM assessment. The permitted limit of Cd adopted by different international organizations and the detection methods in seafood are well described in Table 1.

Cadmium Toxicity

The Cd targets from seafood in humans, some of which are listed in Table 1, include bones, placenta, brain and the central nervous system [42], liver [57], cardiovascular system [58–60], immune system [61–63], reproductive systems [64] and kidneys [65]. Acute Cd toxicity by ingestion of contaminated seafood can cause the following signs and symptoms: increased salivation, choking or vomiting, abdominal pain, vertigo, loss of consciousness, painful spasm of the anal sphincter and impairment of renal function for severe toxicity [66,67]. From chronic toxicity, the kidney is the most affected and the symptomatic signs are the presence of low molecular weight proteins (LMWP) [68], β 2-microglobulin (β2-MG) [68], *N*-acetyl-β-d-glucosaminidase (NAG) [68,69] and retinol-binding protein (RBP) [68] in the urine. These molecules are used as Cd biomarkers in humans [68,69]; other Cd biomarkers that are not validated as well as their detection methods are described in Table 1. Additionally, Cd chronic toxicity affects bones, causing fractures, severe pain, malformations [70], hypercalciuria and impaired vitamin D metabolism [71]. However, the molecular toxicity mechanism of Cd is not well understood. The gastrointestinal route is the main for Cd exposure from seafood contaminated resulting in hepatoxicity in the liver. There are two hypotheses for molecular acute toxicity of Cd: the binding to the Sulfhydryl group and the Kupffer cell activation.

From the first hypothesis, Cd hepatotoxicity occurs through the binding of Cd to sulfhydryl groups (as opposed to other groups, such as phosphate, chloride, carboxyl or amino) on critical molecules in mitochondria [72], which was verified by the protection afforded (depletion) by cysteine-rich compounds such as metallothionein (MT) [37,38] and glutathione (GSH) [39,40]. MT and GSH seem to play an important role in the hepatoprotection in acute Cd toxicity [37–39]. The inactivation of sulfhydryl groups in several molecules can result in the dysfunction of nucleic, mitochondria and endoplasmic reticulum [73], disruption of cytoskeletal organization [74,75] and disruption of the intracellular redox state [40,76] (causing oxidative stress damage to DNA [40] and lipids [40,77–80], affecting DNA synthesis [40], cell cycle regulation [73], activation of transcription factors [73] and apoptosis [81,82]).

In the second hypothesis, Cd activates macrophages resulting in the secretion of cytotoxic inflammatory mediators such as reactive oxygen species (ROS), reactive nitrogen species (RON), eicosanoids and platelet-activating factors and hydrolytic enzymes [73]. The ROS are well known to cause oxidative damage in important intracellular molecules (DNA, proteins and lipids) [40,77–80].

In Cd chronic exposure, the liver (hepatotoxicity) [83] is the first target organ, after which the kidneys (nephrotoxicity) [83,84] and other tissues follow. In all tissues, MT is the protector of Cd toxicity since it binds to free Cd as the CdMT complex [37,38,85–88]. This complex moves to the kidneys [83,89], where it is filtered to blood [90,91] and decomposed by lysosomes liberating Cd [92], which is in turn free to exert toxicity depending on the availability of MT [93–95]. The free Cd causes toxicity by inducing oxidative stress at chronic exposure in the liver and the kidneys [34]. Cd chronic

exposure causes carcinogenicity according to the International Agency for Research on Cancer [96] and the National Toxicology Program of the USA [97]. Cd is a human carcinogen of the group (Group 1) due to its involvement in the increased incidence of breast, lung, gastric, prostate, renal, endometrial, pancreas and kidney cancers [98–105].

2.2. Mercury

Mercury is the most toxic HM found in the aquatic environment. Due to its volatility, mobility and strong tendency to bioaccumulate, Hg receives special attention in the world [106]. Natural sources of Hg include primarily volcanoes, geothermal sources and topsoil enriched in mercury pertains and (from a primary source) re-emission from vegetation, land or water surfaces due to the use of land, biomass burning, meteorological conditions and gaseous mercury at air-water-soil-snow-ice exchange [107,108]. Combustion of fossil fuels (coal; stationary combustion) is the major anthropogenic source of mercury (~60% of the year 2000), followed by artisanal small scale gold mining, non-ferrous metals manufacturing, cement production, waste disposal and caustic soda production [109,110]. Hg is found in the environment in three forms, namely elemental mercury (Hg^0), inorganic mercury [mercurous—Hg_2^{2+} or Hg(I) and mercuric—Hg^{2+} or Hg(II) ions] and organic mercury (methylmercury—$CH_3Hg^+X^-$, dimethylmercury—$(CH_3)_2Hg$, ethylmercury—$CH_3CH_2Hg^+X^-$ and phenylmercury—$C_6H_5Hg^+$), with X being a halide, nitrate or sulphate [111,112]. Hg^0 has high vapor pressure and relatively low solubility in water [111,113], and because of that, it is not the predominant marine aquatic environment. Inorganic mercury species are most soluble in water [113] and have a considerable affinity with organic and inorganic species containing sulfur in their structure (most common in the environment are mercuric sulphide—HgS, mercuric oxide—HgO and mercuric chloride—$HgCl_2$ [111,114]. The most toxic and common mercury compound in the marine aquatic food chain (fishes and invertebrates, among others) is methylmercury (MHg) [115] due to its particular ability to cross the blood-brain barrier [116]. MHg is considerably accumulated as complex MeHgCys (cysteine from peptides or proteins [117] in muscle over a long time [111,112] with differences in bioavailability, tissue distribution and toxicity from methylmercury species from each marine animal [117]. The permitted limits of Hg in seafood are listed in Table 1 as well as the detection methods for Hg monitoring in seafood.

Methyl Mercury Toxicity (from Seafood)

In humans, MeHg consumed from seafood is absorbed in the gastrointestinal tract and later transported to the blood. The main Hg toxicity target organs [116] are the kidneys [35,118] and brain [118–120]. The symptoms from seafood exposure are chiefly observed in the central nervous system, and include, depending on the exposure, constriction of the visual field [116,121,122], sensory disturbances [121,122], ataxia [116,121,122], dysarthria [121,122], auditory disturbances and memory loss [116,121], tremor [116,121,122], sleep disturbance [116], headache [116], fatigue, difficulty concentrating [116], depression [116], diminished fine motor coordination [116], muscle and joint pain [116,122], gastrointestinal upset [116], hair thinning [116], heart rate disturbance [116], hypertension [116], numbness or tingling around the mouth [116], coma and death [112,123]

The molecular toxicity of MeHg seems to be well known and occurs by binding to the sulfhydryl-containing molecules (forming stable complexes) causing structural and functional modifications. Reported sulfhydryl-containing molecules include glutathione, cysteine, homocysteine, N-acetylcysteine, metallothionein and albumin, among others, which bind to MeHg in the order of 10^{15} to 10^{20} [124]. The formation of the complex MeHg-GSH [125] not only reduces the availability of GSH to act as an antioxidant defense (inducing oxidative stress) but also facilitates Hg to gain access across cell membranes through amino acid MT transporters [125–127]. Another molecule involved in Hg toxicity by binding (inhibiting) to sulfhydryl group is thioredoxin reductase, which is critical for cellular stress response, protein repair and protection against oxidative damage including lipid peroxidation [128,129].

MeHg Chronic exposure causes teratogenicity [130–132] and carcinogenicity [133], expressing itself in progeny in the form of congenital malformations and development of tumors, respectively [134]. The data of Hg carcinogenicity are controversial, with some studies that have demonstrated Hg carcinogenicity (DNA damage) and others without evidence [134–137].

2.3. Lead

Lead occurs naturally combined with two or more other elements to form lead compounds sources [138]. Mining and smelting, soldering, battery manufacturing, ammunition, metal water pipes, paint and petrol are reported as anthropogenic sources [138,139]. However, actions have been carried out in many parts of the world to reduce the harmful effects of Pb on people and animals, such as the use of unleaded fuel and bullets and shot as well as in fishing sinkers, among other taken actions [138]. Pb occurs in the environment both organic and inorganic forms with oxidation states of +2 and +4 (Table 1), with inorganic lead being more predominant as Pb(II) [138].

However, organic lead is the most toxic lead form and the reported organic lead includes TtEL [138]. One of the main current human exposure of Pb occurs via drink water and food from different sources including marine animal (seafood), among other [138]. In the marine food chain, Pb is bio concentrated and not biomagnified due to its involvement in calcium turn-over in vertebrates resulting in its accumulation in the bones than in the soft tissues [138,140]. Lead uptake by fish reaches equilibrium only after chronic exposure [138]. The most reported lead species in marine organisms include inorganic lead (PbII), trimethyl lead, X -halide (Cl) (TML) and trimethyl lead (TEL) (Table 1) [141–143]. The human biomarkers of Pb poisoning and their detection methods permitted of the control of the amount of Pb in seafood; analytical detection methods are described in Table 2.

2.4. Lead Toxicity

The main target organs/systems of Pb compounds include the digestive system [144], bones [145], reproductive system [146–150], central and peripheral nervous system [151–154], kidney [154–158] and immunologic system [159–161]. Acute Pb toxicity after ingestion of contaminated seafood usually occurs in brain and kidney, and its absorption in the gastrointestinal tract [162] is influenced by nutritional calcium and iron status and age (children adsorb more, and consequently, are more vulnerable than adults) of exposed humans and solubility and lead species, among others [138]. After absorption, Pb is transported to other organs/systems by the bloodstream, after which it accumulates in blood, soft tissues and bones [163–167]. Acute Pb poisoning symptoms in humans can be grouped in (1) nervous systems dysfunction: poor attention span, headaches, irritability, loss of memory and dullness for adult and acute encephalopathy (persistent vomiting, ataxia, seizures, papilledema, impaired consciousness and coma) for infants [168–180], (2) renal dysfunction: dysfunction of the proximal tubules, manifesting as aminoaciduria, glycosuria, phosphaturia with hypophosphatemia, increased sodium and decreased uric acid excretion, progressive interstitial fibrosis, a reduction in the glomerular filtration rate and azothemia [168,169,181]. Hypertension is another sign of Pb poisoning [169,182,183].

Molecular toxicity of Pb seems to be well understood and occurs at a concentration as low as 5 µg/dl of blood. Pb inhibits three crucial enzymes for humans' body functionating, namely delta-aminolevulinic acid dehydratase, delta-aminolevulinic acid synthase and ferrochelatase [41]. Delta-aminolevulinic acid is involved in the synthesis of porphobilinogen (PBG), which plays an important role on the biosynthesis hemeproteins (hemoglobin, myoglobin, cytochromes, guanylate cyclase and nitric oxide synthase) [184], while ferrochelatase catalyzes the incorporation of iron into the porphyrin ring [169,185]. The inhibition of these heme synthesis enzymes can cause a reduction in the circulating levels of hemoglobin and the inhibition of cytochrome P 450-dependent phase I metabolism [185].

The animal model tests report that at chronic exposure, Pb causes neurotoxicity, neurodevelopmental toxicity [186,187], cardiotoxicity [188,189], nephrotoxicity [190], genotoxicity

(increase in DNA strand breaks) [187] and carcinogenicity (inorganic lead as probably carcinogenic to humans—Group 2A) in the kidneys [187].

3. Heavy Metals Poisoning Treatment

The symptomatic treatment of HM poisoning is very limited and consists generally of HM remotion using metal chelating agents. Ethylenediamine tetra-acetic acid via intravenous administration [191] or hemodialysis to remove Cd by urinary excretion or dialysate in case of severe toxicity (renal dysfunction) [192] or oral administration of dimercaptosuccinic acid [193] is the most commonly used for Cd poisoning. Diethyldithiocarbam ate [194], 2,3-dimercaptosuccinic acid [195] and EDTA calcium disodium [196] have also been reported as chelating agents for Cd poisoning treatment.

MeHg poisoning treatment involves the use of chelating agents containing sulfhydryl group in their structure by different methods, such as hemodialysis administration of penicillamine [197] and sodium 2,3-dimercapto-1-propanesulfonate(DMPS) [198] in case of renal failure. DMPS is also administrated orally [198], intravenously and nasogastrically [199], and often in combination with vitamin E [197]. 2,3-dimercaptopropanol via intramuscular administration has also been reported for Hg poisoning treatment [200]. Lead poisoning treatment is performed using chelating agents accompanied with nutritional interventions of iron and calcium supplementation [181], since iron is involved in the functioning of ferrochelatase (important enzyme for biosynthesis of hemeproteins), which is impaired by Pb, while calcium was tuned over by Pb; hence, there is a need to be reset during the treatment. The reported use of chelating agents for Pb poisoning treatment include intramuscular administration of dimercaprol with an oil solution due to its weak solubility in water (dimercaprol avoid encephalopathy) [168,201], Calcium Disodium EDTA, which is administrated after dimercaprol to avoid the increasing lead concentration in the central nervous system [201], and oral administration of 2,3-meso-dimercaptosuccinic acid [201].

Table 2. Cadmium, mercury, lead and arsenic biomarkers: RBP—retinol-binding protein; LMWP—low molecular weight protein; β2-MG—β2-microglobulin; NAG—N-acetyl-β-d-glucosaminidase (NAG); α -GST—alpha- S-transferase; VDBP—vitamin D-binding protein; α1-MG—alpha 1-microglobulin; 8-OH-G—8-hydroxyguanine; KIM-1—kidney injury molecule-1; cDNA—deoxyribonucleic acid; MT—metallothionein; GASTA1—anti-glutathione S-transferase alpha; Pb-U, -P, -B—lead concentration in urine, plasma, and blood; ALAD—δ-aminolevulinic acid dehydratase; ALA-P,-B,-U - δ-aminolevulinic acid in plasma, blood, and urine; CP-B. -P—coproporphyrin in blood and plasma; P5′N—pyrimidine nucleotidase; NADS - nicotinamide adenine dinucleotide synthase; ZP—zinc protoporphyrin and detection methods:CVAAS—cold vapor atomic absorption spectrometry; GFASS—graphite furnace AAS; ICP—MS—inductively coupled plasma mass spectrometry; FAAAS—flame AAS; ICP-AES—ICP atomic emission spectrometry; CM—colorimetric method; RGSS—radioactivity gamma scintillation spectrometer; EIA—enzyme immune assay; EU—European Union; USEPA—United States Environmental Protection Agency; FAOUN—Food and Agriculture Organization of the United Nations, tHg—total mercury; MeHg—methylmercury; MeHgMe—dimethyl mercury; ANHMRC—Australian National Health and Medical Research Council; ANZFSC—Australia New Zealand Food Standards Code; EtHg—Ethyl mercury; iHg—inorganic mercury; HPLC-UVD and - ECD—high performance liquid chromatograph with ultra- violet detection or with electrochemical detection; DPSAV—differential pulse stripping anodic voltammetry; Cd-HRA—cadmium-hemoglobin radioassay; CF—cytofluorimetry; CD—cluster of differentiation; IC-PICVGAFS—ion chromatography using photo-induced chemical vapor generation atomic fluorescence spectrometric; TDA-AAS-thermal decomposition amalgamation AAS; SM—spectroscopy methods; SPME–GC–FAPES—solid phase microextraction in conjunction with tandem gas chromatography–furnace atomization plasma emission spectrometry; IMGC-AE—isothermal multicapillary gas chromatography with atomic emission detection; CGC-pyro-AFS—capillary gas chromatography coupled to an atomic fluorescence detector via pyrolysis; GC-AES—gas chromatography-atomic emission spectrometry; ESR—electron spin resonance; FP—fluorescent probes; ELIA—enzyme-linked immunosorbent assay; CGCAAS—glass capillary gas chromatography AAS; PPA—profiling protoarray; XRFS—X-ray fluorescence spectroscopy; ETAAS—electrothermal AAS; AE—acid extraction, DD—detergent dilution, NSE—neutral solvent extraction, HF—hematofluorimetry; RCMP—radioactive cytidine 5′-monophosphate; PM—photometric method; LC–(PO)–HG–AFS—liquid chromatography–photo-oxidation–hydride generation–atomic fluorescence spectrometry; HG-AAS—hydride generation–atomic absortion spectrometry; EC-ICP-MS—exchange chromatography ICP-MS; CEHPLC-ICP-MS—cation-exchange HPLC-ICP-MS; CA—Comet assay.

Metal	Biomarkers in Humans	Biomarkers Detection	Limit in Seafood, $mgKg^{-1}$		Detection Method in Seafood		
			Tissue	Value	Technique	LOD, μgKg^{-1}	LOQ, μgKg^{-1}
Cd	LMWP (V) [68], β2-MG (V) [68], NAG (V) [68,69] and RBP(V) [68], α-GST [202], VDBP [203], α1-MG [204], 8-OH-G [36] and KIM-1 [205] in urine	CA [36,68,69,202–205] and EIA [36,68,69,202–205]	Fish muscle	0.1 (FAOUN) (a) [206], (EU) (b) [207]; **0.15** (c) (EU) [207]; **0.25** (d) (EU) [207]; **0.05** (e) (EU) [207]; **0.3** (f) (EU) [207] and **1** (SA) [208]	ICP-MS [209–211]	1-30 [209–211]	4-10.1 [209,210]
	Oxidative stress biomarker in blood [32]	HPLC -UVD and –ECD [32]	Crustacean muscle and abdomen (excluding brown meat of crab)	**0.5** (FAOUN) [206] and (EU) [207]	ICP-AES [212]		
	Genomic/proteomic (*hsp90*) responses in bronchiolar lavage materials cells [213] and Cd-induction of MT in human tumor cell line HepG2 [214]	cDNA microarray analysis [213] and Cd-HRA [214]			FAAS [14,19,54,215]		
			Bivalve mollusks and Cephalopods (without viscera)	**1** (FAOUN) [206] and (EU) [207]	DPSAV [216]		
	Autophagy in human CD34+ hematopoietic progenitor cells [63]	CF [63]	Seaweed or dried bivalve mollusks	**3** (EU) [207]	ETAAS [217]	0.045 [217]	0.015 [217]

Table 2. *Cont.*

Metal	Biomarkers in Humans	Biomarkers Detection	Limit in Seafood, mgKg^{-1}		Detection Method in Seafood		
			Tissue	Value	Technique	LOD, µgKg^{-1}	LOQ, µgKg^{-1}
Hg	Porphyrins [218]; Hg level(exposure to Hg^0) [219]; GSH depletion [35]; MT induction [220]; Renal dysfunction (presence of NAG, RBP, α1,β1-MG, Kim-1) [221]	HPLC-FD [218]; CV-AAS [222]; SM [35]; RGSS [220]	Fish muscle	**1** (FAOUN) [206], (SA) [208] and (UE) [(g)] [207] **0.3** (China) [223]; 0.5 (UE) [207]; **0.4** (Japan) [224]	CVAAS (tHg) [215,224–228]	0.2-4600 (tHg) [215,224–228]	2-9.9 (tHg) [215,224–228]
	GSH depletion [229]; MT induction [230]	SM [229]; RGSS [230]	Crustaceans	**0.5**[(h)] (EU) [207]	LC-ICP-MS (MHg and EHg) [231]; HPLC-ICP-MS (iHg and MHg) [232]; ICP-MS (tHg) [209,210,231]; IC-PICVGAFS (iHg and MHg) [233]; ICP-AES (tHg) [212]; LC-UV-CV- AFS (iHg, tHg and MHg) [234]	5.30 [209]; 2 [210]; 0.25 (tHg) [231]; 0.1 (MHg) [231]; 0.2 (EHg) [231]; 0.02 (iHg) [232]; 0.03 (MHg) [232]; 80,000 (MHg MHg) [233]; 100,000 (iHg) [233]; 0.4 (iHg) [234]; 1 (tHg) [234]; 0.3 (MHg) [234]	16.2 [209], 6 [210]; 1.2 (iHg) [234]; 3 (tHg) [234]; 1 (MHg) [234]
	Hg level in hair [219,235] (exposure to MHg) [235]; GSH depletion [119]; Oxidative stress [236];	CVAFS [235]; SM [119]	Fishery products	**0.5** (EU) [207]	AAS(tHg) [54]; TDA-AAS(tHg) [237]		3 (tHg) [237]
	Hg level in blood (exposure to MeHg) [123]; GASTA1 increasing [238] in serum	ICP-MS [33]; PPA [238]			CGC-pyro-AFS (iHg and MMHg) [239]; GC-AES (MHg and iHg) [240]; IMGC-AE [241]; CGC-AAS [242]; SPME-GC-FAPES (iHg and MHg) [243]	1 pg (iHg) [239]; 2 pg (MMHg) [239]; 0.003 (MHg) [240]; 0.0125 (iHg) [240]; 20 (iHg) [241]; 80 (MHg) [241]; 0.1 ng [242]; 1.5 (MHg) [243]; 0.7 (iHg) [243]	
Pb	Pb-B, Pb-U, Pb-P MPb-U and MPb-P [244–249]	GFAAS [244] and ICP-MS [245–249]	Fish muscle	**0.3** (EU) [207]; 0.4 (SA) and FAOUN [206]	GFASS [225,226]	1.8 [225]; 2 [226]	6.2 [225]
	ALAD inhibition in the blood [250–252]	PM [250]	Bivalve mollusks	**1.5** (EU) [207] and 1 (FAOUN) [206]	XRFS		
	CP-U and CP-B increasing activity [253–255]	HPLC-MS [253–255]	Crustaceans	**0.5** (EU) [(g)] [207] and FAOUN [206]	ICP-MS [209–211]; ICP-AES [212]	2.70 [209]; 4 [210]; 10 – 240 [211]; 18 [256]	8.1 [209]; 12 [210]; 62 [256]
	ZP alteration in blood [257–266]	AE [257,258], DD [259,260], NSE [261,262], HF [261,263,264] and HPLC [265,266]	Cephalopods (without viscera)	**1** (EU) [207] and SA [208]	FAAS [19,215,267]; AAS [14]	1 [267];	
	ALA-U, ALA-B and ALA-P alteration [268]	HPLC-UVD [268]			ETAAS [217]	0.0732 [217]	0.0244 [217]
	P5′N alteration in blood [269–272]	CM [269], RCMP [270] and HPLC [271,272]			DPSAV [216]		
	NADS alteration in blood [273,274]	HPLC [274] and EIA [273]					

(a) Species: *Dicologoglossa mneata* (Wedge sale), *Anguilla anguilla* (Eel), *Trachurus trachurus* (Horse Mackerel or Scad), *Mugil labrosus labrosus* (grey mullet), *DipZodus vulgaris* (Common two-banded seabream), *Sardina pilchardus* (European pilchard or sardine), *Engraulis encrasicholus* (European anchovy), *Luuarus imperialis* (Louvar or Luvar). **(b)** *Scomber* species (mackerel), T*hunnus* species, *Katsuwonus pelamis* and *Euthynnus* species (tuna), *Sicyopterus lagocephalus* (bichique). **(c)** *Auxis* species (bullet tuna). **(d)** *Engraulis* species (anchovy), *Xiplhas* gladius (swordfish) *Sardina pilchardus* (sardine). **(e)** For species not mentioned in (a), (b), (c) and (d). **(f)** *Xiphias gladius* (swordfish). **(g)** Species: *Lophius* species (anglerfish), *Anarhichas lupus* (Atlantic catfish), *Sarda sarda* (bonito), *Anguilla* species (eel), *Hoplostethus* species (emperor, orange roughy and rosy soldierfish), *Coryphaenoides rupestris* (grenadier), *Hippoglossus hippoglossus* (halibut), *Makaira* species (marlin), *Lepidorhombus* species (megrim), *Mullus* species (mullet), Esox lucius (pike), *Orcynopsis unicolor* (plain bonito), *Tricopterus minutes* (poor cod), *Centroscymnus coelolepis* (Portuguese dogfish), *Raja* species (rays), *Sebastes marinus, S. mentella, S. viviparus* (redfish), *Istiophorus platypterus* (sailfish), *Lepidopus caudatus* and *Aphanopus carbo* (scabbard fish), *Pagellus* species (seabream and pandora), shark (all species), *Lepidocybium flavobrunneum* and *Ruvettus pretiosus*, *Gempylus serpens* (snake mackerel or butterfish), *Acipenser* species (sturgeon), *Xiphias gladius* (swordfish) and *Thunnus* species, *Euthynnus* species, *Katsuwonus pelamis* (tuna). **(h)** excluding the brown meat of crab and head and thorax meat of lobster and similar large crustaceans (*Nephropidae* and *Palinuridae*).

4. Concentration of Cadmium, Mercury and Lead in Seafood from African Countries of the Indian Ocean and the Red Sea

The marine environment of the Indian Ocean and the Red Sea has many sources of HM due to human activities such as port activity, mining (metals, gas, and petroleum) activity, discharges of untreated domestic residues and contaminated river water with HM where precious metals and rocks are explored and produced (Table 3) [11–25]. HM can get into humans through diet, which can suppose a risk for health. The HM that constitute a great threat to public health includes Pb, Hg and Cd, which are potent toxicants to humans. The bioaccumulation of these metals in tissues of seafood leads to intoxication causing decreased fertility, cellular and tissue damage, cell death and dysfunction of a variety of organs and death [11–25]. Figure 1 describes the sources of HM in the African countries bordering the Indian Ocean and the Red Sea. Many countries adopted WHO legislation to control and monitor the presence of HM in seafood. There are no data of seafood poisoning caused by HM; this lack does not necessarily implicate the absence of poisoning incidents. Rather, the lack of an operational monitoring program, training staff for recognizing of HM poisoning symptoms and equipped laboratory could partially explain the lack of HM seafood poisoning data.

Table 3. Main sources of HM and countries with a monitoring program in the African countries bordering the Indian Ocean and the Red Sea.

Country	Local	HM Source
Egypt	Gulf of Suez [14] and Red Sea [14,275–281]	Drilling fluids in oil fields, tourism and port activities [14,275–281]
Sudan	Sawaki and Port-Sudan [282]	Port activities [282]
Eritrea	Assed and Massawa Ports	Port activities
Djibouti	Gulf of Tadjourah [283]	Port activities [283]
Somalia	Barawa, Berbera, Bosaso, Eyl, El-Mana, Eyl, Hobyo, Kismaayo, Las Khorey, Mareeg, Merca, Mogadishu, Qandala, Ras Kamboni and Zeila	Port activities
Kenya	Watamu, Mombassa and Kilindini harbors	Port and tourism activities [284,285]
Tanzania	Mwamba Nyama, Mwamba Wamba, Tanga, Zanzibar, Mbegani and the Dar es Salaam harbors	Port activities [286] and storm water runoff from the city roads and garages as a result of the previous usage of leaded petrol [23]
Madagascar	Antsiranana, Mahajanga, Morondava, Toamasina, Tolagnaroand Toliara Ports	Port activities
Mozambique	Maputo and Nacala Ports	Port activities
	Rovuma River Basin	Gas production activities
South Africa	Durban, Elisabeth, Richards Bay, Mossel Bay, Ngqura, East London and Cape Town harbors	Port activities [287]

The incidence of HM is provided in Table 4. In general, all African countries of the Indian Ocean and the Red Sea have enough laboratory conditions to determine the concentration of HM in seafood in the universities and fishery research centers. The concentration of HM in seafood ranges from 0.004 to 49.65 [14,288] and 0.01 to 18.5 $mgKg^{-1}$ [2,284] in the Indian Ocean and the Red Sea, respectively, with most data being concentrated in the Red Sea. Along this geographic area, the main HM sources include ports and gas/petroleum production activities, with the Red Sea having more ports on both coasts, African and Saudi Arabian. Some seafood from the African Red Sea coas,t such as mollusks *Ruditapes decussatus*, *Venerupis pullastra* [281], *Psammobia depressa, Brachidontes sp., Nerita waigiensis, Patella miniata, Lepidochiton cinereus, Morula squamosa, Tridacna squamosa, Morula squamosal* [289] *Barbatus barbatus* and *Patella caerulea* [19], have presented Cd and Pb concentrations higher than the permitted limit by FAOUN/EU regulations (>1 and 1.5 $mgKg^{-1}$, respectively, for fish and shellfish) [206]. *Rhizoprionodon acutus* presented the highest content of Hg in its muscle; this species is one of the recommended to be used for HM monitoring in the marine environment [288]. Fish species, namely *Epinephelus* sp., *Sardinella* sp., *Synodus* sp., *Nemipterus japonicus*, *Carangoides*

bajad, *Lutjanus bohar* and *Gerres oyena* [290], have also presented higher Cd and Pb contents in their muscles (>0.3 and 0.4 $mgKg^{-1}$) than permitted by FAOUN/EU regulations [206]. On the Indian Ocean coast, certain species of fish, namely *Sardinella gibbosan*, *Leiognathus equula*, *Upeneus* spp., *Lutjanus fulviflamma*, *Sphraena jello muscle*, *Monodactylus argenteus*, *Secutor insidiator*, *Mugil mugil*, *Carangoides gymnostethus*, *Geres oyena*, *Crenidens crenidens*, *Chorinemus tol*, *Leptoscarus vaigiensis*, *Spilotichthys pictus*, *Siganus sutor*, *Lenthrinus* sp., *Therapon jarbu* and *Anadara antiquata*, bioaccumulated higher Pb contents in their muscles, with the maximum concentration found 18.5 $mgKg^{-1}$ [284,291]. Other fish species, namely *Xiphias gladius*, *Thunnu,s albacares*, *Katsuwonus pelamis*, *Coryphaena hippurus*, *Xiphias gladius*, *Thunnus albacares* and *Coryphaena hippurus*, bioaccumulated the highest Hg content, ranging from 0.56 to 3.97 $mgKg^{-1}$ [292], higher than the legal limit (0.5 $mgKg^{-1}$) [206]. Invertebrates species such as *Octopus cyanea* and *Saccostrea cucullate* from the Tanzanian coast presented Pb contents below the permitted limit [23,286]. The highest Hg, Pb and Cd content were found in bivalve mollusk *Mytilus galloprovincialis* from the South African coast [287]. The High HM content found in different species for human consumption evidence a threat to public health.

Figure 1. Locals (sites) of main sources of heavy metal in the African coasts of the Indian Ocean and the Red Sea. In these locales, there is port activity, tourism and petroleum activities.

Table 4. Concentration of cadmium, mercury and lead in seafood from African countries bordering the Indian Ocean and the Red Sea.

Country	Local	Date	Seafood	Heavy Metal	Concentration, $mgKg^{-1}$	Detection	Reference
Egypt	Hourghada, Suez and Ismaila	1986–1989	Unknown fish species	Cd	1.24	AAS	[293]
				Pb	0.82		
	Gulf of Suez	2004	*Tridacna Maxima* shell (bivalve mollusk)	Cd	35.83	AAS	[14]
				Pb	1.79		
	Hurghada harbor			Cd	44.65		
				Pb	1.76		
	El-Esh			Cd	39.45		
				Pb	1.65		
	Suez	2014	*Erugosquilla massavensis* muscle (crustacean)	Hg	4.91	AAS	[294]
				Pb	11.49		
				Cd	0.57		
	Ismailia			Hg	4.00		
				Pb	9.70		
				Cd	0.44		
	Lake Timsah	2006–2007	*Ruditapes decussatus* soft tissue (mollusk)	Cd	5.00	FAAS	[281]
				Pb	16.50		
			Venerupis pullastra soft tissue (mollusk)	Cd	9.30		
				Pb	19.00		
	Gulf of Suez	2009	*Patella nigrolineata* soft tissue (bivalve mollusk)	Pb	16.89	FAAS	[289]
				Cd	0.46		
				Hg	0.11		
			Ostrea crestata soft tissue (bivalve mollusk)	Pb	1.03		
				Cd	0.31		
				Hg	0.03		
			Tridacna squamosa soft tissue (bivalve mollusk)	Pb	0.35		
				Cd	0.36		
				Hg	0.01		
			Nerita waigiensis soft tissue (bivalve mollusk)	Pb	14.48		
				Cd	0.37		
				Hg	0.03		
			Lepidochiton cinereus soft tissue (bivalve mollusk)	Pb	1.50		
				Cd	0.35		
				Hg	0.02		

Table 4. *Cont.*

Country	Local	Date	Seafood	Heavy Metal	Concentration, $mgKg^{-1}$	Detection	Reference
			Morula squamosa soft tissue (bivalve mollusk)	Pb	1.16		
				Cd	0.47		
				Hg	0.11		
			Brachidontes sp. soft tissue (bivalve mollusk)	Pb	2.35		
				Cd	0.72		
				Hg	0.11		
			Dinocardum robustum vanhyningi soft tissue (bivalve mollusk)	Pb	0.88		
				Cd	0.04		
				Hg	0.01		
			Brachidontes sp. soft tissue (bivalve mollusk)	Pb	0.75		
				Cd	0.05		
				Hg	0.05		
			Nassarius clathratus soft tissue (bivalve mollusk)	Pb	1.03		
				Cd	0.10		
				Hg	0.02		
			Patella testudinária soft tissue (bivalve mollusk)	Pb	1.05		
				Cd	0.06		
				Hg	0.01		
	Hurghada Sheraton		*Nerita waigiensis* soft tissue (bivalve mollusk)	Pb	1.55		
				Cd	0.11		
				Hg	0.01		
	Safaga		*Lepidochiton cinereus* soft tissue (bivalve mollusk)	Pb	1.62		
				Cd	0.19		
				Hg	0.03		
			Morula squamosa soft tissue (bivalve mollusk)	Pb	11.26		
				Cd	0.36		
				Hg	0.15		
			Tridacna squamosa soft tissue (bivalve mollusk)	Pb	3.19		
				Cd	0.29		
				Hg	0.21		
	Quseir		*Psammobia depressa* soft tissue (bivalve mollusk)	Pb	1.03		
				Cd	1.72		
				Hg	0		

Table 4. *Cont.*

Country	Local	Date	Seafood	Heavy Metal	Concentration, $mgKg^{-1}$	Detection	Reference
	Marsa Alam		*Nerita peloronta* soft tissue (bivalve mollusk)	Pb	14.3		
				Cd	0.58		
				Hg	0.01		
			Nerita peloronta soft tissue (bivalve mollusk)	Pb	0.24		
				Cd	0.12		
				Hg	0.4		
			Morula squamosa soft tissue (bivalve mollusk)	Pb	1.84		
				Cd	0.63		
				Hg	0.15		
			Lepidochiton cinereus soft tissue (bivalve mollusk)	Pb	1.87		
				Cd	0.78		
				Hg	0.1		
	Shalatin		*Nerita undata* soft tissue (bivalve mollusk)	Pb	0.25		
				Cd	0.05		
				Hg	0		
	Gulf of Suez	2003	*Barbatus barbatus* soft tissue (mollusk)	Pb	126.74	FAAS	[19]
				Cd	3.02		
			Patella caerulea soft tissue (mollusk)	Pb	147.55		
				Cd	3.74		
	Hurghada		*Barbatus barbatus* soft tissue (mollusk)	Pb	9.8		
				Cd	1.06		
			Patella caerulea soft tissue (mollusk)	Pb	16.78		
				Cd	1.33		
	Gulf of Suez	2010–2011	*Epinephelus* sp. muscle (fish)	Pb	0.45	AAS	[290]
				Cd	0.20		
			Synodus sp. Muscle (fish)	Pb	0.28		
				Cd	0.04		
			Nemipterus japonicus muscle (fish)	Pb	0.28		
				Cd	0.04		
			Sardinella sp. muscle (fish)	Pb	0.50		
				Cd	0.38		
			Trachurus mediterraneus muscle (fish)	Pb	0.40		
				Cd	0.20		
			Lethrinus sp. muscle (fish)	Pb	0.25		
				Cd	0.23		

Table 4. *Cont.*

Country	Local	Date	Seafood	Heavy Metal	Concentration, mgKg^{-1}	Detection	Reference
	Shalateen		*Epinephelus* sp. muscle (fish)	Pb	0.88		
				Cd	0.12		
			Caranx sp. muscle (fish)	Pb	0.28		
				Cd	0.07		
			Scarus gibbus muscle (fish)	Pb	0.21		
				Cd	0.03		
			Synodus sp. muscle (fish)	Pb	0.51		
				Cd	0.07		
			Nemipterus japonicus muscle (fish)	Pb	0.46		
				Cd	0.06		
			Carangoides bajad muscle (fish)	Pb	0.52		
				Cd	0.08		
			Lutjanus bohar muscle (fish)	Pb	0.51		
				Cd	0.08		
			Thunnus albacares muscle (fish)	Pb	0.32		
				Cd	0.06		
			Gerres oyena muscle (fish)	Pb	0.41		
				Cd	0.11		
			Sargocentron spiniferum muscle (fish)	Pb	0.28		
				Cd	0.06		
	Hurghada		*Epinephelus* sp. muscle (fish)	Pb	0.45		
				Cd	0.05		
			Caranx sp. muscle (fish)	Pb	0.25		
				Cd	0.05		
			Scarus gibbus muscle (fish)	Pb	0.24		
				Cd	0.03		
			Sardinella sp. muscle (fish)	Pb	0.25		
				Cd	0.07		
			Siganus rivulatus muscle (fish)	Pb	0.04		
				Cd	0.05		

Table 4. *Cont.*

Country	Local	Date	Seafood	Heavy Metal	Concentration, $mgKg^{-1}$	Detection	Reference
Djibouti	Gulf of Tadjoura	2016–2018	*Sphyrna lewini* (shark)	Cd	0.48	ICP - MS	[288]
				Hg	12.51		
				Pb	0.08		
			Rhizoprionodon acutus (shark)	Cd	14.50		
				Hg	0.68		
				Pb	0.16		
Kenya	Mombasa	1997–1998	*Sardinella gibbosa* n muscle (fish)	Cd	4.8	FAAS	[284]
				Pb	3.4		
			Leiognathus equula muscle (fish)	Cd	18.5		
				Pb	0.76		
			Upeneus spp muscle (fish)	Cd	6.0		
				Pb	0.4		
			Lutjanus fulviflamma muscle (fish)	Cd	2.0		
				Pb	0.2		
			Sphraena jello muscle (fish)	Cd	9.8		
				Pb	0.09		
			Monodactylus argenteus muscle (fish)	Cd	1.6		
				Pb	0.2		
			Secutor insidiator muscle (fish)	Cd	1.2		
				Pb	0.2		
			Mugil múgil muscle (fish)	Cd	5.0		
				Pb	0.1		
			Carangoides gymnostethus muscle (fish)	Cd	1.6		
				Pb	0.2		
			Geres oyena muscle (fish)	Cd	1.0		
				Pb	nd		
			Crenidens crenidens muscle (fish)	Cd	1.4		
				Pb	0.2		
			Chorinemus tol muscle (fish)	Cd	2.2		
				Pb	0.4		
			Leptoscarus vaigiensis muscle (fish)	Cd	5.8		
				Pb	0.2		
			Spilotichthys pictus muscle (fish)	Cd	4.8		
				Pb	0.15		
			Siganus sutor muscle (fish)	Cd	6.0		
				Pb	0.2		

Table 4. *Cont.*

Country	Local	Date	Seafood	Heavy Metal	Concentration, $mgKg^{-1}$	Detection	Reference
			Lenthrinus sp. muscle (fish)	Cd	1.0		
				Pb	0.2		
			Therapon jarbu muscle (fish)	Cd	0.6		
				Pb	0.04		
Tanzania	Dar es Salaam	2015	*Siganus sutor* muscle (fish)	Cd	0.04	HR-ICP-MS	[295]
				Pb	0.045		
			Lethrinus harak muscle (fish)	Cd	0.14		
				Pb	0.144		
			Rastrelliger Kanagurta muscle (fish)	Cd	0.13		
				Pb	0.067		
	Dar es Salaam	2013	*Octopus cyanea* muscle (octopus)	Pb	7.22	ICP-AES	[286]
	Tanga			Pb	3.24		
	Dar es Salaam	2007–2008	*Saccostrea Cucullate* soft tissue (oyster)	Cd	1.00	ICP-OES	[23]
				Pb	2.00		
				Hg	0.081	SAHgA	
	Dar es Salaam	2016	*Rastrellieger kanagurta* muscle (fish)	Pb	0.03	FAAS	[2]
				Cd	0.06		
			Lutjanus fulvus muscle (fish)	Pb	0.14		
				Cd	0.16		
			Fenneropenaeus indicus muscle (fish)	Pb	0.06		
				Cd	0.01		
	Zanzibar	2011	*Anadara antiquata* muscle (fish)	Pb	3.5	FAAS	[291]
Mozambique Channel	East of Madagascar	2004	*Xiphias gladius* muscle (fish)	Cd	0.60	ICP-AES	[292]
				Pb	0.01		
				Hg	3.97	AHgA	
			Thunnus albacares muscle (fish)	Cd	0.26	ICP-AES	
				Pb	0.02		
				Hg	1.15	AHgA	
			Katsuwonus pelamis muscle (fish)	Cd	0.61	ICP-AES	
				Pb	0.07		
				Hg	0.67	AHgA	
			Coryphaena hippurus muscle (fish)	Cd	0.13	ICP-AES	
				Pb	0.06		

Table 4. *Cont.*

				Hg	0.21	AHgA	
	Northern part		*Xiphias gladius* muscle (fish)	Cd	1.04	ICP-AES	
				Pb	0.12		
				Hg	1.61	AHgA	
			Thunnus albacares muscle (fish)	Cd	0.25	ICP-AES	
				Pb	0.09		
				Hg	0.56	AHgA	
			Coryphaena hippurus muscle (fish)	Cd	0.12	ICP-AES	
				Pb	0.14		
				Hg	0.98	AHgA	
South Africa	Cape Town	2011	*Mytilus galloprovincialis* (bivalve mollusk)	Cd	1.99	ICP - MS	[287]
				Pb	7.30		
				Hg	4.93		

SAHgA—semi-automatic mercury analyzer, AHgA—Advanced Mercury Analyzer, ICP—Inductively Coupled Plasm, MS—Mass Spectroscopy, AES—Atomic Emission Spectrometry, HR—High Resolution, FAAS—Flame Atomic Absorption Spectrometry.

5. Final Considerations and Recommendations

Among several marine organism species, shellfish have been reported to be more sensitive and can be used as marine bioindicators of HM pollution [296]. The habitat change assessment, a description of the natural characteristics of aquatic systems [296], the ability of bioaccumulation of HM and very low locomotion [297–299] and seafood in human diet [1,300], among other aspects, are indicated as advantages of using shellfishes as bioindicators of HM pollution in the marine environment. On the African Red Sea coast, several species of mollusks [19,281,289] and fishes [288,290] have presented Cd, Pb and Hg concentrations higher than permitted limit by FAOUN/EU regulations [206]. Moreover, on the Indian Ocean coast species of fishes and invertebrates were found with a higher content of Pb, Hg and Cd [23,284,286,287,291,292]. These data indicate the need for serious monitoring programs and control of HM in seafood. Many studies of the incidence of heavy metals in the Indian Ocean and the Red Sea involve fishes species (*Cyprinus carpio*, *Carassius auratus*, *Corydoras paleatus*, *Oreochromis mossambicus*, *Dicentrarchus labrax*, *Oreochromis niloticus*, *Oncorynchus mykiss*, *Anabas testudineus*, *Channa punctatus*, *Anguilla Anguilla*, *Sparus aurata*, *Leuciscus idus*, *Pimephales promelas*, *Clarias batrachus*, *Clarias gariepinus*, *Acipenser sinensis*) [301–317]. These studies cannot give realistic information about HM pollution because fishes are migratory species, and consequently, are not suitable as HM bioindicators. The reported potential HM bioindicator species include invertebrate mollusks *Barbatus barbatus* and *Patella caerulea* [19]. There are currently no registered human poisoning cases related to Cd, Hg and Pb in the African countries bordering the Indian Ocean and the Red Sea. The lack of such data may be partly related to the lack of trained health staff to recognize signs/symptoms of heavy metal poisoning in humans. The aquaculture system of most consumed fish and shellfish species is strongly recommended, because it can avoid or minimize the consumption of contaminated seafood.

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