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FOCUSED REVIEW

The Toxicological Effects of Mercury Exposure in Marine Fish

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Abstract
Since the Minamata incident in Japan, the public have become increasingly aware of the negative health effects caused by mercury pollution in the ocean. Consequently, there has been significant interest in the health of humans eating fish exposed to mercury (Hg). However, the toxicity of mercury to the marine fish themselves has received far less attention. In this review, we summarize mercury accumulation in marine fish and the toxicological effects of mercury exposure. Results showed that the bioaccumulation of mercury in marine fish was highly variable, and its concentration was affected by the specific physiological and ecological characteristics of different fish species. Mercury exposure can produce teratogenic, neurotoxic effects, and reproductive toxicity. These effects can then cause harm to cells, tissues, proteins and genes, and ultimately, the survival, growth, and behavior of marine fish. Future studies should afford more attention to the toxicological effect of mercury exposure upon marine fish.

Keywords Mercury · Marine fish · Toxicity

Introduction

Mercury in water is mainly in the form of elemental mercury, divalent mercury and methylmercury. Elemental mercury (Hg^0) is rarely found in seawater due to its high volatility and low bioavailability, so divalent inorganic mercury (Hg^+ and Hg^{2+}) and organic mercury (such as methyl, ethyl, and phenyl) are the major mercury species (Selin 2009; Gonzalez-Raymat et al. 2017). Mercury in the upper oceans has tripled compared to pre-anthropogenic conditions (Lamborg et al. 2014). The bioaccumulation of organic and divalent inorganic mercury in marine fish can directly and indirectly endanger population health; even low concentrations of mercury exposure are associated with developmental retardation and learning disabilities in fetuses, infants and children (Budtz-Jorgensen et al. 2002). The exposure of marine fish to mercury has not only been linked to population health, but could also act as a useful marker to reflect the ecological effects of mercury in the ocean.

The principal way by which humans are exposed to mercury is via ingestion, including the rice ingestion in the special Hg mining area (Li et al. 2012), and the consumption of seafood such as fish contaminated with mercury (Zaza et al. 2015). However, eating fish is not completely unprofitable. Nutrients in fish, such as docose hexaenoie acid (DHA), an n-3 long-chain polyunsaturated fatty acid, are important for the growth and development of the fetal brain, and fish are highly recommended for the prevention of cardiovascular disease (Liu et al. 2018; Anual et al. 2018). In marine fish, exposure to mercury can lead to reduced liver function and metabolism, altered behavior, impaired reproduction, deformity, damage to the gills and olfaction organs, and mortality (O'Bryhim et al. 2017; Huang et al. 2011).

Research studies have measured mercury levels in fish, assessed the health risks to humans of eating fish and have provided guidance for tolerable weekly intake (Anual et al.

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2018; Jeevanaraj et al. 2016; Liu et al. 2018). However, far less is known about the abnormal development of marine fish due to mercury exposure. In fact, the reproductive toxicity and teratogenicity effects of mercury to embryos may also lead to a significant reduction in fishery production and quality. This article reviews the associated bioaccumulation characteristics of marine fishes, focusing particularly on the toxic effects of mercury on marine fish in laboratory studies.

Bioaccumulation of mercury in marine fish

Bacteria and phytoplankton are the primary entry-points of mercury into the food chain. Mercury can be actively absorbed and methylated by bacteria, some of which can be released back into the ocean (Schaefer et al. 2014). Phytoplankton can also actively accumulate methylmercury within their cells (Pickhardt and Fisher 2007); this cytoplasmic methylmercury can be more easily transferred to the trophic chain than inorganic mercury (Harding et al. 2018); this represents a key step in the bioaccumulation and biomagnification of mercury. In the ocean, trophic transfer is the main pathway for marine organisms to take in and accumulate mercury.

The methylation of inorganic mercury in the water column may represent an important source of methylmercury in pelagic marine food webs (Lehnherr et al. 2011), and studies have shown that the total mercury content of predatory pelagic fish, and their prey, is similar to that of dissolved organic mercury in seawater, which increases with water column depth, thus indicating that mercury content in marine predators was affected by their foraging depth (Choy et al. 2009). Horizontal habitats (i.e., distance from the coast), and trophic levels, also result in differences in mercury accumulation between species; moreover, coastal fish are more susceptible to contamination than offshore fish (Le Croizier et al. 2019).

Once mercury enters the body, it binds strongly with protein sulfhydryl groups (SH). As a consequence, the elimination and excretion of the bioaccumulated mercury is very slow and takes a long period of time (Chouvelon 2018; Maulvault et al. 2016). Body length and weight are the main factors responsible for intra-species differences in fish with respect to mercury concentrations. The bioaccumulation of mercury in marine fish is highly variable and its concentration is affected by species-specific differences in physiological and ecological characteristics (Table 1). Furthermore, mercury concentrations in fish tissues are known to be positively correlated with the iron, total organic carbon, ammonia, and nitrogen content within the water column but are negatively correlated with alkalinity, dissolved oxygen and pH (Glover et al. 2010). Consequently, there is a wide range of factors responsible for the affected by observed differences in the bioaccumulation of mercury in marine fish. All of these factors may exert influence over the toxicity of mercury in fish.

The highest total mercury levels in fish tissues were previously shown to be in the muscle, followed by liver or kidney. Fins showed the lowest levels, and there was no significant difference in the total mercury concentrations between different types of muscle (O'Bryhim et al. 2017; Harley et al. 2015). However, a study of mercury concentrations in southern California found that the level of mercury in the liver of the blue marlin was higher than that in the muscle (Vega-Sanchez et al. 2017). Therefore, it is still necessary to carefully examine the assumptions related to organ mercury concentrations and focus on methylmercury concentrations.

Toxic effects of mercury in marine fish

Even at low concentrations, low mercury concentrations in the ocean can still lead to genetic, biochemical, physiological, morphological and behavioral changes in fish (Huang et al. 2011). Mercury toxicity depends on a variety of factors, including speciation, bioavailability and the absorption and the transformation of mercury. These factors are also known to differ in different species, and the effects of mercury toxicity on different individuals, species and stages of life can vary greatly (Morcillo et al. 2016c).

Mercury toxicity in embryonic and larval stages of marine fish

When considering the entire life cycle, it is clear that the embryonic and larval stages are the most sensitive periods for mercury exposure in fish. Frequent mercury exposure can affect the development of several organs (such as abnormal eye development and cardiac malformation) and metabolic processes (Huang et al. 2011). Mercury toxicity can also affect the entire embryonic development process, can activate energy-consuming detoxification processes (Sfakianakis et al. 2015) and consumes a significant amount of energy, resulting in a reduction in energy that could otherwise be used for growth. Mercury toxicity can also cause slow development, morphological abnormality, dysfunction and even death (Sfakianakis et al. 2015). Sub-chronic toxicity tests on red sea bream (*Pagrus major*) indicated that mercury concentrations exceeding 20 µg/L can reduce hatching success, increase mortality and induce teratogenicity in both embryo and larvae (Huang et al. 2011). In a previous paper, the larvae of large yellow croaker (*Pseudosciaena crocea*) were exposed to 0–4 µg/L of methylmercury for 30 days; results showed that the expression of TCTP, GST3, Hsp70 and Hsp27 mRNA (related to immune response) were all up-regulated in the presence of methylmercury and that these changes occurred in a dose-dependent manner (Wu et al. 2018). Furthermore, mercury poisoning can also indirectly

Table 1 Total mercury (THg) in fish muscle tissues from different coastal waters

Mercury in muscle of fish (μg/g)	References (locations)
0.4 ± 0.3 (*Raja clavata*, n = &)	Chouvelon et al. (2018) (Mediterranean Sea, DW)
0.3 ± 0.2 (*L. ocellata*, n = 14)	
10.85 ± 7.62 (*Scyliorhinus canicula*, n = 9)	
7.81 ± 4.02 (*Galeus melastomus*, n = 15)	
7.83 ± 2.97 (*Helicolenus dactylopterus*, n = 13)	
4.96 ± 2.66 (*Lepidorhombus boscii*, n = 5)	
2.63 ± 1.03 (*Phycis blennoides*, n = 15)	
1.53 ± 0.45 (*Micromesistius poutassou*, n = 12)	
3.3 ± 2.1 (*Mustelus canis*, n = 79)	Taylor et al. (2014) (New England, DW)
1.1 ± 0.7 (*Squalus acanthias*, n = 80)	
0.9–1.2 (*Genyonemus lineatus*, n = &)	Meador et al. (1998) (Canada, DW) (Washington, DW)
0.8–1.2 (*Platichthys stellatus*, n = &)	
2.2 (*Paralabrax nebulifer*, n = &)	
0.8–0.9 (*Pleuronectes vetulus*, n = &)	
0.11–0.73 (*Caulolatilus microps*, n = 62)	Matulik et al. (2017) (Florida Bay, WW)
0.03–1.81 (*Serioli dumerili*, n = 32)	
1.55–4.99 (*Carchahinus acronotus*, n = 8)	
1.87–7.18 (*Carcharhinus leucas*, n = 7)	
1.24–5.96 (*Carcharhinus limbatus*, n = 23)	
0.84–2.20 (*Negprion brevirostris*, n = 8)	
0.072–0.123 (*Sparus aurata*, n = 15)	Djinovic-Stojanovic et al. (2015) (Serbia, WW)
0.099–0.164 (*Scorpaena scrofa*, n = 23)	
0.078–0.122 (*Dicentrarchus labrax*, n = 14)	
0.06–0.50 (*Mycteroperca microlepis*, n = 53)	Sinkus et al. (2016) (Atlantic, WW)
0.03–0.89 (*Lutjanus campechanus*, n = 53)	
0.43 ± 0.02 (*Thunnus spp*, n = 5)	Cabañero et al. (2004) (Spanish,WW)
0.42 ± 0.01 (*Aphanopus carbo*, n = 5)	
0.072 ± 0.002 (*Sardina pilchardus*, n = 5)	

&, there is no detail on sampling number

affect the survival skills (i.e. foraging and predator evasion) of fish. If fish are exposed to mercury, their feeding can be affected or even stopped; this may lead to a deficiency of essential nutrients for larval development (Liu et al. 2016).

Teratogenic effects of mercury on marine fish

Malformations due to mercury exposure exert a devastating effect on fish, since they not only affect the survival mode, growth rate and external morphology of the fish, but can also affect the most important behavioral characteristics in the life of fish, such as hunting, avoiding predators and long-distance migration (Mora-Zamorano et al. 2017, 2016; Webber and Haines 2003). Common malformations include those in the spine, bladder, head region and fins. The most common malformations observed in fish occur in the spine. The most obvious spinal deformities include lordosis (*sacral doris*), kyphosis (*sacral curvature*) and scoliosis (*lateral curvature*) (Morcillo et al. 2016b; Sfakianakis et al. 2015). Deformities (particularly skeletal deformities) can interfere with the ability of fish to interact with the environment and reduces their chances of survival (Huang et al. 2011). Poor nutrition can also result in bone deformities (Liu et al. 2016). Moreover, mercury metal ions can cause deformity by altering the integrity of the notochord during development or by inducing bone growth via neuromuscular effects (Avyle et al. 1989).

Hepatotoxicity of mercury on marine fish

Mercury exposure may cause hepatic histopathological damage (including vacuolization, parenchyma disorganization and pyknotic nucleus), and cause other syndromes. A previous study of the liver in marine medaka (*Oryzias melastigma*) exposed to different concentrations of mercuric chloride showed that mercury exposure increased the accumulation of mercury in the liver and subsequently impaired the ultrastructure of the liver (Wang et al. 2013; Chen et al. 2017). Quantitative proteomic analysis further suggested that Hg-induced hepatotoxicity may involve oxidative stress,

cytoskeletal damage, immunotoxicity and changes in energy metabolism, indicating that mitochondria may be the primary target for mercury attack in cells (Wang et al. 2013; Chen et al. 2017). Olsvik et al. (2011) exposed Atlantic cod (*Gadus morhua*) larvae to mercury-rich sediments for 5 weeks and found that calreticulin, HSP70 and heme oxygenase mRNA were significantly up-regulated in fish gills. In the liver, calreticulin, heme oxygenase, transferrin and WAP65 were also upregulated but glutathione peroxidase 4B and zona pellucida 3 were significantly down-regulated.

Neurotoxic effects of mercury on marine fish

Although Hg can produce a variety of toxicity effects in organisms, the neurotoxicity of Hg has always been a significant concern. Mercury can accumulate in the brain and thereafter can cause significant damage. A previous study of marine medaka (*O. melastigma*) exposed to mercury chloride over long periods showed that inorganic mercury may cause neurotoxicity by inducing oxidative stress, cytoskeletal assembly dysfunction and metabolic disorders (Wang et al. 2015; Barboza et al. 2018a). Proteomic analysis of brain tissue from the Atlantic cod (*Gadus morhua*) also showed that after exposure to methylmercury, the levels of 71 proteins changed by 20% or more. These proteins were associated with major known molecular targets and mechanisms of methylmercury-induced neurotoxicity in mammals, such as mitochondrial dysfunction, oxidative stress (Berg et al. 2010). Changes of the proteome in the brain of juvenile beluga (*Huso huso*) further confirmed that methylmercury induces toxicity through oxidative stress and apoptosis, which suggested that chronic methylmercury exposure can cause an important metabolic defect in the brain (Keyvanshokooh et al. 2009). Mercury may also induce morphological changes in the brain, such as changes in the total number and volume of neurons and glial cells in specific areas of the brain, accompanied by changes in swimming behavior; it can also cause neurological damage over the long-term (Pereira et al. 2016). Studies have also shown that neurobehavioral deficits induced by mercury exposure can be passed down steadily to the next generation in white seabream (*Diplodus sargus*); these deficits were conveyed via the sperm and persisted in each generation. This startling discovery showed that such neurological diseases can persist in a fish population for generations, even after the source of the pollution has been removed (Senger et al. 2010).

Reproductive toxicity of mercury to marine fish

Mercury exposure is not entirely risk-free for adult fish. Mercury can accumulate in the gonads of fish and may affect the reproductive system and inhibit the growth and development of fish gonads (Liao et al. 2006). The hypothalamic–pituitary–gonadal (HPG) axis regulates reproductive activity by secreting different hormones and plays a key role during the normal development of the fish reproductive system (Dang et al. 2015). Mercury exposure can interfere with the expression of genes related to the HPG axis and alter sex hormone levels, potentially affecting fish reproduction. In addition, exposure to inorganic mercury is known to induce oxidative stress, leading to histological damage in the gonads of fish such as the thickening of tubule walls (Zhang et al. 2016). After exposure to mercury, testosterone levels in male testes are significantly reduced and spermatogenic degeneration and necrosis can also be detected, including Sertoli cell hypertrophy and interstitial inflammation (Liao et al. 2006; Zhang et al. 2016). Moreover, the connections between follicular cells and oocytes can be disrupted, which may lead to a delay in ovarian development (Zhang et al. 2016).

Other toxicological effect of mercury exposure to marine fish

In fish, the branchialepithelium is the site of gas exchange, acid-base balance, nitrogen waste excretion and ion regulation. Gills can interact directly and continuously with the surrounding environment, and represents the main target organ for environmental pollutants (Evans 1987; Brunelli et al. 2010; Wendelaar Bonga 1997; Barboza et al. 2018b). Inorganic mercury and methylmercury can exert toxic effects on gills by interfering with multiple metabolic pathways. Taurine is a sulfur-containing amino acid mainly involved in the maintenance of cellular homeostasis. Cell swelling caused by hypo-osmotic stress is followed by a regulatory volume decrease (RVD) that occurs via cellular extrusion of ions, mainly taurine (Fugelli and Thoroed 1990). Therefore, wild gold gray mullet (*Liza aurata*) exposure to mercury-containing sediments indicated that the accumulation of mercury in the gills of fish caused obstacles in the process of ionic regulation and osmotic regulation, due to the decrease of taurine and glycerophosphocholine, as well as the increase of creatine levels (Cappello et al. 2016).

In terms of histopathology, mercury can also affect the morphology of the gill epithelium, causing morphological and pathological changes in the sputum (such as cellular necrosis, shortening of the secondary lamellae, hyperplasia of the gill filaments and severe edema), thus resulting in impaired gill function. Rainbow trout (*Oncorhynchus mykiss*) exposed to 50 µg/L mercuric chloride or methylmercury showed a variety of morphological changes, including a reduction in the height of the lamellar cell ridges; other effects included vacuolated epithelial cells and the degeneration of chloride cells (Olson et al. 1973).

In addition to the toxicological effects of mercury exposure upon vital tissues in marine fish, as described above,

there are also other effects which should be considered, for example the effects of such toxicity upon cellular proteins. For example, in a previous study, fibroblast SAF-1 cells, harvested from the marine teleost fish (*Sparus aurata* L.) were exposed to mercury for 24 h; this resulted in the increased production of reactive oxygen species and apoptotic cell death. The corresponding gene expression profile indicated that the potential mechanisms underlying the observed changes included induction of the metallothionein protective system, cellular oxidative stress and apoptosis (Morcillo et al. 2016a). In another study, erythrocytes from the gilthead seabream (*Sparus aurata* L.) and European sea bass (*D. labrax* L.) were exposed to methylmercury for 24 h in vitro; results showed that the gene expression profiles of heat shock protein 70 and 90, superoxide dismutase, metallothionein, glutathione reductase, anti-apoptosis Bcl2-related X protein, catalase, peroxidase, and calpain1, all changed. This showed that oxidative mechanisms were activated in order to protect cells but ultimately the cells could not cope and suffered apoptotic cell death (Morcillo et al. 2016b).

Conclusion

Even at low exposure levels, mercury can not only affect the genetic mutation, tissues, physiology, morphology and behavior of marine fish, but can also affect their survival, growth and development. Generally, the toxicity of pollutant exposure could be tested using an appropriate fish model, such as the zebrafish or medaka which both have a completely sequenced genome, a transparent body and for which laboratory conditions can be easily controlled. However, there are highly variable and significant differences among the teleosts due to a wide range of physiological and ecological characteristics. Carrying out more toxicity tests on marine fish species would help us to further elucidate the toxicological responses of marine fish to mercury.

Mercury accumulation in marine products has become an important global issue that must be addressed. Tracking the source of this mercury and understanding how mercury circulates in the marine ecosystem have become key research targets. It is clear that different publications have reported different orders of mercury pollution in fish tissues. Further experiments, which test the mercury concentration of specific organs, are now required to investigate the reasons for such differences. Moreover, the toxicity effect of mercury upon fish is also related to gender (Zhang et al. 2016); the reasons for this are also unknown and require further study.

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