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REVIEW ARTICLE

Cadmium: Toxic Effects and Physiological Impairments in Fishes

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Abstract

Cadmium (Cd) is widely distributed in aquatic environments and is an extremely toxic metal commonly found in industrial settings as a key component in the production of batteries, pigments, coatings and electroplating. Fish play an integral role in the aquatic ecosystem food web, and any effects that change the population structure of fish may also alter community and food web dynamics. The consumption of fish is recommended because it is a good source of omega-3 fatty acids, which have been associated with health benefits due to its cardio-protective effects. However, the content of heavy metals such as Cd discovered in some fish makes it difficult to establish clearly the role of fish consumption on a healthy diet. Therefore the present mini-review accounts for the recent evidence of the effect of this toxic metal on fish physiological processes.

1. Introduction

Heavy metals in the aquatic environment pose a serious threat to biodiversity and human health. Among heavy metals dispersed in the environment, Lead, Mercury, Arsenic and Cadmium (Cd) are ubiquitous and have severely harmful effects (Jarup, 2003). These nonessential metals gain entry into cells by simple diffusion or through membrane carriers and ion channels (Bridges and Zalups, 2005). Upon entering cells, heavy metals exert multiple adverse effects through interfering with functions of essential metals, generating reactive oxygen species (Ercal et al., 2001; Ahamed and Siddiqui, 2007), disrupting physiological signal transduction (Thevenod, 2009; Druwe and Vaillancourt, 2010), affecting gene expression (Gonzalez et al., 2010), inducing damages to DNA, membranes and proteins, and inhibiting DNA repair (Bertin and Averbeck, 2006; Long et al., 2011). Cd (atomic number 48 and relative atomic mass 112.40) is a toxic ubiquitous environmental pollutant. It is released into aquatic environments from industrial sources involved in mining, ore refining and plating processes as well as from natural sources such as rocks and soils (Choi et al., 2007). Cd that enters aquatic environments accumulates within the bodies of aquatic organisms. The growth, osmoregulation and reproduction of fish are affected by exposure to this metal (Kim et al., 2004). Cd obstructs numerous reproductive processes in fish such as sexual maturation, spermatogenesis, fertilization success and development of the embryonic and post embryonic stages (Jezierska and Witeska, 2001; Dietrich et al., 2011).

Cd toxicity is known since the mid-nineteenth century from workers occupationally exposed to the metal. Its severe effects to human health have been widely studied and documented ever since, from nephrotoxicity to carcinogenicity and reproductive disorders (Nordberg, 2009; Nawrot et al., 2010; Costa et al., 2012). Although its industrial use has been enduring many restrictions, this metal is still widely employed, for instance, in batteries, dyes, metal alloys and even some phosphate fertilizers, to which is added its release from the combustion of fossil fuels and from metal extraction of Cd-containing ores. Thus, Cd reaches the marine environment chiefly through continental runoffs plus urban and industrial effluents. Estuaries and other confined coastal waterbodies are particular areas of concern due to their ability to trap, store and speciate Cd, as well as other inorganic and organic toxicants.

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Cd is normally included in biomonitoring studies with fish and other aquatic organisms. Due to its hazardous nature, Cd is classified as a priority substance by the Directive 2008/105/CE of the European parliament and of the council of the European union, known as the Water Framework Directive, which sets its highest admissible concentration at 1.5 µg L^{-1} (applicable to non-inland surface waters) (Costa et al., 2012).

It has been demonstrated that the feed is the principal source of contamination by metals such as Cd that tends to accumulate in tissues, which in the end are ingested by consumers (Maule et al., 2007; Fernandes et al., 2009; Creti et al., 2010). Its accumulation in the organisms depends on the concentration, route of absorption, environmental conditions and other intrinsic factors (Karakoç and Dinçer, 2003; Bowen et al., 2006; Jezierska and Witeska, 2006; Guinot et al., 2012). Moreover, consumption of seafood is also one of the dominant routes for human exposed to Cd (Jarup, 2003; ATSDR, 2008; Chen et al., 2010; Oyoo-Okoth et al., 2010). Seafood can accumulate Cd via waterborne and dietborne exposure pathways, posing a potential human health risk (Buchwalter et al., 2008; Metian et al., 2008; Ju et al., 2012). Coastal communities depending on fishery resources are more vulnerable to contamination since they tend to consume more fish than the general population.

2. Physiological impairments in fishes

2.1. Effect of Cd on the Hematopoietic Activity

Cd is well known to induce hematotoxicity in fish, often resulting in anemia and immunosuppression (Seong-Gil et al., 2004; Ates et al., 2008; Witeska et al., 2009, 2010). Sometimes the values of hematological parameters of intoxicated fish fluctuate, and their changes are not always directly related to metal concentrations and time of exposure or time post-exposure (Shah and Altindag, 2005; Witeska et al., 2010). These fluctuations may result from translocation of Cd within the organism, and its toxic action on various functions at different time. Cd probably affects not only circulating blood cells, but also newly developing ones in hematopoietic tissues. Very scarce data concerning hematopoietic effects of heavy metals in fish (Ghosh et al., 2007; Som et al., 2009) and mammals (Van Den Heuvel et al., 2001; Celik et al., 2005, 2009) indicate that they are cytotoxic to precursor cells, and various cell lineages show different sensitivity to metal toxicity. Hematological effects of heavy metal intoxication of fish were extensively studied, but very little data concerning metal-induced alterations in hematopoietic system are available.

Kondera and Witeska (2012) reported that Cd disturbed hematopoiesis in carp but on the other hand indicated a considerable compensatory potential of carp hematopoietic system. The pattern of changes after short-term exposures (a rapid increase in cell proliferation rate and early blast frequency, accompanied by an increase in apoptotic rate) and during long-term treatments (gradual increase in the values of these parameters during the exposures) was different but the final effect-reduction in cell turnover rate was very similar. The increase in apoptotic rate was higher when compared to acceleration of precursor cell proliferation. No anemia was observed in peripheral blood or a significant reduction in leukocyte count, and the most pronounced effect of metal exposures was significantly reduced frequency of peripheral phagocytes (neutrophils and monocytes), accompanied by reduction in their metabolic activity (Kondera and Witeska, 2012). According to Kondera and Witeska (2012), these data indicate that Cd may affect hematological and immune status of fish organism by disturbing the process of hematopoiesis. Hematopoietic precursor cells are sensitive to intoxication and heavy metals enhance the rate of their apoptotic destruction. On the other hand, hematopoietic system of carp shows high homeostatic potential and tends to compensate cell loss by activation of mitotic divisions. Anemia and immune-suppression often observed in fish contaminated with Cd may result from toxic effect of metal on the hematopoietic system. Additionally, cellular parameters of hematopoietic tissue: frequency of blast cells and the rate of proliferation and apoptosis are sensitive indicators of sublethal intoxication.

2.2. Effect of Cd on Development

In recent years, fish embryos have gained interest in risk assessment procedures because of their high sensitivity to pollutants and their ecological relevance. The embryonic period is an essential developmental growth phase of a fish, and normal embryo development is directly related to the reproductive function of the fish. Most studies on fish embryotoxicity were focused on the general effects of toxicant on developmental parameters and have ignored the possible confounding influence of environmental stressors on embryo development during pregnancy in females. Quantitative information on the bioavailability of Cd to early developmental stages of fish, such as eggs/embryos is scarce.

Previous studies revealed that the sensitivity of fish embryos and larvae to chemicals is far greater than that of adults (Zhang et al., 2012). Furthermore, while the long biological half-life of Cd allows it to

readily bioaccumulate in exposed organisms (Luckenbach et al., 2001), very little is known about the uptake and elimination of Cd during embryonic and larval stages of fish development. The Cd content of fish eggs increased with time and Cd-concentration of the surrounding medium. Indeed, when Cd is accumulated in fish eggs, it may be detected in the different components at different concentrations however most of the Cd is associated with the chorion. For example, in rainbow trout (*Oncorhynchus mykiss*) 98 % of total Cd was retained by chorion (Beattie and Pascoe, 1978), 94.6% in medaka (*Oryzias latipes*) (Michibata, 1981), 93% Chinook salmon (*Oncorhynchus tshawytscha*) (Hammock et al., 2003) and 61 % in zebrafish (*Danio rerio*) (Burnison et al., 2006). This data suggest that the difference is likely due to the presence of the chorion (a membrane envelope surrounding the egg) which is capable of acting as a barrier to Cd transfer to the developing embryo (Rombough and Garside, 1982). Indeed, Hallare et al. (2005) supported the evidence of the protective effect of the chorion and approved that hatched larvae are more susceptible to Cd than unhatched embryos.

A high percentage of deformed larvae has been also observed following Cd exposure. Several categories of abnormalities have been reported including blastodermal lesions, yolk-sac and heart oedemata, haemorrhages, damaged blood vessels, hypopigmentation, cranio-facial deformities including head and eye hypoplasia, cardiac abnormalities, deformed yolk sac and vertebral deformities including C-shaped larvae, shortened body and altered axial curvature (Cheng et al., 2000, 2001; Lugowska and Witeska, 2004; Sassi et al., 2010; Kessabi et al., 2009; Messaoudi et al., 2009a; Messaoudi et al., 2009b). It had been reported that Cd exposure induces a reduction of myosin heavy chain production in the trunk which is correlated to a disorganization of myotomes in the somites and results in altered spinal curvature (Cheng et al., 2000; Chow and Cheng, 2003).

As previously mentioned, fish cardiovascular system is also a target of heavy metal toxicity. Indeed, various cardiovascular pathologies were described following exposure to heavy metals including haemorrhages, hypertension, oedemata (a result of an alteration of vascular permeability), megalocardias, circulatory collapse, tubular heart, heart rate alteration, red blood cells accumulation, atrium/ventricle morphology alteration, abnormal heart looping and aberrant vascular patterning (Gonzalez-Doncel et al., 2003; Cao et al., 2009; Li et al., 2009). Although these pathologies are frequently reported in the literature, little is known about the exact mechanisms involved in such cardiovascular dysfunctions. Several works previously showed an alteration of the cardiac functions following Cd exposure. It also had been shown that cardiac function and hemodynamic conditions strongly influence cardiac morphogenesis and vascular endothelium modeling (Glickman and Yelon, 2002; Sidi and Rosa, 2004; Barjhoux et al., 2012). Moreover, cardiac morphologic alterations described by Hove et al. (2003) following artificial perturbations of blood flow are very similar to those reported by Barjhoux et al. (2012) (abnormal positioning and heart looping) after Cd exposure.

2.3. Effect of Cd on Reproduction

Examination of multiple biomarkers provides information on the exposure and reproductive health of the exposed fish (McMaster et al., 1996; Murphy et al., 2005). Plasma concentrations of hormones taken at specified stages during the reproductive cycle can be a good indication of disruption of the reproductive process. However, such biomarkers can be confounded by naturally occurring fluctuations in the reproductive cycle, and care must be taken when considering the timing of biomarker measurements (McMaster et al., 2001). Biomarkers are snapshots in time from a dynamic system. There is a need for models that can relate biomarker measurements to the entire reproductive cycle, and that can extrapolate biomarkers to ecologically relevant endpoints such as the production of eggs.

Disturbances of reproductive function in field-caught female fish have been inferred from changes in endocrine and reproductive function biomarkers, such as altered sex steroid hormone, atypical gonadotropin and vitellogenin concentrations in circulation, abnormal gonadal and oocyte growth. A large body of work exists outlining the effects of Cd on gametogenesis in both males and females, and implicating its compounds in early embryolethality and implantation failure. In addition, fish studies have shown a wide range of anomalies following exposure at specific stages of embryogenesis, and emerging evidence has indicated that Cd may also be linked to pathological processes in late pregnancy and in the early postnatal period, causing third trimester complications and minor but significant problems in the offspring of exposed individuals (Thompson and Bannigan, 2008). The acute toxicity of Cd to fish has been shown to be species specific, and the sensitivity can change at various life stages (Zhang et al., 2012).

2.3.1. Males

In fish, the male reproductive system is especially sensitive towards adverse effects of heavy

metals. Although heavy metal concentrations in water are rarely directly dangerous for fish, heavy metals are known to accumulate in fish tissues becoming extremely harmful (Kime et al., 1996). Therefore accumulation of heavy metals in gonads is related to decreased quality of gametes, including sperm motility. Cd ions affect many mechanisms involved in fish reproduction such as seasonal hormonal cycles, spermatogenesis and may cause degeneration of testis via necrosis (Kime, 1998, 1999; Mousa and Mousa, 1999). In addition, compete between Cd2+ ions and other bivalent ions, e.g. zinc ions may affect, therefore, the fish reproduction (Favier, 1992).

Fish spermatozoa are generally characterized by short-lasting motility. Among fish rainbow trout spermatozoa are distinguished as having one of the shortest motility periods of about 30–60s. Considering the very short time available for fertilization after the release of sperm into water (15 s), any negative effect (lowering sperm velocity as an example) on sperm motility may dramatically decrease the fertilization success (Gage et al., 2004). It was reported that sperm motility characteristics are sensitive and reliable indicators of aquatic pollution (Lahnsteiner et al., 2004; Abascal et al., 2007; Dietrich et al., 2010). There are many possible ways in which heavy metals may affect sperm motility activation and movement (Lahnsteiner et al., 2004). Teleost sperm plasmalemmal structure is highly permeable to low molecular weight substances which allows heavy metals to penetrate the sperm cell (Lahnsteiner et al., 1999). Heavy metals may bind to flagellum proteins affecting sperm movement symmetry and beat-cross frequency, or bind to enzymes affecting metabolism of the sperm cell (Dietrich et al., 2010). It was also reported that Cd causes deformation of the testis, induces seasonal changes in the androgen level in brook trout (*Salvelinus fontinalis*) (Sarosiek et al., 2009) and alters hormone synthesis in rainbow trout (*Oncorhynchus mykiss*) testis (Vetillard and Bailhache, 2005). Sarosiek et al., (2009) demonstrated that heavy metal ions not only distort the motility parameters of spermatozoa but also inhibit the activity of sperm enzymes, thus, consequently, may inhibit their fertilizing capacity. In view of the common carp's sensitivity to pollution (Lam et al., 1998) and the accumulation of high quantities of heavy metals in fish ponds (Szulkowska-Wojczak et al., 1992), these data indicate that the activity of sperm enzymes in common carp (*Cyprinus carpio*) may serve as a marker of pollution in water bodies (Sarosiek et al., 2009).

Besides lowering the percentage of sperm motility, Cd affects sperm speed and movement trajectory (Chyb et al., 2001; Dietrich et al., 2010). These changes can be monitored by the computer-assisted sperm analysis system (CASA), which measures fast and objectively the effect of heavy metal toxicity on fish sperm quality (Lahnsteiner et al., 2004; Abascal et al., 2007). Since calcium is a necessary element for sperm movement, the displacement of calcium ions by Cd may cause the inhibition of sperm motility in many species (Dietrich et al., 2010a). Cd toxicity also includes the disruption of the blood-testis barrier and the inhibition of spermatogenesis and gamete maturation (Dietrich et al., 2010b).

2.3.2. Females

In female fish, Cd inhibits gonadotropin stimulated steroidogenesis and ovarian maturation (e.g. common carp *Cyprinus carpio* (Mukherjee et al., 1994). Trout oogenesis appeared to be delayed by extensive exposition to Cd (Brown et al., 1994), while eggs obtained from females exposed to Cd did not develop to the fry stage. A direct exposition of rainbow trout fertilized eggs to Cd induced premature hatching, mortality, and developmental abnormalities (Woodworth and Pascoe, 1982). Vitellogenesis has been shown to be altered in winter flounder (*Pleuronectes americanus*) populations collected from Cd contaminated areas (Pereira et al., 1992) or after experimental Cd exposure (Pereira et al., 1993). In rainbow trout, Cd treatments inhibited estradiol stimulated transcription and translation of vitellogenin (Vg) (Hwang et al., 2000). In oviparous species, during vitellogenesis, Vg is produced by the liver, released, and transported by blood to the ovaries, then it is incorporated and processed into oocytes to form the major yolk protein (Wallace, 1978). The first step of this process is controlled by circulating estradiol (E2) (Tata and Smith, 1979), the action of which is mainly mediated by the estrogen receptor (ER), a nuclear receptor that functions as a ligand dependant transcription factor. Cd has been shown to inhibit rainbow trout ER (rtER) biological activity by diminishing its interaction with DNA (Le Guevel et al., 2000). These data suggest that Cd could have a wide range of effects and could interfere with other E2-controlled gene expression.

Murphy et al. (2005) used a physiological model to simulate how two non estrogenic EDCs that act via different mechanisms could affect vitellogenesis in fish. Vitellogenesis results in the production of the yolk precursor protein vitellogenin. Vitellogenesis is sensitive to disruption by EDCs and of ecological relevance because vitellogenin production is directly related to the reproductive output (fecundity and egg quality) of individual fish (Tyler and Sumpter, 1996). Cd can affect *Vg* gene

expression by a direct binding to rtER followed by the inhibition of its transactivation function (Le Guevel et al., 2000). In addition to this direct inhibition of rtER transactivation function, Vetillard and Bailhache (2005) showed that Cd reduces rtER mRNA levels in the liver, which is likely to lower the amount of rtER, the main regulatory factor for *Vg* gene expression (Flouriot et al., 1997). Both mechanisms could be involved in the strong inhibition of *Vg* gene expression. However, when fish were exposed to a high dose of E2, Cd was not able to totally inhibit rtER upregulation and did not modify *Vg* gene expression (Vetillard and Bailhache, 2005). This could explain why Cd is able to delay ovulation or to disrupt vitellogenesis in trout (Brown et al., 1994) but does not totally abolish vitellogenin production. It has also been reported that Cd induces accumulation of gonadotropin in the pituitary of catfish, *Clarias batrachus* (Jadhao et al., 1994) which could be the consequence of *GnRH* gene expression activation (Vetillard and Bailhache, 2005). Taken together, these data provide evidence that Cd is an important endocrine disrupter that may act on different tissues and alter genetic programs that may or may not be controlled by E2.

Because of a lack of protection of the egg membrane, a large amount of water- soluble Cd likely penetrated into the embryos and accumulated around the eggs, especially at high concentrations, and finally led to death (Cao et al., 2009). Zhang et al. (2012) reported a considerable reduction in the hatching rate and delayed hatching were observed in Cd treatment groups. Similar results of reduced or delayed hatching rate in Cd exposure has been demonstrated by previous studies in zebrafish, common carp, Atlantic salmon (*Salmo salar*) and spotted rainbow fish (Williams and Holdway, 2000; Fraysse et al., 2006; Zhang et al., 2012).

The impacts of Cd on the ovaries of female fish will cause their offspring (F1 larvae) to exhibit delayed or no metamorphosis and interference with their gonad development (Flament et al., 2003). In addition, the influence of long-term exposure of Cd to goldfish (*Carassius auratus*) caused a decrease in the gonadosomatic index (GSI), and ovulation did not occur (Szczerbikm et al., 2006). Those studies demonstrated that maternal Cd can significantly interfere with the reproduction of females and the growth of their larvae. However, there are some beneficial factors that are passed down to larvae through maternal effect, such as exhibiting a small quantity of Cd which could enhance the resistance of ambient Cd in F1 larvae (Wu et al., 2008; Wu et al., 2012). This observation corroborate with our comparative study to Cd sensitivity of two mosquitofish populations (*Gambusia affinis*). Indeed, we showed that the population from Cd polluted site was found to be more resistant to Cd than that from the reference site and so are the offspring (F1) of the two populations (Annabi et al., 2009).

2.3.3. Comparison between Males and Females

Cd has been shown to be responsible for a number of reproductive abnormalities in males and females fishes. Das (1988), found that Cd reduced the gonadosomatic index of both male and female asian cyprinids (*Labeo bata*). Futhermore, spermatids and spermatozoa were absent in the testes of Cd-exposed males. Likewise, the ovaries of exposed females contained no mature oocytes and had a significantly higher proportion of atretic follicles relative to ovaries from unexposed females. Cd has also been shown to induce the production of vitellogenin in Atlantic croaker (*Micropogonias undulates*) (Thomas, 1989). In addition, both male and female Japanese medaka (*Oryzias latipes*) exposed to Cd experience present a significant reduction in gonadal steroid release relative to controls (Tilton et al., 2003). The ability of Cd to alter reproductive health may be, in part, related to its ability to activate both androgen and estrogen receptors. Several *in vitro* studies have shown that Cd has estrogenic and androgenic properties (Sellin and Kolok, 2006). Additionally, it was reported that exposure to Cd damages gonads and impairs gametogenesis. In addition to alterations in gametogenesis, exposure to Cd has been shown to alter steroidogenesis. Alterations in circulating sex steroids have been documented for male and female Japanese medaka (*Oryzias latipes*) exposed to Cd (Foran et al., 2002; Tilton et al., 2003). Cd reduces also the expression of rainbow trout E2 receptor α (long isoform) genes in the liver and increases the expression of salmon gonadotropin releasing hormone genes in the brain (Vetillard and Bailhache, 2005). The abnormalities in gonad and endocrine function caused by exposure to Cd could potentially impair reproductive success (Sellin and Kolok, 2006).

According to the model simulation of Murphy et al. (2005), measurements of steroid levels from contaminant exposed fish that were taken during the first 2 months of gonadal recrudescence showed the greatest difference from control fish and that estradiol is a more sensitive biomarker than testosterone. Previous research indicates that depressed steroid levels translate into reproductive impairment (McMaster et al., 2001). Therefore, proper interpretation of estradiol concentrations from field-caught fish is predicated upon knowing the stage in the reproductive cycle of the individual fish. The simulation of Cd effects demonstrated how the model could be used to simulate EDCs that affect

multiple sites on the hypothalamus–pituitary–gonad–liver (HPGL) axis. Cd affects the HPGL axis at the pituitary by stimulating the release of LH, and Cd also acts on the ovary to enhance steroidogenesis (Thomas and Khan, 1995). Murphy et al., (2005) simultaneously imposed both of these effects in Cd exposure simulation.

The imposition of higher rates of gonadotropin introduction and testosterone synthesis assumed under Cd exposure resulted in increased total estrogen receptor concentrations, total testosterone, and total estradiol concentrations. Predicted total testosterone concentrations under Cd exposure increased similar to baseline conditions, but attained a slightly higher maximum concentration at 1.4 times baseline. Predicted total estradiol concentrations under Cd exposure rose more rapidly and maintained maximum concentrations at 3.2 times that of baseline conditions. Free testosterone and estradiol concentrations also reached higher peak concentrations under Cd exposure than under baseline conditions. Free testosterone concentrations under Cd exposure peaked at a concentration 1.4 times that of baseline conditions. Free estradiol levels were more sensitive to Cd exposure and reached a maximum at 3.3 times that observed under baseline conditions (Murphy et al., 2005). On the other hand, fish from the contaminated sites have been shown to be partially feminized, as evidenced by the presence of VTG in males (e.g. male mediterranean killifish *Aphanius fasciatus* (Annabi et al., 2012).

4. Conclusion

There is now extensive evidence of reproductive and developmental abnormalities in fish and wildlife populations exposed to a wide variety of chemicals in the environment such as Cd. The aquatic environment is a sink for endocrine physiological disrupting chemicals and other organic chemicals. Fish immersed in the aquatic environment bioaccumulate lipophilic chemicals via ingestion from food items and via absorption of contaminants through their gills and scales. The levels of contamination by Cd in fish are of considerable interest because fish consumption is an important source of intake Cd for the general population. In summary, human populations consume a large number of sea food mainly fish that can be detectable amounts of heavy metals; however the potential benefit of fish consumption is very important. Health institutions, public and private organizations must have a continuous communication about risk benefit of fish consumption, this confirms the interest to analyze of bases on which a public policy is elaborated, as well as, the responsibility for regulating the quality and improve the balance between benefit and risk of the fish human consumption. Therefore the intake of fish should be regulated; information regarding the species of fish consumed and its possible levels of content of heavy metals can be of benefit to diminish the hazard to public health.

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