**Toxicity of Heavy Metal in Live Stock**

Mona S. Zaki1, Refat A. Youssef2, and Sami Shalby3

1Hydrobiology Department, National Research Centre, Cairo, Egypt

2Soils and Water Uses Department, National Research Centre, Cairo, Egypt

3Animal Reproduction Department, National Research Center, Cairo, Egypt

[Dr\_mona\_zaki@yahoo.co.uk](mailto:Dr_mona_zaki@yahoo.co.uk)

**Abstract:** Cancer The IARC has classified cadmium as a human carcinogen (group I) on the basis of sufficient evidence in both humans and experimental animals 22. IARC, however, noted that the assessment was based on few studies of lung cancer in occupationally exposed populations, often with imperfect exposure data, and without the capability to consider possible confounding by smoking and other associated exposures (such as nickel and arsenic). Cadmium has been associated with prostate cancer, but both positive and negative studies have been published. Early data indicated an association between cadmium exposure and kidney cancer 23. Later studies have not been able clearly to confirm this, but a large multi-centre study showed a (borderline) significant over-all excess risk of renal-cell cancer, although a negative dose–response relationship did not support a causal relation 24. Furthermore, a population-based multicentre-study of renal cell carcinoma found an excess risk in occupationally exposed persons25. In summary, the evidence for cadmium as a human carcinogen is rather weak, in particular after oral exposure. Therefore, a classification of cadmium as ‘probably carcinogenic to humans’ (IARC group 2A) would be more appropriate. This conclusion also complies with the EC classification of some cadmium compounds (Carcinogen Category 2; Annex 1 to the directive 67/548/EEC).

[Mona S. Zaki, Refat A. Youssef, Sami Shalby. **Toxicity of Heavy Metal in Live Stock.** *Researcher* 2018;10(1):8-12]. ISSN 1553-9865 (print); ISSN 2163-8950 (online). <http://www.sciencepub.net/researcher>. 2. doi:[10.7537/marsrsj100118.02](http://www.dx.doi.org/10.7537/marsrsj100118.02).

**Keywords**: Toxicity; Heavy Metal; Live Stock

**Introduction:**

Anthropogenic activities, such as mining and industrial processing, are the main sources of heavy metal contamination in the environment. Under certain conditions, these metals may accumulate to a toxic concentration level which can lead to ecological damages. Several methods have been proposed to estimate the potential health risks of pollutants, divided mainly into carcinogenic and noncarcinogenic effects. For carcinogenic contaminants, the observed or predicted exposure concentrations are compared with thresholds for adverse effects or the toxicant reference value (TRV) as determined by dose–effect relationships. The probability risk assessment technique has been adopted by a number of researchers to fully utilize available exposure and toxicity data. However, these methods are only applied to quantify the magnitude of health risks of carcinogenic pollutants, but not for quantifying noncancer risks. Current noncancer risk assessment methods do not provide quantitative estimates on the probability of experiencing noncancer effects from contaminant exposures. These are typically based on the target hazard quotient (THQ), which is a ratio of determined dose of a pollutant to the dose level (a Reference Dose or RfD). If the ratio is less than 1, there will not be any obvious risk. Conversely, an exposed population of concern will experience health risks.

Although the THQ-based risk assessment method does not provide a quantitative estimate on the probability of an exposed population experiencing a reverse health effect, it indeed provides an indication of the risk level due to pollutant exposure. This risk estimation method has recently been used by  and proved to be valid and useful. This noncancer risk assessment method was also applied in this study.

The U.S. EPA is developing approaches for quantitative estimates of health risks for noncarcinogenic effects. Similar techniques used in cancer risks (i.e., dose–response data and linear low dose extrapolation) are being applied for these purposes, but are not yet widely use.

The suburban and urban areas of Tianjin City, China, are polluted by various heavy metals but information on the health risks of these metals is quite limited. The main objective of this study is to estimate the health risks of heavy metals, such as Cu, Zn, Pb, Cd, Hg, and Cr, via consumption of vegetables and fish to the general public in four districts (Dong Li, Xi Qing, Jin Nan, and Bei Chen) and the urban area of Tianjin using the target hazard quotient (THQ) estimates.

Because dietary habits are among the major concerns of Man, and are increasing in importance in almost all countries, ingestion, both in amount and quality, of the nutrients for good functioning of the human organism is fundamental. Among the products available, fish is currently considered one of the most interesting.

Fish is healthy, nutritious, and highly essential in a balanced diet, being an important source of proteins and lipids of high biological value, with long chain polyunsaturated fatty acids, and also liposoluble vitamins. Epidemiological studies indicate that populations with a fish diet have a lower risk of coronary heart disease, hypertension, and cancer. Thus, all over the world, fish assume enormous importance and consumption is increasing. The annual consumption of fish in Portugal, estimated in 1999 as 58.1 kg per capita year−1, proves that Portugal is the biggest consumer of fish among all the EU countries and one of the biggest in the world.

Fish can, on the other hand, be a source of contamination, because of the amounts of heavy elements they can contain, some of which are highly toxic.

Although nutrient composition data for common food, including fish, are widely available, knowledge of the elements present in fish is confined to selected minerals and trace element levels, especially for heavy metals, are known for a few seafood species only. Depending on their biological roles, elements may be divided into essential elements, the biological roles of which are known, non-essential elements, with unknown functions, if any, and toxic elements, which may be ingested in food or water or absorbed from the air. Most of these elements are detected in fish and shellfish, which inevitably supply both essential and toxic elements to the human organism. Even elements considered essential by some authors may be toxic when ingested in excess.

The concentration of elements in fish is influenced by a number of factors such as seasonal factors and biological differences (species, size, age, sex, and sexual maturity), nourishment source, environment (water chemistry, salinity, temperature, and contaminants), and the method of food processing.

Heavy metal contamination of aquatic ecosystems has been recognised as a serious pollution problem. All heavy metals are potentially harmful to most organisms at some level of exposure and adsorption. Contamination of aquatic environments with potentially harmful substances, in particular non-degradable heavy metals, and its subsequent impact on organisms, is more dramatic within estuaries and semi- Fevzi Yılmaz 8 closed coastal zones, especially when they are near highly populated or industrial areas. Heavy metals may enter an estuary from different natural and anthropogenic sources, including industrial or domestic sewage, storm runoff, leaching from landfills, shipping and harbour activities, and atmospheric deposits. Fish are often at the top of the aquatic food chain and may concentrate large amounts of some metals from the water. In addition, fish are most indicative factors in fresh water systems, for the estimation of trace metal pollution and risk potential of human consumption. For the normal metabolism of fish, the essential metals like copper and zinc must be taken up from water, food or sediment. However, similar to the route of essential metals, non-essential ones are also taken up by fish and accumulate in their tissues. Anguilla anguilla, European eel, is an amphihaline species, which migrates to the depths of the Sargasso Sea to spawn. Its food includes virtually the whole aquatic fauna occurring in the eel's area, augmented with animals living out of water, e.g. worms. Utilized fresh, dried or salted, smoked and frozen; can be fried, boiled and baked. They live on the bottom, under stones, in the mud or in crevices. Mugil cephalus, Flathead mullet, is a cosmopolitan in coastal waters of the tropical and subtropical zones of all seas that often enters estuaries and rivers. Mainly diurnal, feed on zooplankton, benthic organisms and detritus. Adult fish tend to feed mainly on algae while inhabiting fresh waters. Marketed fresh, dried, salted, and frozen; or sold fresh or smoked. They are very important commercial species in many parts of the world. Oreochromis niloticus, Nile tilapia, occurs in a wide variety of freshwater habitats like rivers, lakes, sewage canals and irrigation channels. Herbivorous, feeds on water plants and epiphyton, and some invertebrates and phytoplankton or benthic algae. These fish species are considered to be important part of the diet, around the estuaries study area, Köyceğiz Lake. This area has also an economic importance for fishery, especially for Mugilidae sp. But, on the other hand, the region is under the effect of pressure of pollution originating from touristic activities, boat and shipping traffic, agriculture and similar industries, gradually degrading the highly suitable environmental factors for the survival and growth of these commercial fishes. The contaminated fish from this area may become a public health concern. Therefore, in order to assess the metal contamination of the aquatic environment of lagoon system, information on elemental concentration in fish species in Köyceğiz Lake and Lagoon System becomes of great importance.

There are agricultural lands and industrial plants (iron–steel plants, LPG plants, oil transfer docks, and cargo ship’s ballasts water). In this region, there are several big lagoons and among these the third biggest lagoon is Tuzla. Due to heavy industrial and agricultural activities in the region, the bay has the polluted coastal waters of Turkey.

Therefore, mainly untreated agricultural municipal and industrial wastes affect the lagoon direct or indirectly. This lagoon has a great importance for the local fisheries activities. According to the report of the Ministry of Agriculture and Rural Affairs of Turkey, 30 tons of fish was supplied from the lagoon, in 1995. Besides being an important area for local fisheries, Tuzla lagoon is also stated as the wild life protection area because of its biodiversity. Although, a few studies were completed on determination of pesticide pollution (C¸ etinkaya & Altan, 1998; Erbatur, Kusvuran, & Erbatur, 1997; \_ Izcankurtaran & Yılmaz, 2001), this research, on determination of heavy metal pollution, was carried out for the first time for Tuzla Lagoon.

Dramatically during the 20th century, one reason being that cadmium-containing products are rarely re-cycled, but often dumped together with household waste.

Cigarette smoking is a major source of cadmium exposure. In non-smokers, food is the most important source of cadmium exposure. Recent data indicate that adverse health effects of cadmium exposure may occur at lower exposure levels than previously anticipated, primarily in the form of kidney damage but possibly also bone effects and fractures. Many individuals in Europe already exceed these exposure levels and the margin is very narrow for large groups.

Therefore, measures should be taken to reduce cadmium exposure in the general population in order to minimize the risk of adverse health effects. The general population is primarily exposed to mercury *via* food, fish being a major.

Source of methyl mercury exposure, and dental amalgam. The general population does not face a significant health risk from methyl mercury, although certain groups with high fish consumption may attain blood levels associated with a low risk of neurological damage to adults. Since there is a risk to the fetus in particular.

**Cadmium**

*Occurrence, exposure and dose*

Cadmium occurs naturally in ores together with zinc, lead and copper.

Cadmium compounds are used as stabilizers in PVC products, colour pigment, several alloys and, now most commonly, in re-chargeable nickel cadmium batteries. Metallic cadmium has mostly been used as an anticorrosion agent (cadmiation). Cadmium is also present as a pollutant in phosphate fertilizers. EU cadmium usage has decreased considerably.

During the 1990s, mainly due to the gradual phase-out of cadmium products other than Ni-Cd batteries and the implementation of more stringent EU environmental legislation (Directive 91/338/ECC). Notwithstanding these reductions in Europe, however, cadmium production, consumption and emissions to the environment worldwide have increased dramatically during the 20th century. Cadmium containing products are rarely re-cycled, but frequently dumped together with household waste, thereby contaminating the environment, especially if the waste is incinerated.

Natural as well as anthropogenic sources of cadmium, including industrial emissions and the application of fertilizer and sewage sludge to farm land, may lead to contamination of soils, and to increased cadmium uptake by crops and vegetables, grown for human consumption. The uptake process of soil cadmium by plants is enhanced at low pH4.

Cigarette smoking is a major source of cadmium exposure. Biological monitoring of cadmium in the general population has shown that cigarette smoking may cause significant increases in blood cadmium (B-Cd) levels, the concentrations in smokers being on average 4–5 times higher than those in non-smokers4. Despite evidence of exposure from environmental tobacco smoke, however, this is probably contributing little to total cadmium body burden.

Food is the most important source of cadmium exposure in the general non-smoking population in most countries. Cadmium is present in most foodstuffs, but concentrations vary greatly, and individual intake also varies.

Considerably due to differences in dietary habits. Women usually have lower daily cadmium intakes, because of lower energy consumption than men. Gastrointestinal absorption of cadmium may be influenced by nutritional factors, such as iron status.

B-Cd generally reflects current exposure, but partly also lifetime body burden8. The cadmium concentration in urine (U-Cd) is mainly influenced by the body burden, U-Cd being proportional to the kidney concentration.

Smokers and people living in contaminated areas have higher urinary cadmium concentrations, smokers having about twice as high concentrations as non-smokers.

**Health effects:**

Inhalation of cadmium fumes or particles can be life threatening, and although acute pulmonary effects and deaths are uncommon, sporadic cases still occur. Cadmium exposure may cause kidney damage. The first sign of the renal lesion is usually a tubular dysfunction, evidenced by an increased excretion of low molecular weight proteins [such as 2-microglobulin and 1-microglobulin (protein HC)] or enzymes [such as N-Acetyl--D-glucosaminidase (NAG)]4,6. It has been suggested that the tubular damage is reversible11, but there is overwhelming evidence that the cadmium induced tubular damage is indeed irreversible. WHO6 estimated that a urinary excretion of 10 nmol/mmol creatinine (corresponding to *circa* 200 mg Cd/kg kidney cortex) would constitute a ‘critical limit’ below which kidney damage would not occur. However, WHO calculated that *circa* 10% of individuals with this kidney concentration would be affected by tubular damage. Several reports have since shown that kidney damage and/or bone effects are likely to occur at lower kidney cadmium levels. European studies have shown signs of cadmium induced kidney damage in the general population at urinary cadmium levels around 2–3 g Cd/g creatinine 12,13.

**Mercury**

*Occurrence, exposure and dose*

The mercury compound cinnabar (HgS), was used in pre-historic cave paintings for red colours, and metallic mercury was known in ancient Greece where it (as well as white lead) was used as a cosmetic to lighten the skin. In medicine, apart from the previously mentioned use of mercury as a cure for syphilis, mercury compounds have also been used as diuretics [calomel (Hg2Cl2)], and mercury amalgam is still used for filling teeth in many countries.

Metallic mercury is used in thermometers, barometers and instruments for measuring blood pressure. A major use of mercury is in the chloralkali industry, in the electrochemical process of manufacturing chlorine, where mercury is used as an electrode.

The largest occupational group exposed to mercury is dental care staff.

During the 1970s, air concentrations in some dental surgeries reached 20 g/m3, but since then levels have generally fallen to about one-tenth of those concentrations.

Inorganic mercury is converted to organic compounds, such as methyl mercury, which is very stable and accumulates in the food chain. Until the 1970s, methyl mercury was commonly used for control of fungi on seed grain.

The general population is primarily exposed to mercury *via* food, fish being a major source of methyl mercury exposure27, and dental amalgam.

Several experimental studies have shown that mercury vapour is released from amalgam fillings, and that the release rate may increase by chewing28.

Mercury in urine is primarily related to (relatively recent) exposure to inorganic compounds, whereas blood mercury may be used to identify exposure to methyl mercury. A number of studies have correlated the number of dental amalgam fillings or amalgam surfaces with the mercury content in tissues from human autopsy, as well as in samples of blood, urine and plasma26. Mercury in hair may be used to estimate long-term exposure, but potential contamination may make interpretation difficult.

**Organic mercury**

Methyl mercury poisoning has a latency of 1 month or longer after acute exposure, and the main symptoms relate to nervous system damage31. The earliest symptoms are parestesias and numbness in the hands and feet.

Later, coordination difficulties and concentric constriction of the visual field may develop as well as auditory symptoms. High doses may lead to death, usually 2–4 weeks after onset of symptoms. The Minamata catastrophe in Japan in the 1950s was caused by methyl mercury poisoning from fish contaminated by mercury discharges to the surrounding sea. In the early 1970s, more than 10,000 persons in Iraq were poisoned by eating bread baked from mercury-polluted grain, and several thousand people died as a consequence of the poisoning. However, the general population does not face significant health risks from methyl mercury exposure with the exception of certain groups with high fish consumption.

A high dietary intake of mercury from consumption of fish has been hypothesized to increase the risk of coronary heart disease32. In a recent case-control study, the joint association of mercury levels in toenail clippings and docosahexaenoic acid levels in adipose tissue with the risk of a first myocardial infarction in men was evaluated33. Mercury levels in the patients were 15% higher than those in controls (95% CI, 5–25%), and the adjusted odds ratio for myocardial infarction associated with the highest compared with the lowest quintile of mercury was 2.16 (95% CI, 1.09–4.29; *P* for trend = 0.006).

Another recent case-control study investigated the association between mercury levels in toenails and the risk of coronary heart disease among male health professionals with no previous history of cardiovascular disease.

Mercury levels were significantly correlated with fish consumption, and the mean mercury level was higher in dentists than in non-dentists. When other risk factors for coronary heart disease had been controlled for, mercury levels were not significantly associated with the risk of coronary heart disease34.

These intriguing contradictory findings need to be followed up by more studies of other similarly exposed populations.

**References**

1. *Department of Epidemiology*, Lars Järup, Hazards of heavy metal contamination 2017.
2. Department of the Environment, Transport and the Regions. *Statistics Release 184 1999 UK Air Emissions Estimates* (28 March 2001).
3. Berglund M, Elinder CG, Järup L. *Humans Exposure Assessment. An Introduction.* WHO/SDE/ OEH/01.3, 2001.
4. NRC. *Human Exposure Assessment for Airborne Pollutants. Advances and Opportunities*. Washington, DC: National Research Council, National Academy Press, 1991.
5. Jarup L, Berglund M, Elinder CG, Nordberg G, Vahter M. Health effects of cadmium exposure—a review of the literature and a risk estimate. *Scand J Work Environ Health* 1998; 24 (Suppl 1):1–51.
6. Hossn E, Mokhtar G, El-Awady M, Ali I, Morsy M, Dawood A. Environmental exposure of the pediatric age groups in Cairo City and its suburbs to cadmium pollution. *Sci Total Environ.*
7. WHO. *Cadmium*. Environmental Health Criteria, vol. 134. Geneva: World Health Organization, 1992.
8. Flanagan PR, McLellan JS, Haist J, Cherian MG, Chamberlain MJ, Valberg LS. Increased dietary cadmium absorption in mice and human subjects with iron deficiency. *Gastroenterology* 1978; 74: 841–6.
9. Järup L, Rogenfelt A, Elinder CG, Nogawa K, Kjellström T. Biological half-time of cadmium in the blood of workers after cessation of exposure. *Scand J Work Environ Health* 1983; 9: 327–31.
10. Seidal K, Jorgensen N, Elinder CG, Sjogren B, Vahter M. Fatal cadmium-induced pneumonitis. *Scand J Work Environ Health* 1993; 19: 429–31.
11. Barbee Jr JY, Prince TS. Acute respiratory distress syndrome in a welder exposed to metal fumes. *South Med J* 1999; 92: 510–2.
12. Hotz P, Buchet JP, Bernard A, Lison D, Lauwerys R. Renal effects of low-level environmental cadmium exposure: 5-year follow-up of a subcohort from the Cadmibel study. *Lancet* 1999; 354: 1508–13.
13. Buchet JP, Lauwerys R, Roels H, Bernard A, Bruaux P, Claeys F, Ducoffre G, DePlaen P, Staessen J, Amery A, Lijnen P, Thijs L, Rondia D, Sartor F, Saint Remy A, Nick L. Renal effects of cadmium body burden of the general population. *Lancet* 1990; 336: 699–702.
14. Jarup L, Hellstrom L, Alfven T, Carlsson MD, Grubb A, Persson B *et al.* Low level exposure to cadmium and early kidney damage: the OSCAR study. *Occup Environ Med* 2000; 57: 668–72.
15. Friberg L. Health hazards in the manufacture of alkaline accumulators with special reference to chronic cadmium poisoning. *Acta Med Scand* 1950; Suppl 240: 1–124.
16. Bernard A, Roels H, Buchet JP, Cardenas A, Lauwerys R. Cadmium and health: the Belgian experience. *IARC Scientific Publications* 1992; 118: 15–33.
17. Järup L, Persson B, Elinder C-G. Decreased glomerular filtration rate in cadmium expose solderers. *Occup Environ Med* 1995; 52: 818–22.
18. Hellström L, Elinder CG, Dahlberg B, Lundberg M, Järup L, Persson B, Axelson O. Cadmium exposure and end-stage renal disease. *Am J Kidney Dis* 2001; 38: 1001–8.

12/20/2017