INTRODUCTION

Exposure to metals like lead, cadmium, mercury, tin, and arsenic threaten life and human health so many International bodies including World Health Organization (WHO) regularly carry out extensive studies and review of their effects on human health. Heavy metals are in use by humans for thousands of years and in spite of knowing several of its ill effects on human health, the usage continues sometimes even flourishing, particularly in less developed nations. Normally humans absorb trace amounts of these elements through food, drinking water, air, etc.

Food contains a wide range of metals such as sodium, potassium, iron, calcium, boron, magnesium, selenium, copper, zinc, etc., that are essential in trace quantities for the up-keeping of our cellular processes a key to maintaining the metabolism of the human body. However, at high concentrations these very metals can cause poisonings resulting in impaired mental and central nervous function, as well as damage to vital organs. Long-term exposure may result in slowly progressing physical, muscular, and neurological degenerative conditions including cancer. Metals that have no functional effects in the body can surely be harmful to our health if we regularly consume foodstuffs containing them.

In recent years, a number of consumer products around the world, have had links to heavy metal contamination. Very recently in India, excess lead in Maggie noodles and the eventual ban on its consumption has again highlighted the importance of strict quality control in food products. All this very clearly emphasize that only a strong regulatory agency can take concrete steps to detect and minimize the presence of hazardous heavy metals in food and aid in preventing us from inadvertently ingesting these harmful elements that can impair our health and well-being.

WHAT IS A HEAVY METAL?

We define heavy metals as those metals that have a specific density of more than 5g/cm3 or their high atomic weight. In this context, the majority threat to human health is from our exposure to lead, cadmium, mercury, tin and arsenic (arsenic is a metalloid; however, we usually classify it as a heavy metal). Extensive studies on these metals, their adverse effects on human health are available along with regular reviews by International bodies and WHO.

Heavy metals are in use for thousands of years for example lead is in use for over 5000 years, in areas like building materials, pigments for glazing ceramics, water pipes, etc. Ancient Romans made use of lead acetate to sweeten old wine, probably some of them consuming even as much as a gram of lead a day. Early Romans made use of mercury as a salve to alleviate teething pain in infants, and as a remedy for syphilis. Claude Monet used cadmium pigments extensively in the mid 1800’s, in his artwork. Humans well are aware of the adverse health effects of heavy metals for a long time now, but still use and expose themselves to heavy metals sometimes even increasing its use and exposure in specific areas. Common examples are use of mercury for gold mining, arsenic in wood preservation, tetra-ethyl lead as an additive to petrol, etc.

Atmospheric emissions of heavy metals still tend to be the greatest concern in terms of human health, more due to the larger quantity, the widely spread out dispersion, and a maximum potential for human exposure. Lead emissions are mainly from road transport and so uniformly distributed over space. We can associate Cadmium emissions primarily with non-ferrous metallurgy and fuel combustion, whereas the spatial distribution of anthropogenic mercury emissions reflects mainly the level of coal consumption in different regions.

Exposure of people to potentially harmful chemicals could happen through physical and biological agents in air, food, water, or soil. However, the key to exposure is contact, between the agent and the outer boundary of the human body, like the airways, the skin, or the mouth. We define exposure as a function of concentration and time, an event that occurs at the boundary when there is contact between a human and the environment with a contaminant of a certain concentration for a specific interval of time. In short, for exposure to happen co-existence of contaminants and people has to occur. Metals are natural components of the earth’s crust and can be naturally present in food or can enter food due to our industrial and agricultural processes. Chemically, these elements can exist as a pure metal, viz., tin or lead, or compounds formed by combination of a metallic element with a non-metallic element for example a combination of sodium with chlorine (Sodium chloride or common salt) or oxygen (an oxide).

Other potentially toxic metal contaminants in food and water include chromium and uranium; workplace metal contaminants include beryllium and nickel. However, we do not find these metallic elements normally in food at levels that could cause toxicity. Nickel contamination in foodstuffs could be generally possible if we make use of hydrogenated oils and fats (viz., Vanasaptai) in food preparations. However, in this paper we limit our discussion to only mercury, lead, cadmium, tin, mercury, and arsenic.

TOXICITY OF HEAVY METALS

The two main aspects regarding toxicity of these metals are,
1. They have no known metabolic function, but when present in the body they disrupt normal cellular processes, leading to toxicity in a number of organs;
2. Heavy metals mercury and lead accumulate in biological tissues, getting stored in the liver or the kidney. Since heavy metals excrete at a slow rate in comparison to its uptake, bio-accumulation occurs; this happens in all animals, including food animals like fish, livestock, including humans and thus it becomes necessary to control toxic metal levels in foodstuffs to protect human health.

MERCURY

Prehistoric cave paintings made use of mercury compound cinnabar (HgS) for red colors. Metallic mercury (as well as white lead) in ancient Greece found use as a cosmetic to lighten skin. Apart from prehistoric uses, it also finds use as diuretic [calomel (Hg2Cl2)]. Mercury amalgam is still finding use for filling teeth in many countries. Metallic mercury usage in thermometers, barometers, and
Instruments for measuring blood pressure is common. Mercury electrodes find major use, in the electrochemical process of manufacturing chlorine in the chloralkali industry. The largest occupational groups exposed to mercury are the dental care staffs.

Excessive exposure to mercury causes damage to the central nervous system (neurotoxicity) and the kidney. Different forms of mercury (i.e., mercury metal, inorganic mercury salts such as mercuric chloride and organic forms of mercury such as methyl mercury) produce different toxicity patterns. The general population exposed to low levels of mercury in their diet relates to the potential neurotoxicity of the organic forms of mercury, viz., methyl mercury, especially in young children. Organic forms of mercury can cross the placental barrier between the mother and the baby in the womb. Epidemiological studies in human and toxicological studies in animals by international expert bodies show that toxicity can result in neurological disturbances ranging from an impaired learning to obvious brain damage. Acute mercury exposure may give rise to lung damage. Chronic poisoning leads to neurological and psychological symptoms, such as tremor, changes in personality, restlessness, anxiety, sleep disturbance and depression. However, these symptoms are reversible after cessation of exposure. Metallic mercury may cause kidney damage, which is also reversible after exposure has stopped. Metallic mercury is an allergen, causing contact eczema. Mercury from amalgam fillings may sometimes give rise to oral lichen in some individuals getting relief on removal of dental amalgam fillings. Due to the blood–brain barrier there is no central nervous involvement related to inorganic mercury exposure.

Inorganic mercury when converted to organic methyl mercury becomes very stable and accumulates in the food chain. Commonly people ingest mercury via food, with fish being a major source of methyl mercury exposure. Dental amalgam release mercury vapors with the release rate increasing by chewing. Mercury in urine is primarily due to recent exposure to inorganic compounds, whereas blood mercury is due exposure to methyl mercury. Methyl mercury poisoning having latency of 1 month or longer after acute exposure may cause nervous system damage, the earliest symptoms being parestesias and numbness in the hands and feet. Later, coordination difficulties and concentric constriction of the visual field could develop along with auditory symptoms. High doses may lead to eventual death. The biggest health risks from methyl mercury exposure are from high fish consumption. Intake of mercury from consumption of fish may also increase the risk of coronary heart disease.

LEAD

Consumption of food containing lead is the major source of exposure for the public. Short-term exposure to high levels of lead causes brain damage, paralysis (lead palsy), anemia, and gastrointestinal symptoms. Longer-term exposure causes damage to the kidneys, reproductive and immune systems in addition to effects on the nervous system. Low-level lead exposure effects the intellectual development in young children and, like mercury, can cross the placental barrier, and accumulate in the fetus. Infants and young children are more vulnerable to the toxic effects as they absorb lead more readily. Even short term, low-level exposures to lead can effect on neurobehavioral development of young children. The general population gets exposure to lead from air and food. Earlier, lead in foodstuff originated from pots used for cooking and storage, and use of lead acetate to sweeten port wine. During the last century, lead emissions to ambient air have further polluted our environment, mostly originating from leaded petrol. However, increasing use of unleaded petrol could decrease blood lead levels in the general population. Occupational exposure to inorganic lead occurs in mines, smelters, when welding of lead painted metal, in battery plants, in the glass industry, etc.

Airborne lead deposits on soil and water, and reaches humans via the food chain. We get lead as our lungs absorb the inhaled inorganic lead. 10–15% of lead comes from food. In case of children, almost 50% could be through the gastrointestinal tract. Lead binds itself to erythrocytes in our blood. Elimination is slow and principally via urine. Lead accumulates in the skeleton, and releasing very slowly. Half-life of lead in blood is about 1 month and in the skeleton 20–30 years. In adults, inorganic lead does not penetrate the blood–brain barrier, whereas this is not the case in children. The high gastrointestinal uptake and the permeable blood–brain barrier make children especially susceptible to lead exposure and subsequent brain damage. Organic lead compounds penetrate body and cell membranes. Tetra methyl lead and tetra-ethyl lead penetrate the skin easily. These compounds may also cross the blood–brain barrier in adults, thereby suffer from lead encephalopathy (sleeplessness and restlessness).

The symptoms of acute lead poisoning are headache, irritability, abdominal pain and various symptoms related to the nervous system. Children may exhibit behavioral disturbances, learning and concentration difficulties. In severe cases of lead encephalopathy, the affected person may suffer from acute psychosis, confusion and reduced consciousness. People with long time exposure may suffer from memory deterioration, prolonged reaction time and reduced ability to understand. Individuals with average blood lead levels under 3 μmol/l are likely to show signs of peripheral nerve symptoms with reduced nerve conduction velocity and reduced dermal sensibility.

In less serious cases, the most obvious sign of lead poisoning is disturbance of hemoglobin synthesis, and long-term lead exposure may lead to anemia. Blood lead levels in children below 10μg /dl could be so far acceptable, but recent data indicate that certain genetic and environmental factors can increase the detrimental effects of lead on neural development, thereby rendering certain children more vulnerable to lead neurotoxicity. Lead is a ‘possible human carcinogen’ and may cause lung & stomach cancer.

1. Since there is a risk to the fetus in particular, pregnant women should avoid a high intake of fish, such as shark, swordfish and tuna; fish (such as pike, walleye and bass) taken from polluted fresh waters.
2. Avoid amalgams in dental fillings.
3. Avoid use of leaded petrol and phase out any remaining uses of lead additives in motor fuels.
4. Do not use any lead based or glazed food containers, which may leach lead into food.
5. Abandon the use of lead-based paints.

CADMIUM

The principal effect of cadmium is its toxicity to the kidney. It also causes lung damage (including induction of lung tumors) and skeletal changes in occupationally exposed populations. Although cadmium is poorly absorbed into the body, it also is excretes very slowly accumulating in the
kidney causing renal damage and a secondary suspect in causing prostate cancer, kidney cancer, and renal cell carcinoma. The kidney of food animals is a major source of cadmium in human diet even though we find lower levels in many other foods.

Cadmium occurs naturally in ores together with zinc, lead and copper. Cadmium compounds find use as stabilizers in PVC products, color pigment, several alloys and, now most commonly, in rechargeable nickel–cadmium batteries. Metallic cadmium is as an anti-corrosion agent (cadmiation) and is present as a pollutant in phosphate fertilizers. Cadmium containing products are rarely re-cycled. When dumped together with other household waste, it only contaminates the environment, especially so during waste incineration. Natural cadmium along with industrial emissions, application of fertilizer and sewage sludge to farmlands, leads to soil contamination, and increased cadmium uptake by crops and vegetables especially at low pH 4. Cigarette smoking is also a major source of cadmium exposure causing significant increases in blood cadmium (B-Cd) levels. Cadmium concentrations in smokers on an average are 4–5 times higher than people who do not smoke. Food however, is the most important source of cadmium exposure as it is present in most foodstuffs, although concentrations vary greatly, with individual intake due to differences in dietary habits. Women usually have lower daily cadmium intakes, due to lower energy needs. Nutritional factors, like iron status influences gastrointestinal absorption of cadmium. B-Cd generally reflects the current exposure, and partly lifetime body burden unlike urine (U-Cd) cadmium concentration that is largely due to the body burden, being proportional to the cadmium concentration in the kidney. Smokers and people living in contaminated areas have higher urinary cadmium concentrations, with smokers having almost double levels.

**TIN**

Tin is comparatively less toxic than mercury, cadmium and lead. Presence of tin in food in higher levels is largely in canned food due to incorrectly manufactured tins, where tin present in the can leaches into the food. This occurs more in the case of acidic foodstuffs (canned tomatoes) consumption of which can result in gastrointestinal irritation and stomach upsets in some individuals at concentrations above 200 mg/kg. Organotin compounds reach humans primarily through diet of fish and fish products. These compounds are widely prevalent in the aquatic environment due to their agricultural use as anti-fouling agents and biocides. Tin is used in canned foods to protect the steel base from corrosion both externally (aerobic conditions) and internally when in contact with foods (anaerobic). Although the use of can lining lacquers enables a satisfactory packing, improper lining can cause enough damage.

Highly pigmented foods like beetroot, berries, etc., can have their colors bleached by tin dissolution. The tin in canned food is likely to be in the form of inorganic tin salts rather than tin in covalently bound organometallic compounds. The tin content of canned foods depends on whether the can is lacquered or food is stored in opened cans, the presence of oxygen, reducible organic compounds, the pH of the food, the presence of plant pigments, the storage conditions (i.e., time & temperature), food additives, etc. The chemistry of the food product can greatly change due to internal corrosion in plain tinplate cans. The point to note is that fruits, vegetables, and tomatoes themselves have significant natural variation in, pH (acid type) and concentration, dependent on variety, maturity, time/place/conditions of harvest, soil chemistry, and agricultural practices that are difficult for a canner to control, ultimately affecting the level of tin uptake by the packed product. Acidic foods are more aggressive to the tin coating in metal cans and canned acidic foods have higher tin contents. Tomato-based products tend to have high levels of tin as nitrate in the food accelerates corrosion of the tin. Tin concentrations of canned foods increase with storage time and temperature.

**ARSENIC**

Arsenic exists both in inorganic and organic forms and in different valence states. Arsenic is a widely distributed metalloid, occurring in rock, soil, water, and air. Inorganic arsenic is present in groundwater used for drinking in several countries all over the world (e.g., Bangladesh, Chile, and China). We generally find organic arsenic compounds (arsenobetaine) in fish, which may give rise to human exposure. Smelting of non-ferrous metals and the production of energy from fossil fuel are the two major industrial processes that lead to arsenic contamination of air, water, and soil. Other contamination sources are the manufacture and use of arsenical pesticides and wood preservatives. General population exposure to arsenic is mainly via intake of food and drinking water. Food is the most important source, but in some areas, arsenic in drinking water is a significant source of exposure to inorganic arsenic. Absorption of arsenic in inhaled airborne particles is highly dependent on the solubility and the size of particles. Soluble arsenic compounds are easily absorbed from the gastrointestinal tract. However, inorganic arsenic is extensively methylated in humans and the metabolites are excreted in the urine. Inorganic arsenic is significantly more toxic than organic arsenic compounds like dimethylarsinate. Likewise, trivalent forms of arsenic viz., arsenic trichloride, are more toxic than the pentavalent arsenates. Pentavalent arsenates become toxic only after metabolic conversion to the trivalent form of arsenic. Chromium also behaves similar to arsenic. Inorganic arsenic exposure causes cancer and classified as a human carcinogen. Continual low-level exposure to arsenic causes skin, vascular and nervous system disorders. Intake of large quantities leads to gastrointestinal symptoms, severe disturbances of the cardiovascular and central nervous systems, and eventually leading to death.

In survivors, we may observe bone marrow depression, haemolysis, hepatomegaly, melanosis, polynuropathy, and encephalopathy. Ingestion of inorganic arsenic may induce peripheral vascular disease, which in its extreme form leads to gangrenous changes. People exposed to arsenic via drinking water show excess risk of mortality from lung, bladder and kidney cancer, the risk increasing with increasing exposure. There is also an increased risk of skin cancer and other skin lesions, such as hyperkeratosis and pigmentation changes. Studies on various populations exposed to arsenic by inhalation, such as smelter workers, pesticide manufacturers and miners in many different countries consistently demonstrate an excess lung cancer. Studies show that lung cancer risk increases with increasing arsenic exposure. The latest WHO evaluation concludes that arsenic exposure via drinking water can relate to cancer in the lungs, kidney, bladder, skin, etc., sometimes even observing precancerous skin lesions. Uncertainties in the estimation of past exposures are important when assessing the exposure–response
relationships, but it would seem that drinking water arsenic concentrations of approximately 100 μg/l have led to cancer at these sites, and that precursors of skin cancer have been associated with levels of 50–100 μg/l. The relationships between arsenic exposure and other health effects are less clear. There is relatively strong evidence for hypertension and cardiovascular disease, but the evidence is only suggestive for diabetes and reproductive effects and weak for cerebrovascular disease, long-term neurological effects, and cancer at sites other than lung, bladder, kidney, and skin.

CONCLUSION

The Food Safety & Standards Act 2006 sets maximum levels for heavy metals in individual foodstuffs. For other food contact materials, including those made from metal and alloys, regulation sets a general requirement that migration of substances should not endanger the health of consumers. Food Operators/Manufacturers are responsible for the safety of food they produce, transport, store, or sell. They must also ensure that their products comply with the legislative limits for heavy metals as laid down by law. Codex Alimentarius adopts a code of practice for the prevention and reduction of inorganic tin contamination in canned foods. It is important that all food operators should identify the ‘Critical Control Points’, (CCPs) in their processes along their food operators chain to enable them develop and apply proper HACCP systems to ensure that there are no unforeseen sources of metal contamination.

An important point we have to note is that, it is the responsibility of the food operators to test their products for mercury, lead, cadmium, tin, and arsenic content at the point of sale, carrying out sampling and analysis in accordance with the principles outlined in official protocols in accredited laboratories and to comply with the requirements of regulations.

New York City Bans Expandable Polystyrene Foam

New York City is joining a growing group of cities in banning Expandable Polystyrene Foam (EPS) a long-lasting material worrying to environmentalists but appealing to businesses. Starting 1 July 2015, New York City will not offer, allow sale, permit to possess any single-use EPS products including cups, bowls, plates, takeaway containers and trays peanuts packs, etc. Companies have six months to comply or face a fine.

What is EPS anyway?

EPS or “Styrofoam” was invented by scientist Otis Ray McIntire in 1941. To make it, small beads of the polymer polystyrene are steamed with chemicals until they expanded to 50 times their original volume. After cooling and settling, the pre-expanded beads are then blown into a mould such as that of a drink cup or cooker and steamed again, expanding further, until the mould is completely filled and all of the beads have fused together. The finished product is a lightweight, inexpensive material that is about 95% air. The insulating properties and cheap manufacturing costs of EPS have made it a popular choice for businesses. Popularly also known as “Thermocol” it is most commonly used for packaging foodstuffs, medical supplies, electrical consumer goods and insulation panels.

What makes it so bad for the environment?

It is estimated that Americans alone throw away 25 billion polystyrene coffee cups a year. In 2006, for instance, 135 tons of polystyrene waste was dumped into Hong Kong landfills. Environmentalists say that EPS waste causes outsized trouble when it leaks into marine environments and contaminates water.

Douglas McCauley, a marine biology professor at the University of California, Santa Barbara, says, there are two main issues that polystyrene causes for marine animals, mechanical and chemical. “The [mechanical root] is very straight-forward,” said McCauley, “Oftentimes, we find polystyrene foam lodged in the intestines that causes blockages that can be lethal. If you think about how we worry about a mild blockage from eating the wrong thing, imagine eating a ball of Styrofoam. That’s what some of these animals are doing.” Chemically, absorbent properties make EPS even more dangerous. “Polystyrene foams essentially act like little pollutant sponges, picking up and concentrating some of the nastiest contaminants in the ocean,” McCauley says. “Then something like a sea turtle comes along and eats this thinking it is a jellyfish.”

Why you cannot recycle EPS?

The difficulty recycling EPS was a main reason New York City enacted the ban. Kathryn Garcia, New York City’s sanitation commissioner, said, “It has not been proven that recycling dirty foam can be done on a large scale, and there is no demonstrated market for this material.” Due to the chemical process that turns polystyrene beads into EPS, it’s nearly impossible to turn an EPS plate, for example, into an EPS takeaway box. “You couldn’t just take recycled Styrofoam cups and make moulds again because it’s already expanded,” says Joe Biernacki, professor of chemical engineering at Tennessee Tech University. “What you need are virgin polystyrene beads.”

There is research being done to see if EPS can be re-collapsed inexpensively to form beads again but so far there a few practical ways to recycle. One method that has been tested is thermal recycling. In this process, the recycled EPS is burned in municipal incinerators, leaving behind carbon dioxide and water vapors. This makes it a good fuel for waste-to-energy programs that use heat. While thermal recycling could be an effective re-use of polystyrene waste, its viability is offset by the cost of transporting loads of light, bulky polystyrene to recycling centers.

What are the alternatives?

Paper-based alternatives and products made of recyclable resin-based polypropylene that is often used for plastic takeaway containers is a better option, even though comparatively are more expensive. Indian style metallic reusable containers or mud based environmentally friendly “Kullad” that can be made plentiful and cost less could lead the way at least in India. Do you agree?