International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Review Article

Heavy Metals Toxicity

Shikha Bathla, Tanu Jain

Research Scholar, Department of Food and Nutrition, Punjab Agricultural University, Ludhiana-141004.

Corresponding Author: Shikha Bathla

Received: 15/02/2016 *Revised: 13/04/2016* *Accepted: 18/04/2016*

ABSTRACT

A heavy metal is a member of a loosely defined subset of elements that exhibit metallic properties. It mainly includes the transition metals, some metalloids, lanthanides, and actinides. Many different definitions have been proposed based on density, atomic number or atomic weight, and some on chemical properties. Heavy metal toxicity can result in damaged central nervous function, lower energy levels, and damage to blood composition, lungs, kidneys, liver, and other vital organs. Long-term exposure may result in slowly progressing physical, muscular, and neurological degenerative diseases. Exposure to toxic or heavy metals comes from many sources like in fish, chicken, vegetables, vaccinations, dental fillings and deodorants. Remedies to combat heavy metal toxicity can be to adopt the practice of kitchen gardening and also to ensure plethora supply of antioxidant includes fruits and vegetables in the diet Increase intake of *miso soup* (made from soya) and garlic and regular exercise and brisk walking. Increase intake of water to detoxify the harmful effect of heavy metals. Use of lead free paints and avoids carrying metal accessories.

Key words: heavy metals, lead, selenium, mercury, silicon.

INTRODUCTION

Metals occurrence in the environment has become a concern because the globe is experiencing a silent epidemic of environmental poisoning, from the ever increasing amounts of metals released into the biosphere. A heavy metal is a member of a loosely defined subset of elements that exhibit metallic properties. Heavy metals are those, which have specific gravity (density) of more than 5g/cm3 in their standard state. It is not that every heavy metal that is harmful to life. Living organisms require varying amounts of "heavy metals". Iron, cobalt, copper, manganese, molybdenum, and zinc are required by humans. Excessive levels can be damaging to the organism. Heavy metal exerts their toxic effect by combining with one or more reactive groups essential for the normal physiological functions. They react in body with ligands containing oxygen (OH, -COO,-OPO_3H, >C=O) sulphur (-SH, -S-S-), and nitrogen (-NH and >NH) and affect the body by interaction with essential metals, formation of metal protein complex, age and stage of development, lifestyle factors, immune status of the host (Young 2005; Nolan 2003; Duruibe *et al* 2007). Metal toxicity or metal poisoning is the toxic effect of certain metals in certain forms and doses on life. Some metals are toxic when they form poisonous soluble compounds. Certain metals have no biological role, i.e. are not essential minerals, or are toxic when in a certain form. In the case of lead, any measurable amount may have negative health effects (CDC 2012).

Indicators of environmental metal pollution. Heavy metals are naturally occurring elements that are found throughout the earth's crust, most environmental contamination and human exposure result from anthropogenic activities such as mining and smelting operations, industrial production and use, and domestic and agricultural use of metals and metal-containing compounds (He *et al* 2005; Shallari *et al* 1998; Paul *et al* 2012). Environmental contamination can also occur through metal corrosion, atmospheric deposition, soil erosion of metal ions and leaching of heavy metals, sediment re-suspension and metal evaporation from water resources to soil and ground water (Nriagu 1989). Natural phenomena such as weathering and volcanic eruptions have also been reported to significantly contribute to heavy metal pollution (Bradl *et al* 2002; Shallari *et al* 1998; Paul *et al* 2012). Industrial sources include metal processing in refineries, coal burning in power plants, petroleum combustion, nuclear power stations, and high tension lines, plastics, textiles, microelectronics, wood preservation, and paper processing plants (Aruti *et al* 2010).

Heavy metals: Any metal (or metalloid) species may be considered a "contaminant" if it occurs where it is unwanted, or in a form or concentration that causes a detrimental human or environmental effect. Metals/metalloids include lead (Pb), cadmium (Cd), mercury (Hg), arsenic (As), chromium (Cr), copper (Cu), selenium (Se), nickel (Ni), silver (Ag), and zinc (Zn). Other less common metallic contaminants include aluminium (Al), cesium (Cs), cobalt (Co), manganese (Mn), molybdenum (Mo), strontium (Sr), and uranium (U) (Singh *et al* 2011). Factors affecting the thresholds of dietary toxicity of heavy metal in soil-crop system include the soil type, which includes soil pH, organic matter content, clay mineral and other soil chemical and biochemical properties; and crop species or cultivars regulated by genetic basis for heavy metal transport and accumulation in plants and hence, influencing the thresholds for assessing dietary toxicity of heavy metals in the food chain (Ejaz *et al* 2007).

I. LEAD

Global Perspective of Lead Poisoning: Over 120 million people are overexposed to lead all over the world and 99 percent of the most serious cases are in the developing world and more than 51.3 per cent of children in Indian metros below 12 years of age have their blood lead levels above 10 ug/dl. Lead poisoning (also known as plumbism, colica Pictonum, saturnism, Devon colic, or painter's colic) is a medical condition in humans and other vertebrates caused by increased levels of the heavy metal lead in the body (Venkatesh *et al*, 2009)

Routes of exposure to lead include contaminated air, water, soil, food, and consumer products. Occupational exposure is a common cause of lead poisoning in adults. According to estimates made by the National Institute of Occupational Safety and Health (NIOSH), more than 3 million workers in the United States are potentially exposed to lead in the workplace (Staudinger 1998). The main threats of lead toxicity to children is lead paint that exists in homes and prevention of lead exposure can range from individual efforts (e.g. removing lead-containing items such as piping or blinds from the home) to nationwide policies (e.g. laws that ban lead in products, reduce allowable levels in water or soil, or provide for cleanup and mitigation of contaminated soil, etc.).

The increased lead in the body can be detected by the presence of changes in blood cells visible with a microscope and dense lines in the bones of children seen on X-ray, but the main tool for diagnosis is measurement of the blood lead level. When blood lead levels are recorded, the results indicate how much lead is circulating within the blood stream, not the amount being stored in the body. The Centers for Disease Control (US) has set the standard elevated blood lead level for adults to be 10 (µg/dl) of the whole blood. For children the number is set much lower at 5 (µg/dl) of blood as of 2012 down from a previous 10 (µg/dl).Children are especially prone to the health effects of lead and as a result, blood lead levels must be set lower and closely monitored if contamination is possible. The major treatments are removal of the source of lead by chelation therapy (CDC, 2012).

Classification: The amount of lead in the blood and tissues, as well as the time and course of exposure, determine toxicity. Lead poisoning may be acute (from intense exposure of short duration) or chronic (from repeat low-level exposure over a prolonged period), but the latter is much more common.

Acute poisoning: In acute poisoning, typical neurological signs are pain, muscle weakness, paraesthesia and, rarely, symptoms associated with encephalitis (Pearce, 2007). Abdominal pain, nausea, vomiting, diarrhea, and constipation are other acute symptoms. Lead's effects on the mouth include astringency and a metallic taste. Gastrointestinal problems, such as constipation, diarrhea, poor appetite, or weight loss, are common in acute poisoning. Absorption of large amounts of lead over a short time can cause shock (insufficient fluid in the circulatory system) due to loss of water from the gastrointestinal tract. Hemolysis (the rupture of red blood cells) due to acute poisoning can cause anemia and hemoglobin in the urine. Damage to kidneys can cause changes in urination such as decreased urine output. People who survive acute poisoning often go on to display symptoms of chronic poisoning (Brunton, 2007).

Chronic poisoning: Chronic poisoning usually presents with symptoms affecting multiple systems but is associated with three main types of symptoms including gastrointestinal, neuromuscular, and neurological. Central nervous system and neuromuscular symptoms usually result from intense exposure, while gastrointestinal symptoms usually result from exposure over longer periods. Signs of chronic exposure include loss of short-term memory or concentration, depression, nausea, abdominal pain, loss of coordination, and numbness and tingling in the extremities. Fatigue, problems with sleep, headaches, stupor, slurred speech, and anemia are also found in chronic lead poisoning (Pearce 2007, Brunton et al 2007, Kosnett 2007, Patrick 2006, James *et al* 2005, Rambousek 2008).

Effects on children: Children are more at risk for lead poisoning because their smaller bodies are in a continuous state of growth and development. Lead is absorbed at a faster rate compared to adults, which causes more physical harm than to older people. The signs and symptoms are loss of appetite, abdominal pain, vomiting, weight loss, constipation, anemia, kidney failure, irritability, lethargy, learning disabilities, and behavioral problems. Slow development of normal childhood behaviors, such as talking and use of words, and permanent mental retardation are both commonly seen (Cleveland *et al*, Meyer 2003, Bellinger *et al* 2008, Merrill *et al* 2007, Casarett *et al* 2007).

Complications: Lead can affects all the body's organ but predominantly, the nervous system, then bones and teeth, kidneys, cardiovascular and also reproductive system. Hearing loss and tooth decay are also the complications of lead poising. However, the health effects experienced by adults are similar to those in children, although the thresholds are generally higher (White *et al* 2007, Lanphear *et al* 2007).

Renal system: The toxic effect of lead causes nephropathy and may cause Fanconi syndrome, in which the proximal tubular function of the kidney is impaired (Rubin and Strayer 2008). Long-term exposure at levels lower than those that cause lead nephropathy have also been reported as nephrotoxic in patients from developed countries that had chronic kidney disease or were at risk because of hypertension or diabetes mellitus (Eking *et al* 2006). Even lower levels of lead in the blood can also damage the kidneys (Grant 2009).

Cardiovascular system: Evidence suggests lead exposure is associated with high blood pressure, and studies have also found connections between lead exposure and coronary heart disease, heart rate variability, and death from stroke, but this evidence is more limited (Navas-Acien *et al* 2007).

Reproductive system: Lead affects both the male and female reproductive systems. In men, when blood lead levels exceed 40 μg/dL, sperm count is reduced and changes occur in volume of sperm, their motility, and their morphology (Grant 2009). Elevated blood lead level in pregnant woman's can lead to miscarriage, prematurity, low birth weight and also the problems with development during childhood (Cleveland *et al* 2008). Lead is able to pass through the placenta and also into the breast milk (Dart *et al* 2004).

Nervous system: The brains of adults who were exposed to lead as children show decreased volume, especially in the prefrontal cortex, on MRI. Areas of volume loss are shown in color over a template of a normal brain (Cecil *et al* 2008). Lead affects the peripheral nervous system (especially motor nerves) and the central nervous system. Lead causes the axons of nerve cells to degenerate and lose their myelin coats (Dart *et al* 2004). Lead poisoning interferes with the normal development of a child's brain and nervous system; therefore children are at greater risk of lead neurotoxicity than adults (Sanders *et al* 2009).

Prevention: Testing kits are commercially available for detecting lead that turned into red showed the presence of lead (Rossi 2008 and Sanborn *et al* 2002). In most cases, lead poisoning is preventable by avoiding exposure to lead. Prevention strategies can be divided into individual measures, preventive medicines and public health measures (Guidotti and Ragain 2007).

II. MERCURY

Mercury is a toxic substance, which has no known function in human biochemistry or physiology and does not occur naturally in living organism. It occurs in three forms (elemental, inorganic salts, and organic compounds). Mercury contamination results from mining, smelting, and industrial discharges. Mercury in water can be converted by bacteria to organic mercury (more toxic) in fish. It can also be found in thermometers, dental amalgams, fluorescent light bulbs, disc batteries, electrical switches, folk remedies, chemistry sets and vaccines.

Health effects of mercury: It includes disruption of the nervous system, damage to brain functions, DNA damage and chromosomal damage, allergic reactions, resulting in skin rashes, tiredness and headaches and negative reproductive effects, such as sperm damage, birth defects and miscarriages (Horowitz *et al* 2002).

Elemental form of mercury: It is liquid at room temperature that volatizes readily. It rapidly distributes in body by vapor. At high concentrations, vapor inhalation produces acute necrotizing bronchitis, pneumonitis, and death and long term exposure affects CNS (ATSDR 1999).

Inorganic form of mercury: It is poorly absorbed in GI tract, but can be caustic. Acrodynia (Pink disease) occurs usually from dermal exposure. Maculopapular rash, swollen and painful extremities, peripheral neuropathy, hypertension, and renal tubular dysfunction are some other effects (Bjorklund 1995).

Organic form of mercury: It is lipid soluble and well absorbed via GI, lungs and skin. It can cross placenta and into breast milk. Toxicity occurs with long term exposure and effects the CNS. Signs progress from paresthesias to ataxia, followed by generalized weakness, visual and hearing impairment, tremor and muscle spasticity, and then coma and death (Clarkson and Magos 2006).

Diagnosis and Treatment: Diagnosis is made by history and physical and lab analysis. Inorganic mercury can be measured by 24 hour urine collection; organic mercury is measured by whole blood. The most important and effective treatment is to identify the source and end the exposure. Chelating agents (DMSA) may enhance inorganic mercury elimination (Ibrahim *et al* 2006).

Prevention: Roll onto a sheet of paper and place in airtight container. Use of vacuum cleaner should be avoided because it causes mercury to vaporize (unless it is a Hg Vac) Consultation with environmental cleaning company is advised with large spills. State advisories on public limit or avoid consumption of certain fish from specific bodies of water (Mozaffarian and Rimm 2006).

III. SELENIUM

Selenium (Greek- *selene* meaning "Moon") was discovered in 1817 by Jöns Jacob Berzelius, who noted the similarity of the new element to the previously-known tellurium (named for the Earth) by Berzelius, 1818. It is a nonmetal with properties that are intermediate between those of its periodic table column-adjacent chalcogen elements sulfur and tellurium. It rarely occurs in its elemental state in nature, or as pure ore compounds. Dietary selenium comes from nuts, cereals, meat, mushrooms, fish, and eggs. Brazil nuts are the richest ordinary dietary source (though this is soil-dependent, since the Brazil nut does not require high levels of the element for its own needs). In descending order of concentration, high levels are also found in kidney, tuna, crab, and lobster (Barclay *et al* 1995 and Mutanen, 1986).

Selenium Toxicity: Although selenium is an essential trace element, it is toxic if taken in excess. Exceeding the Tolerable Upper Intake Level of 400 micrograms per day can lead to selenosis. Symptoms of selenosis include a garlic odor on the breath, gastrointestinal disorders, and hair loss, sloughing of nails, fatigue, irritability, and neurological damage. Extreme cases of selenosis can result in cirrhosis of the liver, pulmonary edema, and death (Public Health Statement 2009).

IV. SILICON

It is a non metallic element with an atomic weight of 28. It is not found free in nature. Highest occurrence is found in the oats (4250 mg/kg wet weight). Lower levels are found in the animal sources. It is distributed in various organs of human body as follows:

Distribution: The human body contains approximately 7 grams of silicon, widely distributed in the tissues. High levels are present in bone, nails, tendons and the walls of the aorta, with nails containing the highest levels (up to 1500 mg/kg) and kidneys. Lower levels are present in red blood cells or serum (approximately 44 mg/kg for red cells and 20 mg/kg for bound silicic acid in plasma, in liver, spleen and lung. Silicon is found in breast milk.

Metabolism: The absorption is facilitated by aquaporins (a family of small (channel) proteins present in the intracellular membranes, where they facilitate the transport of water and/or small neutral solutes like urea, boric acid, silicic acid). In humans several aquaporins for OSA are identified. Average daily intakes of Si vary between 13 and 62 mg/d. Mean Si intakes in men (33 mg/d) are significantly higher than in women (25 mg/d). Silicon intake decreases with age. The dietary silicon intake is significantly correlated with urinary silicon excretion. (Bowen and Peggs 1984, Kayonga-Male and Jia 1999, Pennington 1990, Takizawa *et al* 1988).

Excretion: Silicon is predominantly and rapidly excreted in the urine, with smaller amounts being eliminated in the feces .The most sensitive test available to assess the reaction of white cells to silicon in the body is a lymphocyte chemical sensitivity (silicon) test. There is slow leakage (“bleeding”) of the silicone gel from the implants through the semi-permeable membrane envelope and also into and through the capsule that surrounds the implants. This is picked up by the microphages (scavenger cells) of our immune system and is broken down inside these cells which travel all over the body. The gel breaks down inside these cells, which travel all over the body. The gel breaks down into Silica and Silicon which causes an immune system deregulation. Thus, there are antibodies produced against the silicon and also against the silicon and protein complex (organ systems) so that you get autoimmune illness. As well, there is also damage that is not related to the immune system, because the silicone gel causes oxidants (damaging molecules) to be produced that directly damage our cell walls, DNA, and enzyme systems. All of this adds up to slowly-developing chronic debilitating illness affecting every organ system of the body (Expert Group on Vitamins and Minerals 2003).

CONCLUSION

Heavy metal toxicity is serious hazards as it may lead to health disorders like cardiovascular system, kidney stones; damage the neurons in the brains. By adopting this sound approach like boiling of water, kitchen gardening, lead free paints, consumption of non-contaminated sea foods, consumption of antioxidant rich foods help in combating these health problems.

REFERENCES

- Aruti A, Fernández-Olmo I, Irabien A. Evaluation of the contribution of local sources to trace metals levels in urban PM2.5 and PM10 in the Cantabria region (Northern Spain). J Environ Monit. 2010. 12(7): 1451-1458.
- ATSDR. Toxicological Profile for Mercury. Atlanta, GA: Agency for Toxic Substances and Disease Registry. http://www.atsdr.cdc.gov/toxprofiles/tp46.pdf.1999.
- Barclay, Margaret NI, MacPherson A, James D. Selenium content of a range of UK food. J food composition and analysis. 1995. 8 (4): 307-318.
- Bellinger DC. Very low lead exposures and children's neuro development. Current Opinion in Pediatrics. 2008. 20 (2): 172-7.
- Berzelius JJ, Lettre de M, Berzelius AM. Berthollet sur deux métaux nouveaux (Letter from Mr. Berzelius to Mr. Berthollet on two new metals). Annales de chimie et de physique. 1818. 7:199-206.
- Bjorklund G. Mercury and Acrodynia. Journal of Orthomolecular Medicine. 1995. 10:3
- Bowen HJM, Peggs A. Determination of the silicon content of food. Journal of Science Food and Agriculture. 1984. 35:1225-29.
- Bradl H. Heavy Metals in the Environment: Origin, Interaction, and Remediation Volume 6. 2002. London: Academic Press.
- Brunton LL, Goodman LS, Blumenthal D et al. Principles of toxicology. Goodman and Gilman's Manual of Pharmacology and Therapeutics. McGraw-Hill Professional. 2007. ISBN 0-07-144343-6.
- Casarett LJ, Klaassen CD, Doull J et al. Toxic effects of metals. Casarett and Doull's Toxicology: The Basic Science of Poisons (7th ed.). McGraw-Hill Professional. 2007. ISBN 0-07-147051-4.
- CDC. Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention. Centers for Disease Control and Prevention. 2012.
- Cecil KM, Brubaker CJ, Adler CM et al. Decreased Brain Volume in Adults with Childhood Lead Exposure. In Balmes, John. PLoS medicine. 2008. 5 (5): e112.
- Clarkson TW, Magos L. The toxicology of mercury and its chemical compounds. Crit Rev Toxicol. 2006. 36 (8): 609 62.
- Cleveland LM, Minter ML, Cobb KA et al. Lead hazards for pregnant women and children: part 1: immigrants and the poor shoulder most of the burden of lead exposure in this country. Part 1 of a two-part article details how exposure happens, whom it affects, and the harm it can do. The American journal of nursing. 2008. 108 (10): 40-9.
- Dart R C, Hurlbut K M, Boyer-Hassen LV. Lead. In Dart, RC. Medical Toxicology (3rd ed.). Lippincott Williams & Wilkins. 2004. SBN 0-7817-2845-2.
- Duruibe JO, Ogwuegbu, MOC, Egwurugwu, JN. Heavy metal pollution and human biotoxic effects. Int J Phy Sci. 2007. 2: 112-118.
- Ejaz ul I, Yang Xiao-e, He Zhen-li, Qaisar M. Assessing potential dietary

toxicity of heavy metals in selected vegetables and food crops. J Zhejiang Uni Sci B. 2007. 8: 1-13.

- Ekong EB, Jaar BG, Weaver VM. Lead-related nephrotoxicity: a review of the epidemiologic evidence. Kidney international. 2006. 70 (12): 2074-84.
- Expert Group on Vitamins and Minerals. 2003. Risk Assessments. Mineral-Silicon. Part B. pp 1-7.
- Grant LD. Lead and compounds. In Lippmann, M. Environmental Toxicants: Human Exposures and Their Health Effects (3rd ed.). Wiley-Interscience. 2009. ISBN 0-471-79335-3.
- Guidotti TL, Ragain L. 2007. Protecting children from toxic exposure: three strategies. Pediatric clinics of North America. 54 (2): 227-35.
- He ZL, Yang XE, Stoffella PJ. Trace elements in agro ecosystems and impacts on the environment. J Trace Elem Med Biol. 2005. 19(2-3):125-140.
- Horowitz Y, Greenberg D, Ling G, Lifshitz M. Acrodynia: a case report of two siblings. Arch Dis Child. 2002. 86 (6): 453.
- Ibrahim D, Froberg B, Wolf A, Rusyniak DE. Heavy metal poisoning: clinical presentations and pathophysiology. Clin Lab Med. 2006. 26 (1): 67-97.
- James W, Berger T, Elston D. Andrews' Diseases of the Skin: Clinical Dermatology. (10th ed.). 2005. Saunders. ISBN 0-7216-2921-0.
- Kayonga-Male H, Jia, X. Silicon bioavailability studies in young rapidly growing rats and turkeys fed semi-purified diets. A comparative study. Biological Trace Element Research. 1999. 67: 173-186.
- Kosnett MJ. Heavy metal intoxication and chelators. In Katzung, B.G. Basic and Clinical Pharmacology. McGraw-Hill Professional. 2007. ISBN 0-07-145153-6.
- Lanphear BP, Hornung R, Khoury et al. Low-Level Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis. Environmental health perspectives. 2005. 113 (7): 894-9.
- Merrill JC, Morton JJP, Soileau SD. Metals. In Hayes, AW. Principles and Methods of Toxicology (5th ed.). 2007. CRC Press. ISBN 0-8493-3778-X.
- Meyer PA, McGeehin MA, Falk H. A global approach to childhood lead poisoning prevention". International journal of hygiene and environmental health. 2003. 206 (4-5): 363-69.
- Mozaffarian D, Rimm E B, 2006. Fish intake, contaminants, and human health: evaluating the risks and the benefits. JAMA. 296 (15):1885-99.
- Mutanen M. Bio-availability of selenium. Ann Clin Res. 1986. 18: 48-54.
- Navas-Acien A, Guallar E, Silbergeld EK, Rothenberg SJ. Lead Exposure and Cardiovascular Disease-A Systematic Review. Environmental health perspectives. 2006. 115 (3): 472-82.
- Nolan K. Copper Toxicity Syndrome, J. Orthomol. Psychiatry. 2003. 12(4): 270 - 282.
- Nriagu JO. A global assessment of natural sources of atmospheric trace metals. Nature. 1989. 338:47-49.
- Patrick L. Lead toxicity, a review of the literature. Part 1: Exposure, evaluation, and treatment". Alternative medicine review: A journal of clinical therapeutic. 2006. 11 (1): 2-22.
- Paul BT, Clement GY, Patolla AK, Dwayne JS. Heavy Metals Toxicity and the Environment. 2012. PMCID: PMC4144270. EXS. 2012; 101: 133–164. doi: 10.1007/978-3-7643-8340-4_6
- Pearce JM. Burton's line in lead poisoning. European neurology. 2007. 57(2): 118-9.
- Pennington JAT. Silicon in foods and diets. Food Additives and Contaminants. 1990. 8: 97-118.
- Rambousek AJ. The symptoms and treatment of industrial poisoning. Industrial Poisoning from Fumes, Gases, and Poisons of Manufacturing Processes. 2008. ISBN 1-4086-7025-9.
- Rossi E. Low Level Environmental Lead Exposure - A Continuing

Challenge. The Clinical biochemist. Reviews / Australian Association of Clinical Biochemists. 2008. 29 (2): 63-70.

- Rubin R, Strayer DS. Environmental and nutritional pathology. Rubin's Pathology: Clinicopathologic Foundations of Medicine (5th ed.). Lippincott Williams & Wilkins. 2008. ISBN 0-7817-9516-8.
- Sanborn MD, Abelsohn A, Campbell M, Weir E. Identifying and managing adverse environmental health effects: 3. Lead exposure. Canadian Medical Association Journal. 2002. 166 (10): 1287-92.
- Sanders T, Liu Y, Buchner V, Tchounwou P B. Neurotoxic Effects and Biomarkers of Lead Exposure: A Review. Reviews on environmental health. 2009. 24 (1): 15-45.
- Shallari S, Schwartz C, Hasko A, Morel JL. Heavy metals in soils and plants of serpentine and industrial sites of Albania. Sci Total Environ. 1998. 19209:133 142. [PubMed]
- Singh R, Gautam N, Mishra A. Gupta R Heavy metals and living systems: An overview. Indian J Pharm. 2011. 43(3): 246–253.
- Staudinger KC, Roth VS. Occupational lead poisoning. Am Fam Physician. 1998. 57 (4): 719-26.
- Takizawa Y, Hirasawa F, Noritomo E, Aida M, Tsunoda H, Uesugi S. Oral ingestion of syloid tomice and rats and its chronic toxicity and carcinogenicity. Acta Medica Bio. 1988. 36: 27-56.
- White LD, Cory SDA, Gilbert ME, et al. New and evolving concepts in the neurotoxicology of lead". Toxicology and applied pharmacology. 2007. 225 (1): 1-27.
- Yang G, Zhou R. Further Observations on the Human Maximum Safe Dietary Selenium Intake in a Seleniferous Area of China". Journal of trace elements and electrolytes in health and disease. 1994. 8: 159-65.
- Young RA. Toxicity Profiles: Toxicity Summary for Cadmium, Risk Assessment Information System, RAIS, University of Tennessee (rais.ornl.gov/tox/profiles/cadmium.shtml). 2005.

How to cite this article: Bathla S, Jain T. Heavy metals toxicity. Int J Health Sci Res. 2016; 6(5):361-368.

****