

Release of Heavy Metals from Industrial Waste and E-Waste Burning and Its Effect on Human Health and Environment

Aprajita Singh^{1,2}, Raina Pal¹, Charu Gangwar^{1,2}, Akhil Gupta^{1s}, Anamika Tripathi^{1*}

¹ Pollution Ecology Research Laboratory, Department of Botany, Hindu College, Moradabad, India

² School of Biotechnology, IFTM University Moradabad, India

Abstract:

Electronic waste is the discarded electrical and electronic devices. It is the fastest growing waste in the today's world than any other waste stream. Heavy metals are part of manufacturing process of many common household items, such as batteries, electronic, electroplated metal parts, textile dyes, steel etc. Burning of the E-waste leads to the release of the harmful and toxic metals like lead, chromium, cadmium, mercury, arsenic, and barium, adverse health effects of heavy or toxic metals have been known for long time, exposure to these metals continues and is even increasing in some parts of the world, particularly in developing countries. Therefore prolonged periods of chronic exposure to trace close of toxic metals cause harmful effects on the human health.

Keywords: E waste, Human Health, Heavy Metals, Electronic and Electrical devices.

I. INTRODUCTION

Increasing in industrial and e-waste leads to the increasing of the pollution, as waste from the industries are emitted to the air or surface waters, industrial pollution and waste encompass the full range of unwanted substances. Therefore on the other side as the e-waste include the discarded electrical and electronic wastes, basically the waste from the IT sectors like computer, laptops, printers, scanner etc i.e. the Grey line. Burning of this electrical and electronic waste leads to the release of various toxic and harmful metals in the air and it also affect the soil as well where the burning is taking place. These substances are harmful to human health and environment which are getting emitted from industrial waste and e-waste burning.

Sustained exposure to ambient air pollution leads to increased rates of respiratory disease, chronic obstructive pulmonary disorder, and lung cancer. Thus air pollution acts as a catalyst for rising healthcare costs, placing an ever increasing stress on an already under-funded and ill equipped health system.

Combustion from burning e-waste creates fine particulate matter. Exposure to PM (Particulate Matter) is responsible for about "3 percent of adult cardiopulmonary disease mortality; about 5 percent of trachea, bronchus, and lung cancer mortality; and about 1 percent of mortality in children from acute respiratory infection in urban areas worldwide", amounting to 800,000 premature deaths and over 6 million lost life years (Cohen A. and Anderson, 2005). Also noted that since only the mortality aspects of exposure to PM were considered, the overall impact could actually be underestimated. Studies have also shown that increasing atmospheric concentrations of PM have a direct effect on mortality and morbidity.

The toxic effect of these metals depends on exposure level, route of exposure, period of exposure, chemical form, bioavailability, as well as on the individual age, nutritional and health status. Passing through human body, various factors such as absorption, distribution, metabolism and excretion influence their toxicity.

Most of the heavy binds to sulfhydryl group thus inhibiting enzyme activity, disrupting cellular transport and causing changes in protein function. The toxicity of heavy metal includes the blocking of active groups of important functional molecule example enzymes, polynucleotides, transport systems for essential nutrients and ions and substitution of essential ion from cellular sites (Kakkar and Jaffry, 2004).

Heavy metals in the past were commonly defined as those having a specific density of more than 5g/cm³ but in recent time, the definition s based on the chemical properties and toxicity (John 2002, Jarup 2003).

Health risk assessment of heavy metals or any chemical require an accurate exposure assessment and determination of quantitative relationship between internal dose of metal and adverse health effects and known dose response relationship. To assess magnitude of human exposure to toxic heavy metal. Heavy metals are reported to be tumor promoters (Rhee et al., 2000). They are thought to promote the mutagenic effects of DNA damaging agents, while alone may not themselves be mutagenic (Goyer, 1996)

The International Agency for Research on Cancer (IARC) has recently classified outdoor air pollution as a Class-I carcinogen, linking it to over 220,000 lung cancer deaths per annum globally (Straif et al, 2013).

Hereafter, chronic mostly non-carcinogenic toxic effects of several metals including arsenic, cadmium, chromium, nickel, zinc, lead, mercury and barium. The current knowledge in the field of metal biochemistry of oxidation stress indicates that metal induced and metal enhanced formation of free radicals and other reactive species can be regarded as a common factor in determining metal induced toxicity together with their carcinogenicity (Jamova and Volka 2011). Recently, more attention and concern has been given to metal compounds that have toxic effect at low level of exposure than those produce overt clinical and pathological signs and symptoms (Kalia and Flora 2005)

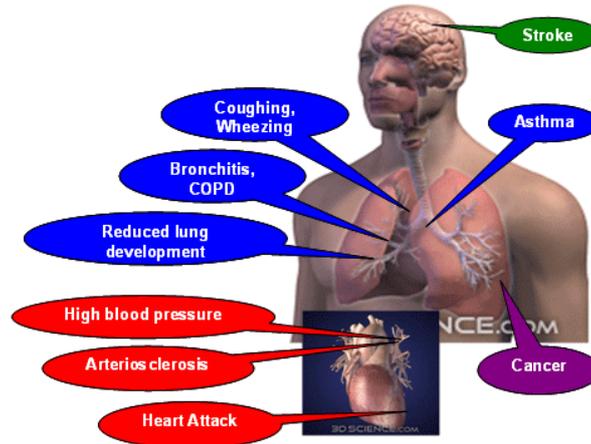
Some metals can interfere with hormones biosyntheses, secretion and metabolism that may lead to adverse health outcomes such as reproductive disorders, thyroid and neuro-developmental and endocrine related cancer. The existing evidences of a

relationship between exposure to metal and hormones alterations related to exposure to metals among the general population (Meeker et al., 2010). A chemical is considered to be mutagenic if it is capable of inducing heritable changes in the genotype of a cell as consequence of alteration or loss of genes, chromosomes or parts of chromosomes.

Various heavy metals released from the industrial and e-waste burning which have many harmful and toxic effects on the human body as well as on the environment which has been discussed below.

The problem of e-waste has become an immediate and long term concern as its unregulated accumulation and recycling can lead to major environmental problems endangering human health. E-waste is much more hazardous than many other municipal wastes because electronic gadgets contain thousands of components made of deadly chemicals and metals. It has been known that the pollutants of electronic wastes can leads to severe pollution to the environment. It has been reported that about 50% to 80% of e-waste from the developed countries are exported to Asia and Africa. Electronic waste not only produce enormous environmental pollution but also can bring about the toxic or genotoxic effects on the human body, threatening the health of current residents and the future generations living in the local environment.

II. EFFECT OF HEAVY METALS ON HUMAN HEALTH AND ENVIRONMENT



LEAD (Pb)

Effect on human health: lead is naturally occurring element that can be harmful to humans when ingested or inhaled; children are more susceptible to lead than adults due to higher gastrointestinal uptake and the permeable blood brain barrier (Jarup, 2003). It induces brain disorders; lead may also cause hypertension, kidney damage, anemia and adverse reproductive outcomes (Kakkar and Jaffery, 2004). EPA has determination that lead is a probable human carcinogen. Lead can affect every organ every organ and system in the body. Long-term exposure of adults can result in decreased performance in some tests that measures functions of the nervous systems; weakness in fingers, wrists or ankle, small increases in blood pressure and anemia. In pregnant women, high levels of exposure to lead may cause miscarriage. High level exposure in men can damage the organs responsible for sperm production. Adverse effect of Pb occurs at lower threshold levels than in adults. Pb has effects on erythropoiesis and haem biosynthesis. In young children hormonal imbalance of metabolites of vitamins D, namely 1,25-dihydroxy-vitamin D, drop in IQ(52-54). Children may be affected by behavioral disturbances, learning and concentration difficulties (Haileslassie and Gabremedhin 2015).

Table: Toxic Effects of Lead

EFFECTS OF LEAD
Hyperactivity
Headaches
Hearing loss
Loss of synaptic plasticity
Neuropathy
Loss of dermal sensitivity
Damage to heme formation pathway
Slow growth
Learning disabilities
Genotoxicity

Effect on environment: The main sources of lead entering an ecosystem are atmospheric lead paint chips, used ammunition, fertilizers and pesticides and lead-acid batteries, e-waste burning especially PCBs (printed circuit boards) or other industrial products. The transport and distribution of lead from major emission sources, both fixed and mobile are mainly through air. Lundsledt 2011 reported that lead, a problematic metal, is highly abundant in e-waste. Pb accumulated in the environment and produce both high acute and chronic effects on biological system (i. e. plants, animal and microorganism). The threshold lead level allowed for fruits and small fruits are .10 and .20 mg/kg, respectively (EC 2006). Dissolve chemical form of lead are extremely toxic in the aquatic environment, when present in the high concentration (Huang J. 2014). Lead can remain in

dangerous form for a very long time. Lead emission to ambient air due to e-waste burning have further polluted our environment.

MERCURY [Hg]

Effect on human health: EPA (2005) has determined that mercuric chloride and methylmercury are possible human carcinogen. The nervous system is very sensitive to all forms of mercury. Exposure to high level can permanently damage the brain, kidneys, and developing fetus. Effect on brain functioning may result in the irritability, shyness, tumors, change in vision and memory problem. Short term exposure leads to the lung damage, increase in blood pressure or heart rate, skin rashes and eye irritation. A number of studies have shown that mercury binds to the DNA and can cause strand breaks in vitro (IPCS, 2003), with weak genotoxic activity but without causing point mutations (WHO, 2005) and with negative effect on sperm cells. The health effect from mercury, in particular methylmercury, upon infants and children depend on the dose, with severe symptoms presenting with exposure to doses of 100 ug/kg/day, mild symptoms with greater than 10 ug/kg/day, and sub-clinical symptoms with less than 1 ug/kg/day (Hymen M, 2004). Some symptoms include late development in walking and talking, decreased performance, neurological development disorders, intellectual disorders and autism (Weiss, 2000). In adult's peripheral neuropathy, Parkinsonia symptoms, tremor and ataxia, impaired hearing, tunnel vision, headache, fatigue, impaired sexual function, and depression (Kakkar and Jaffery, 2004). Continuous exposure conditions to elemental Hg can lead to its accumulation in the thyroid. The acute exposure to elemental Hg vapors can cause "pink disease" or acrodynia It is also responsible for minimata disease.

Table: Mercury Toxic Effects

Organs	Effects
Reproductive system	Impaired sexual functions, teratogenic effect in reproduction.
Kidney	Proteinuria renal syndrome, acute renal failure
Respiratory system	Irritation, respiratory failure
Skin	Allergic dermatitis, chelitis, gingivitis, stomatitis
Visual system	Changes in vision
Nervous system	Deafness, hyperactivity disorders, inability to concentrate, insomnia, memory impairment, Parkinson neuropathy.

Effect on environment : It builds up in the environment by biomagnifications. Release of mercury to air, water and through products may cause adverse impacts on environment and has significant risk to aquatic life and ultimately to human beings due to contaminated air, water and even food chain. The average daily intake of Hg from food is in the range 2-20 ug. However concentration of the mercury is increasing in the environment due to many activities through industrial smoke, e-waste burning, fossil fuel burning, mining, smelting. All mercury that is released in the environment will eventually end up in soil or surface water.

CADMIUM [Cd]

Effect on human health: These compounds are known Human Carcinogens determined by EPA. Smokers get exposed to significantly higher cadmium level than non-smoker (Jarup et al., 1998). Severe damage to lungs may cause through breathing high levels of cadmium. Long term exposure to lower level leads to the kidney disorders, lung damage and fragile bones. Observed alteration of DNA, as consequences of cadmium applications in experimental models of mammalian cell cultures, higher plants and intact animals, include decreased fidelity of DNA synthesis, microbial DNA repair, gene mutations and chromosomal abnormalities. These positive results were often weak and seen at high concentrations that also caused cytotoxicity. However, cadmium does not appear to possess significant genotoxic potential via the oral route (WHO, 2011). The mechanisms of carcinogenesis of cadmium are complex. They include modulation of gene expression and signal transduction, interference with enzymes of the cellular antioxidants systems and generation of reactive oxygen species and DNA damage, the inhibition of different types of DNA repair and the induction of apoptosis, role in the disruption of E-cadherin-mediated cell-cell adhesion (Malaguarnera, 2012, Satarug and Moore, 2012).

Cadmium targets various transporters thus cadmium is a toxic metal, omnipresent in the environment brush-border and basolateral membrane (Sabolic et al., 2012). High exposure of cadmium in human caused the itai- itai disease (Aoshima 2012).

Effect on environment: Cadmium and its compounds are highly toxic. Natural and anthropogenic sources have increased cadmium levels in biosphere as a consequence of extensive electronic waste burning and industrialization leads to environmental pollution. Cadmium including industrial emission or e-waste burning may lead to the contamination of soil, and to increase cadmium uptake process of soil cadmium by plants is enhanced at low pH C(Hailesslassie and Gabremedhin 2015). Cadmium occurs in almost all soils, surface waters and plants and is considered a potential threat to wildlife species. When not properly recycled it can leach out in soil, harming microorganism and disrupting the soil ecosystem (Sharma et al 2012). Occurrence of cadmium in drinking water is usually less than 1ug/l and daily oral intake is 10-35 ug, the guideline value of WHO for cadmium on drinking water is 0.003 mg/l (WHO, 2011)

CHROMIUM [Cr]

Effect on human health: People can be exposed to chromium through breathing, eating or drinking and through skin contact with chromium and chromium compounds. According to EPA inhaling high levels of the chromium leads to the irritation in the nose and breathing problems, such as asthma, cough, and shortness of breath or wheezing. Skin contact can cause skin ulcers; allergic reactions consisting of severe redness and swelling of the skin have been noted. Long term exposure can cause damage to liver, kidney, circulatory and nerve tissue as well as skin irritation. Chromium is essential for normal glucose metabolism. Chromium is also a human carcinogen via inhalation and sufficient evidence exists for its causal relationship with lung and nasal cavity cancer in humans (Quievryn et al., 2002). Recently China found increased risk for stomach cancer mortality associated with high concentration of chromium in drinking water (Beaumont et al., 2008; Smith and Steinmaus, 2009). Observed in animals hepatic and renal failure, sperm abnormalities and damage to male reproductive systems (Jomova and Valko, 2011).

Effect on environment: It is found naturally in rocks, plants, soil and volcanic dust, human and animals. Chromium being released to the environment by leakage, poor storage, chemical, leather or inadequate industrial waste disposal practices (EPA, 2010; Sciacca and Oliveri, 2009). These application will increase chromium level in water. Although the level of chromium in air, and water is generally very low. The guide lines value of WHO for total chromium in drinking water is 0.05 mg/l (WHO, 2011). Most of the chromium in air will eventually settle down and end up in water and soil. Chromium in soil will strongly attach to soil particles and as a result it will not move towards the ground water. Crops contains system that arrange the chromium uptake to be low enough not to cause any harm.

BARIUM [Ba]

Effect on human health: Barium is not considered to be the essential element for human nutrition, short term exposure can cause vomiting, abdominal cramps, diarrhea, difficulties in breathing, increased or decreased blood pressure, muscle weakness. Large amount of barium intake can cause, high blood pressure, change in heart rhythm or paralysis and possibly death by EPA(2005). At high concentration, barium causes vasoconstriction by its direct stimulation of arterial muscle, paralysis chocking of nervous system. Depending on the dose and solubility of the barium salt, death may occur in a few hours or a few days. The acute toxic oral dose is between 3 to 4g (WHO, 2011)

There is no evidence that barium is carcinogenic or mutagenic. Barium has been shown to cause nephropathy in vitro, but toxicology end point of greatest concern to human appears to be its potential to cause hypertension (WHO, 2011).

Effect on environment: A barium level in environment is very low. The air that most people breathe contains about 0.0015 parts of Ba per billion parts of air. Basically industrial waste is responsible for the pollution or increasing in the barium concentration in the air (ASTDR, 2007). Some barium compounds that are release during industrial processes dissolve easily in water and are found in lake, river and the stream. When fish and other aquatic organisms absorb the barium compounds and barium will accumulate in their bodies. The mean daily intake of the barium from food, water and air is estimated to be slightly more than 1 mg/day. However, where barium levels in water are high, drinking- water may contribute significantly to barium intake (WHO, 2011). Barium compound that are persistent usually remain in soil surfaces or in the sediment of soil present in water.

ARSENIC [As]

Effect on human health: Arsenic is toxic to both plants and animals and inorganic arsenicals are proven carcinogens in humans (Ng, 2005; Murcott, 2012).the toxicity of arsenic to human health ranges from skin lesions to cancer of the brain, liver, kidney, and stomach (Smith et al., 1992). Short term exposure can cause nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of “pins and needle” in hands and feet. Long term exposure to arsenic may lead to darkening of the skin and the appearance of small “corns” or “warts” on the palms by EPA. Arsenic induced also chromosomal aberrations, including micronuclei and aneuploidy and sister-chromatid exchanges, enhances oxidative stress and influences the production of nitrogen monoxide. Methylated and dimethylated forms, also exhibits genotoxicity at higher exposure levels. Arsenic does not cause DNA damage directly, but inhibits DNA synthesis and repair and also affects DNA methylation in tumor suppressor genes (Brown, 2008). Generally inorganic arsenic species are more toxic than organic form to living organism, including humans and other animals (Goessler and Kuehnelt, 2002; Ng, 2005). Ingestion of arsenic trioxide of 70-80 mg has been reported to be fatal for humans (Vallee et al., 1960).

Effect on environment: Found naturally in small concentration in soil, water, rock and air. Arsenic released in the atmosphere through various human activities like industrial smoke emission, burning of electric and electronic equipments leads to the emission of large amount of heavy and toxic metals in the air which causes pollution in high amount. It also comes from natural resources like volcanoes, microorganisms, anthropogenic sources, from pesticides. Arsenic is an enhancer of alteration of genetic material of fish and of all animals that eat polluted fish (ATSDR, 2007).

NICKEL [Ni]

Effect on human health: It affects the immune and reproductive systems and developing organisms. Inconsistent results of animal studies have been reported for the reproductive toxicity (decreased sperm motility and count, sperm abnormalities decreased fertility) and developmental effects (fetal loss and decreased survival) of nickel, human data are very cleared (Mitchell et al., 2011; ASTDR, 2005). At cellular level, it increases oxidative products and proteins (Mitchell et al., 2005). Uptake of nickel in large amount has many consequences such as heart disorders, lung embolism, hypoglycemia, teratogenic effects, asthma and chronic bronchitis, respiratory failure, allergic reactions from jewellery, lung disorders. While the

deficiency of nickel have been linked with hyperglycemia, depression, reproductive failure, sinus congestion, fatigue and growth problem in humans.

Effect on environment: Nickel is the compound that occurs in the environment only at trace levels. Food contains very small concentration of the nickel. It is greatly toxic at comparatively low amount. Heavy metal poisonousness is the product of multifaceted of chief noxious ion with other vital or non essential ions. The metal can be a source of decrease hydrolysis product viz α - amylase, phosphatase, RNA and protein. It disturb the enzyme activities by substituting metal ions from the metallo- enzyme and prevent various physiological developments of plants (Agrawal, 1999). Nickel doesn't have any harmful effect on the environment.

III. CONCLUSION

It is concluded on experimental studies that the emission of various harmful metals from industrial and e-waste burning leads to the various health problem in the human beings and also the some drastic effects on environment. These are not the good sign for the future, as industries are increasing day by day and so e-waste. There is not much action is taken to stop the illegal e-waste burning and to stop disposing the industrial pollution in the water and air. Thus chances of cancer even increasing, people are dying due to various respiratory diseases like asthma, bronchitis etc. animals also getting effected manly water live, due to dispose of waste in the water streams. This review on toxic metals shows that these metals from e-waste industry and e-waste buring sites can accumulate and persist in soil and air are mainly carcinogenic and very harmful to human health and environment so the necessary step should be taken.

REFERENCES

- [1] Agrawal S. K., (1999). Studies on the effect of the auto exhaust emission on the mitragynapa trifolia. Ajmer India: MDS University; Master thesis.
- [2] Agency for toxic substances and diseases registry (ATSDR). (2005). Toxicological Profile for Nickel (update). Atlanta, GA: U.S. Department of public Health and Human Services, Public Health Services.
- [3] ATSDR., (1999). Tox FAQs. Mercury. Agency for Toxic Substances and Diseases Registered.
- [4] Akerson A., Lundh T., Vahtir M., Bjillerup P., Lidfeldt J., Nerbrand C., Sanesier G., Stromberg U., Skerfving S. (2005). Tubular and glomerular kidney effects in Swedish Women with low environmental cadmium exposure. Environ Health Prespect .
- [5] Aoshima K. 2012. Itai- Itai disease: cadmium induced renal tubular osteomalacial. Nihon Eiseigaku Zasshi. 67 (4): 455-63.
- [6] ASTDR (2007). Toxicological profile for baruins and baruins compounds. U. S. Department of Health and Human Services.
- [7] Beaumont J.J., Sedman R.M., Reynolds S.D., Sherman C.D., Li L.H., Howd R.A., Sandy M.S., Zeise L., Alexeeff G.V. (2008). Cancer mortality in Chinese population exposed to hexavalent chromium in drinking water. Epidemiology, 19(1):12-23.
- [8] Brown J. P. (2008). Risk assessment for arsenic in drinking water. In Howd R. A. and Fan A.M. (EDs) , Risk assessment for chemical in drinking water. New York: Wiley and Sons; pp. 228-30.
- [9] Cohen A. and Anderson R. H. (2005). The global burden of disease due to outdoor air pollution. J. Toxicol. Environ. Hlth.
- [10] EPA. (1998). Toxicological Review of Baruins and Compounds. U. S. Environmental Agency.
- [11] EPA. (2010). IRIS Toxicological review of hexavalent chromium (external review draft). <http://cf/arb.epa.gov/ncea/iris/drafts/ricodisplay.cfm?derd=221433>.
- [12] EC (2006) Commission of the European communities. Commision Regulation (EC) No. 1881/2006. Regulation of setting maximum levels for certain contaminants in foodstuff. Official J. European Union L364-5/L364-24.
- [13] EPA-U.S. Environmental Protection Agency (2005). Cancer guideline.
- [14] Goessler W., Kuehnelt D. (2002). Analytical methods for the determination of arsenic and arsenic compounds in the environment. Environmental chemistry of Arsenic, 27-50.
- [15] Huang J, Nkrumah P T, Anim D O and Mensah (2014). E-waste disposal effect on the aquatic environment: Accra, Ghana. Review of environmental contamination and toxicology., Review of environmental contamination and toxicology 229. DOI 10.1007/978-3-319-03777-6-2.
- [16] Hyman M., (2004). The impact of mercury on human health and the environment. Alternative Therapies; 6(10): 70-5.
- [17] Hailesslassie T. and Gabremedhin K, (2015). Hazards of Heavy Metal Contamintion in Ground Water.
- [18] International Journal of Toxicology enhancement and Emerging Engineering Research, vol. 3, issue 02 ISSN 2347-4289.
- [19] Internatoinal Agency for the Research on Cancer (IARC). (2004). Inorganic and organic lead compounds. IARC monographs on the evaluation of carcinogenic risks to human. Vol. 87. Lyon
- [20] IRAC (1993). Cadmium and cadmium compounds. In: Beryllium, Cadmium, Mercury and exposure in glass manufacturing industries. IRAC mono graph on the evolution of carcinogenic risk to humans, vol. 58. Lyon: International Agency on Research on cancer; 119-237.
- [21] IPCS. (2003). Concise International Chemical Assessment Document so: elemental mercury and inorganic mercury compounds: Human Health Aspect Geneva, World Health Organisation and International Programme on Chemical Safety.

- [22] Jarup L., Berg Lund M., Elinder C. G., Nordbug G., Vahter M., (1998). Health effect of cadmium exposure: a review of the literature and a risk estimate. *Scand. J. Work Environ Health*; 24 (Supp L1) :1-51.
- [23] Jarup L. (2003). Hazards of heavy metal contamination. *British Medical Bulletin*; 68:167-82.
- [24] Jomovo K., Valko M., (2011). Hazards Of heavy metal contamination. *British Medical Bulletin*. 68: 167-82.
- [25] Joseph. (2003). Hazards of heavy metal contamination. *British Medical Bulletin*. 68: 167-82.
- [26] John H D, (2002). "Heavy Metals" a Meaning Term? (IUPAC Technical Report). *Pure and Applied Chemistry* pp. 793-807.
- [27] Kalia K., Flora S. J. (2005). Strategies for safe and effective therapeutic measures for chronic arsenic and lead poisoning. *J. Occup Health*: 47. 1-21.
- [28] Kakker P., Jaffery F. N. (2004). Biological markers for metals toxicity. *Environ. Toxicol. Pharmacol.* 19: 335-49.
- [29] Lundsledt s., (2011). Recycling and disposal of electronic waste: health hazards and health impacts. Swedish Environmental Protection Agency Report 6417. <http://www.naturvardsverket.se/publicationer/6400/978-91-620-6417-4.pdf>. Assessed 23 July 2012
- [30] Malaguarnera M. (2012). Cadmium and cancer risk. ICS 2012, Cadmium Symposium Sassari, Italy, Abstract book: pp, 39.
- [31] Meekar J.D., Rossano M. G., ProtasmB., Padmanabhan V., Diamond M. P., Puscheck E., Daly D., Paneth N., Wirth J. J. (2010). Environmental exposure to metals and male reproductive hormones: Circulating testosterone is inversely associated with blood molybdenum. *Fertil Steril*; 93 (1): 130.
- [32] Murcott S. (2012). Arsenic contamination in the world an international sourcebook IWA monographs. ISBN: 9781780400389, pages: 344.
- [33] Mitchell E., Frisbie S., Sarkar B. (2011). Exposure to multiple metals from groundwater a global crisis: Geology, Climate change, health effects, testing, and mitigation. *Metalomics*, 3:874-908.
- [34] Ng J.C. (2005). Environment contamination of arsenic and its toxicological impact on humans. *Environmental chemistry*, 2(3): 146-60.
- [35] Quievryn G., Messer J., Zhitkovich A. (2002). Carcinogenic chromium(VI) induces cross-linking of vitamin C to DNA in vitro and in human lung A549 cells. *Biochemistry*; 41:3156-67.
- [36] Satarug S., Moore MR. (2012). Emerging role of cadmium and heme oxygenase in type 2 diabetes and cancer susceptibility. *Tohoku J. Expt. Med.* 228: 267-288
- [37] Sciacca S., Oliveri C. G., (2009). Mutagens and carcinogens in drinking water. *Mediterr J. Nutr. Mateb*, 2: 157-62.
- [38] Sharma P., Fulekar M.H., Bhawan P. (2012). E-waste a challenge for tomorrow. *Research J. Of Recent Sciences* vol. 1 (3), 86-93.
- [39] Smith A. H., Sturimaus C. M., (2009). Health effect of arsenic and chromium in drinking water: Recent Human Findings. *Annu. Rev. Public Health*. 29 (30) : 107-22
- [40] Smith A.H., Hopenhayn-Rich C., Bates M.N., Goeden H.M., Hertz-Picciotto I., Duggan H.M., Wood R., Kosnett M.J., Smith M.T. (1992). Cancer risks from arsenic in drinking water. *Environmental Health Perspectives*, 97: 259-67.
- [41] Straif, K., Cohen A. and J. Samet, "Air Pollution and Cancer," International Agency for Research on Cancer, 2013
- [42] Thompson J., Bonnigan J. (2008). Cadmium: toxic effect on the reproductive system and the embryo. *Reprod. Toxicol.* 25(3) : 304-15.
- [43] Vallee B.L., Ulmer D.D., Wacker W.E.C. (1960). Arsenic toxicology and biochemistry. *Arch. Ind. Health*, 21:132-51.
- [44] Weiss B., (2000). Vulnerability of children and the developing brains to neurotoxic hazards. *Environ. Health Perspect*; 108: 375-81.
- [45] WHO (World Health Organisation), (1995). Lead. *Environmental Health Criteria*, vol. 165. Geneva, Switzerland
- [46] WHO . (2011) guidelines for Drinking water Quality. 4th ed. WHO, Geneva, Switzerland. ISBN 978 92 4 154815 1.
- [47] WHO (2005). Mercury in drinking water. Background document for development of WHO guidelines for drinking water quality. Geneva.