

Heavy Metal Content in Water of Bhavan's College Lake of Andheri, Mumbai

P. U. Singare^{1,*}, M. S. Talpade², D. V. Dagli¹, V. G. Bhawe¹

¹Department of Chemistry, Bhavan's College, Munshi Nagar, Andheri (West), Mumbai 400058, India.

²Department of Chemistry, Shri. Jagdishprasad Jhabarmal Tibrewala University, Jhunjhunu, Rajasthan 333001, India

*E-mail address: pravinsingare@gmail.com

ABSTRACT

The present research work deals with the quantification of toxic heavy metals in the water samples collected from Lake of the Bhavan's College campus of Andheri, Mumbai. The results of the present investigation indicates that yearly average concentration of toxic heavy metals like Pb, Zn, Cr, Fe and Hg was 0.16, 5.56, 2.09, 5.19 and 0.02 ppm respectively which were very much above their permissible limits set for inland surface water, while the yearly average concentration of Ni was found to be 2.76 ppm which was close to the maximum limit of 3.0 ppm. The results of the present investigation points out the need to implement common objectives, compatible policies and programs for improvement in treatment facilities for the treatment of discharged sewage and laboratory effluents.

Keywords: Toxic Heavy Metals; Lake water; AAS; Cold Vapour Technique; Hydride generation; Health Hazards; Water Pollution; Bhavan's College; Andheri; Mumbai.

1. INTRODUCTION

Heavy metal pollution is an ever increasing problem of our creeks, lakes and rivers [1-12]. These toxic heavy metals entering in aquatic environment are adsorbed onto particulate matter, although they can form free metal ions and soluble complexes that are available for uptake by biological organisms [13, 14]. The increase in residue levels of heavy metal content in water, sediments and biota has resulted in decreased productivity and increase in exposure of humans to harmful substances [15]. Many of these metals tend to remain in the ecosystem and eventually move from one compartment to the other within the food chain. Food chain contamination by heavy metals has become a burning issue in recent years because of their potential accumulation in biosystems through contaminated water, soil/sediment and air [16-19]. Therefore a better understanding of heavy metal sources, their accumulation in the soil and the effect of their presence in water and soil seem to be

particularly important issues of present-day research on risk assessments [20]. Incidence of heavy metal accumulation in fish, oysters, sediments and other components of aquatic ecosystems have been reported globally [8, 9, 21-23]. Today it is realized that solution to such environmental problem can only be achieved through comprehensive, systematic and sustained approach. Hence in the present investigation, efforts are made to quantify the accumulation of toxic heavy metals in water of Bhavan's College Lake of Andheri city of Mumbai, India.

The study was carried out with an objective to generate the pollution load data from regular scientific study so as to implement compatible policies and programs to gauge the extent of pollution due to toxic heavy metals in the lake water, to evaluate effectiveness of pollution control measures already in existence, to assess nature and extent of pollution control needed, and for rational planning of pollution control strategies and their prioritization.

2. MATERIALS AND METHODS

2. 1. Area of study

The study was performed at Bhavan's College Lake, located at Andheri, Mumbai city which is one of the most heavily populated and industrialized cities of Maharashtra. It is situated between 18° 96' north latitude and 72° 81' east longitude. The average elevation ranges from 10 meters to 15 meters. The major portion of the city is at the sea level while the maximum height of the city is at 450 meters. It covers a total area of 603.4 km². Climate is subtropical, with mild winters and warm summers.

The weather is typical coastal sultry and humid. The average rainfall records from 1500 mm to 2000 mm. The place experiences the onset of the monsoon in the month of June and experiences monsoon till the end of September. The average temperature recorded varies from 25 to 37 degrees.

2. 2. Water Sampling and preservation

The sampling was carried out daily in morning session along different locations of the college campus. The grab samples collected in two sessions for a month were mixed separately to give gross sample. Such gross samples were drawn and preserved for analysis to give the pollution data for the period of twelve months from June 2011 to May 2012. The water samples collected from different sampling stations were filtered using Whatman No. 41 (0.45 µm pore size) filter paper to remove suspended particles. Filtrates were preserved in polythene bottles. In order to prevent the precipitation of metals 2 mL nitric acid was added to the filtrate.

The samples were concentrated to tenfold on a water bath and subjected to nitric acid digestion using the microwave-assisted technique, setting pressure at 30 bars and power at 700 Watts [24, 25]. About 400 mL of the sample was transformed into clean glass separating funnel in which 10 mL of 2% ammonium pyrrolidine dithiocarbamate, 4 mL of 0.5 M HCl and 10 mL of methyl isobutyl ketone (MIBK) are added [26]. The solution in separating funnel was shaken vigorously for 2 min and was left undisturbed for the phases to separate.

The MIBK extract containing the desired metals was then diluted to give final volumes depending on the suspected level of the metals [27]. The sample solution was then aspirated into air acetylene flame in an atomic absorption spectrophotometer.

2. 3. Heavy Metal Analysis by AAS Technique

The analysis for the majority of the trace metals like Copper (*Cu*), Lead (*Pb*), Zinc (*Zn*), Calcium (*Ca*), Chromium (*Cr*), Cadmium (*Cd*), Nickel (*Ni*), Iron (*Fe*), Mercury (*Hg*) and Arsenic (*As*) was done by Perkin- Elmer ASS-280 Flame Atomic Absorption Spectrophotometer. Arsenic (*As*) was determined by hydride generation coupled with an atomic fluorescence detector, while mercury (*Hg*) was analyzed with a cold-vapour atomic adsorption spectrophotometer. The calibration curves were prepared separately for all the metals by running different concentrations of standard solutions. A reagent blank sample was analyzed and subtracted from the samples to correct for reagent impurities and other sources of errors from the environment. Average values of three replicates were taken for each determination.

3. RESULTS AND DISCUSSION

The experimental data on toxic heavy metals in water samples collected along the Bhavan's College Lake of Andheri, Mumbai from the month of June 2011 to May 2012 is presented in Table 1.

The problem of environmental pollution due to toxic metals has begun to cause concern now in most major metropolitan cities. The toxic heavy metals entering the ecosystem may lead to geoaccumulation, bioaccumulation and biomagnifications. A number of elements are normally present in relatively low concentrations usually less than a few ppm in conventional irrigation waters and are called trace elements. Heavy metals are a special group of trace elements which have been shown to create definite health hazards when taken up by plants. Under this group are included Arsenic (*As*), Cadmium (*Cd*), Chromium (*Cr*), Nickel (*Ni*), Copper (*Cu*), Lead (*Pb*), Mercury (*Hg*) and Zinc (*Zn*). These are called heavy metals because in their metallic form, their densities are greater than 4 g/cc.

From the results it appears that the *Cu* content in the lake water was minimum of 0.04 ppm and maximum of 0.06 ppm (Table 1). The observed annual average concentration of *Cu* in the water was 0.05 ppm, which was below the permissible limit of 3.0 ppm set for inland surface water [28]. It is important here to note that *Cu* is highly toxic to most fishes, invertebrates and aquatic plants than any other heavy metal except mercury. It reduces growth and rate of reproduction in plants and animals. The chronic level of *Cu* is 0.02–0.2 ppm [29]. Aquatic plants absorb three times more *Cu* than plants on dry lands [30]. Excessive *Cu* content can cause damage to roots, by attacking the cell membrane and destroying the normal membrane structure, inhibited root growth and formation of numerous short, brownish secondary roots [29]. Copper is highly toxic in aquatic environments and has effects in fish, invertebrates, and amphibians, with all three groups equally sensitive to chronic toxicity [31, 32]. Copper will bio concentrate in many different organs in fish and mollusks. Copper also causes reduced sperm and egg production in many species of fish, such as fathead minnows, as well as early hatching of eggs, smaller fry (newly hatched fish) and increased incidence of abnormalities and reduced survival in the fry [33]. Copper is present in normal human serum (the liquid part of blood) at concentrations of 120-140 µg/L. Signs of toxicity will be seen if the copper concentration rises significantly above this range [34, 35]. Copper can affect olfaction (sense of smell) by competing with natural odorants for binding sites, by affecting activation of the olfactory receptor neurons or by affecting intracellular signaling in the neurons [36]. Copper can impact populations and ecosystems as

well as individual aquatic organisms. For example, when sea scallops were exposed to environmentally realistic concentrations of copper, i.e., 10-20 µg/L, sperm and egg production decreased. While mammals are not as sensitive to copper toxicity as aquatic organisms, biomagnifications play critical role in their toxicity. Toxicity in mammals include a wide range of animals and effects such as liver cirrhosis, necrosis in kidneys and the brain, gastrointestinal distress, lesions, low blood pressure and fetal mortality [37-39]. Acute toxicity of copper sulphate to tilapia (*Oreochromis niloticus*) and catfish (*Clarias gariepinus*) species was investigated [40]. They observed behavioral changes, mostly locomotor responses (avoidance) among the test animals on exposure to the different concentrations of copper sulphate.

In the present study, the monthly concentration of *Zn* was in the range of 2.08 ppm to 7.02 ppm (Table.1). The results of the present investigation indicate that the annual average concentration of *Zn* in water samples was 5.56 ppm, which is above the permissible limit of 5.0 ppm set for inland surface water [28]. *Zn* may result in necrosis, chlorosis and inhibited growth of plants [29]. Previous studies have reported toxic effect of *Zn* on some aquatic organisms such as fish [41]. Although there is low toxicity effect of *Zn* in man, however, the prolonged consumption of large doses has been reported to show some health complications such as fatigue, dizziness and neutropenia [42].

The monthly concentration of *Ni* in the lake water samples was found to be in the range of 1.79 ppm - 3.02 ppm (Table 1). The annual average concentration of *Ni* in the water samples was observed to be 2.76 ppm, which is close to the limit of 3.0 ppm set for inland surface water [28]. Short-term exposure to *Ni* on human being is not known to cause any health problems, but long-term exposure can cause decreased body weight, heart, liver damage and skin irritation [29]. Sunderman in 1959 reported the carcinogenic action of nickel carbonyl on rat [43]. *Ni* can accumulate in aquatic life, but its magnification along in food chain is not confirmed. Khunyakari *et al.* [44] investigated toxicity of nickel, copper, and zinc in *Poecilia reticulata*. Heavy metal exposure caused increased mucus like secretion over gills, excessive excretion, anorexia and increased fin movement. Copper was found to be the most toxic followed by zinc and nickel.

Pb is one of the oldest metals known to man and is discharged in the surface water through paints, solders, pipes, building material, gasoline etc. Atmospheric fallout is usually the most important source of lead in the freshwaters. In the present investigation, it was observed that the maximum concentration of *Pb* was 1.09 ppm and the minimum was 0.04 ppm (Table 1). The annual average concentration of toxic *Pb* in water samples was found to be 0.16 ppm, which is above the permissible limit of 0.1 ppm set for inland surface water [28]. Acute toxicity generally appears in aquatic plants at concentration of 0.1–5.0 ppm. In plants, it initially results in enhanced growth, but from a concentration of 5 ppm onwards, this is counteracted by severe growth retardation, discoloration and morphological abnormalities.

There is an adverse influence on photosynthesis, respiration and other metabolic processes. Acute toxicity of *Pb* in invertebrates is reported at concentration of 0.1–10 ppm [29]. Higher levels pose eventual threat to fisheries resources. A number of studies have investigated effects of prolonged *Pb* exposure on freshwater fish. These studies report a wide range of effects induced by chronic exposure to elevated *Pb* concentrations, including effects on pituitary function, gonadosomatic index, oocyte growth [45]. Fewer studies have examined the physiological and biochemical effects of prolonged *Pb* exposure on freshwater invertebrates, but reports of *Pb* toxicity evaluated by mortality are plentiful. Exceptions include reports of behavioral effects in pulmonate snails [46] possibly linked directly to *Pb*-induced modifications of neuron membrane properties [47, 48].

The monthly hexavalent *Cr* content in the lake water samples was found to be minimum of 1.90 ppm and maximum of 2.90 ppm (Table 1), while the annual average concentration was calculated as 2.09 ppm, which was very much above the permissible limit of 0.1 ppm set for inland surface water [28]. The probable source of *Cr* compounds are pigments, mordents and dyes in the textiles and tanning agent in the leather. Acute toxicity of *Cr* to invertebrates is highly variable, depending upon species [29]. For invertebrates and fishes, its toxicity is not much acute. *Cr* is generally more toxic at higher temperatures and its compounds are known to cause cancer in humans [49]. The toxic effect of *Cr* on plants indicate that the roots remain small and the leaves narrow, exhibit reddish brown discoloration with small necrotic blotches [30]. Symptoms of *Cr* phytotoxicity include inhibition of seed germination or of early seedling development, reduction of root growth, leaf chlorosis and depressed biomass [50]. There are many studies on *Cr* toxicity in crop plants. Chromium significantly affects the metabolism of plants such as barley (*Hordeum vulgare*) [51], *citrullus* [52], *cauliflower* [53], and maize (*Zeamays*) [50]. Chromium is highly toxic non-essential element for microorganism and plants [54]. Marked toxicity of chromium was found with respect to photosynthetic pigment, photosynthesis, nitrate reductase activity and protein content of some alga [55]. Chromium toxicity produces chlorosis and necrosis in plants [54].

Cd is contributed to the surface waters through paints, pigments, glass enamel, deterioration of the galvanized pipes etc. The monthly concentration of *Cd* in lake water samples was found to be minimum of 0.02 ppm and maximum of 0.88 ppm (Table 1), with the average annual concentration of 0.20 ppm. The values obtained were found to be below the permissible limit of 2.0 ppm set for inland surface water [28]. There are a few recorded instances *Cd* poisoning in human beings following consumption of contaminated fishes. It is less toxic to plants than *Cu*, similar in toxicity to *Pb* and *Cr*. It is equally toxic to invertebrates and fishes, [15]. In aquatic systems, cadmium is most readily absorbed by organisms directly from the water in its free ionic form *Cd* (II) [56].

The acute toxicity of cadmium to aquatic organisms is variable, even between closely related species, and is related to the free ionic concentration of the metal. Cadmium interacts with the calcium metabolism of animals. In fish it causes lack of calcium (hypocalcaemia), probably by inhibiting calcium uptake from the water. Effects of long-term exposure can include larval mortality and temporary reduction in growth [56]. Mammals can tolerate low levels of cadmium exposure by binding the metal to a special protein that renders it harmless. In this form, the cadmium accumulates in the kidney and liver. Higher levels of exposure, however, lead to kidney damage, disturbed calcium and vitamin D metabolism, and bone loss [57]. Seabirds in general are known to accumulate high levels of cadmium. Kidney damages have been reported in wild colonies of pelagic sea birds having cadmium level of 60-480 µg/g in the kidney [58].

In the present study, the maximum monthly concentrations of *Fe* in the lake water samples was 5.67 ppm and the minimum concentration was 5.01 ppm (Table 1). However, it was observed that, the average annual concentration of toxic *Fe* was 5.19 ppm, which is very much higher than the permissible limit of 3 ppm set for inland surface water [28]. The presence of high concentration of *Fe* may increase the hazard of pathogenic organisms; since most of these organisms need *Fe* for their growth [29]. The *Hg* level in the lake water samples was found to be in the range 0.01 ppm - 0.03 ppm (Table 1). The average yearly concentration of toxic *Hg* was 0.02 ppm, which was very much above the maximum limit of 0.01 ppm set for inland surface water [28]. *Hg* is generated naturally in the environment from the degassing of the earth's crust from volcanic emissions.

Table 1. Heavy metals content in water samples collected from Lake in the Bhavan's College campus of Andheri, Mumbai.

Heavy Metals (ppm)	June 2011	July 2011	Aug. 2011	Sept. 2011	Oct. 2011	Nov. 2011	Dec. 2011	Jan. 2012	Feb. 2012	Mar. 2012	Apr. 2012	May 2012	Avg.
Cu	0.05	0.05	0.06	0.06	0.05	0.06	0.05	0.05	0.06	0.05	0.05	0.04	0.05
Pb	0.08	0.08	0.04	0.04	0.09	0.10	0.09	0.08	1.09	0.08	0.10	0.09	0.16
Zn	6.03	7.02	7.01	2.08	5.01	5.03	5.01	4.02	6.01	6.0	7.0	6.53	5.56
Cr	2.04	2.00	2.04	2.01	2.01	2.01	1.90	2.90	2.06	2.01	2.01	2.04	2.09
Cd	0.11	0.85	0.02	0.02	0.02	0.88	0.33	0.03	0.02	0.04	0.05	0.03	0.20
Ni	3.00	3.01	2.99	2.89	3.02	2.99	1.79	3.01	1.89	2.88	3.00	2.67	2.76
Fe	5.40	5.55	5.67	5.11	5.21	5.01	5.01	5.01	5.01	5.01	5.01	5.30	5.19
Hg	0.01	0.03	0.01	0.02	0.02	0.01	0.02	0.03	0.01	0.03	0.01	0.01	0.02
As	0.05	0.06	0.05	0.06	0.06	0.05	0.05	0.06	0.06	0.06	0.06	0.06	0.06

Atmospheric *Hg* is dispersed across the globe by winds and returns to the earth in rainfall, accumulating in aquatic food chains and fish in lakes. The organic form is readily absorbed in the gastrointestinal tract (90-100 %), lesser but still significant amounts of inorganic mercury are absorbed in the gastrointestinal tract (7-15 %). Previous study have reported that *Hg* in dissolved form enter the fish through the gills [59], further studies have indicated that inorganic *Hg* get adsorbed to the suspended particulate matter and settles down [60], further gets methylated and ultimately enter the food chain, resulting in bioaccumulation [61]. The monthly concentration of *As* in lake water samples was found to be in the range 0.05 ppm to 0.06 ppm (Table 1), while its average concentration was calculated as 0.06, which was below the maximum limit of 0.2 ppm set for inland surface water [28]. *As* is the most common cause of acute heavy metal poisoning in adults. It is released into the environment by the manufacturing of chemicals and glasses. Arsenic gas is a common byproduct produced by the manufacturing of pesticides that contain arsenic. *As* usually accumulates in soil, water and airborne particles from which it is taken up by various organisms.

The concentrations of the dangerous inorganic arsenics that are currently present in surface waters enhance the chances of alteration of genetic materials of fish. This is mainly caused by accumulation of arsenic in the bodies of plant-eating freshwater organisms. Plants absorb arsenic fairly easily, so that high-ranking concentrations may be present in food. Marine algae accumulate arsenate from seawater, reduce it to arsenite, and then oxidize the arsenite to a large number of organoarsenic compounds.

The algae release arsenite, methylarsonic acid, and dimethylarsinic acid to seawater. Dissolved arsenite and arsenate are more toxic to marine phytoplankton than to marine invertebrates and fish [62]. High concentrations of *As* in water can have an adverse effect on health [63-65]. Chronic arsenic poisoning can cause serious health effects including cancers, melanosis (hyperpigmentation or hypopigmentation or white spots), hyperkeratosis (harden skin), restrictive lung disease, peripheral vascular disease (blackfoot disease), gangrene, diabetes mellitus, hypertension and ischaemic heart disease [66].

4. CONCLUSION

Environmental problems concerning land and water bodies cannot be addressed in isolation. They are intricately interwoven with each other and are interdependent, linked by complex atmospheric, geological, physical, chemical and biological interactions. The human activities that effect, and arise from this environment also depend on economic and social factors. The problem is beyond the limits of physical and institutional bodies, and therefore, there is a need to set common objectives and implement compatible policies and programmes.

Today it is realised that solution to any environmental problem can only be achieved through a comprehensive, systematic and sustained approach. During the past few years, attempts were made by various groups to develop strategies directed towards more integrated approach in aquatic environments. Data on pollution in surface waters points out to the need of regular monitoring of water resources and further improvement in the waste water treatment methods.

What is more fundamentally lacking is a consistent internationally recognised and data driven strategy to assess the quality of water bodies like lake and generation of international standards for evaluation of levels of contaminants.

This will be helpful in saving the lake ecosystem from further heavy metal pollution. The pollution problem if neglected will be a cause of great concern can cause irreparable ecological damage in the long-term well masked by short term economic prosperity.

REFERENCES

- [1] Aghor A., Chemicals make Thane creek the worst polluted waterbody, Daily DNA, August 4, 2007. Mumbai, India, 2007, Obtained through the Internet: http://www.dnaindia.com/mumbai/report_chemicals-make-thane-creek-the-worst-pollutedwaterbody_1115439 [Accessed 01/03/2010].
- [2] Singare P. U., Mishra R. M., Trivedi M. P., *Frontiers in Science*. 2(3) (2012) 28-36.
- [3] Singare P. U., Mishra R. M., Trivedi M. P., *Advances in Analytical Chemistry* 2(3) (2012) 14-24.
- [4] Singare P. U., Trivedi M. P., Mishra R. M., *Science and Technology* 2(4) (2012) 87-97.
- [5] Singare P. U., Trivedi M. P., Mishra R. M., *American Journal of Chemistry* 2(3) (2012) 171-180.
- [6] Lokhande R. S., Singare P. U., Pimple D. S., *World Environment* 1(1) (2011) 6-13.
- [7] Lokhande R. S., Singare P. U., Pimple D. S., *Resources and Environment*. 1(1) (2011) 13-19.
- [8] Singare P. U., *Interdisciplinary Environmental Review* 12(4) (2011) 298-312.
- [9] Singare P. U., *Thane lakes high on metal content: Study*, Daily Times of India, August 10, 2011. Mumbai, India.
[Online]. Available: <http://timesofindia.indiatimes.com/city/mumbai/Thane-lakes-high-on-metal-contentStudy/articleshow/9547159.cms>
- [10] Singare P. U., Lokhande R. S., Bhanage S. V., *International Journal of Global Environmental Issues*. 11(1) (2011) 79-90.
- [11] Singare P. U., Lokhande R. S., Naik K. U., *Interdisciplinary Environmental Review* 12(3) (2011) 215-230.
- [12] Singare P. U., Lokhande R. S., Naik K. U., *Interdisciplinary Environmental Review* 11(1) (2010) 90-107.
- [13] Spooner D. R., Maher W., Otway N., *Arch. Environ. Contam. Toxicol.* 45(1) (2013) 92-101.
- [14] Saxena D. K., Srivastava K., Singh S., *Current Science* 94(7) (2008) 901-904.
- [15] Moore J. W., Ramamoorthy S., *Heavy Metals in Natural Waters: Applied Monitoring and Impact Assessment*, Springer-Verlag, New York 1984, pp.28-246.
- [16] Lokhande R. S., Kelkar N., *Indian Journal of Environmental Protection* 19(9) (1999) 664-668 .
- [17] Menounou N., Presley B. J., *Arch. Environ. Contam. Toxicol.* 45(1) (2003) 11-29.

- [18] Swamy Y. V., Roy Chaudhury G., Das S. N., Sengupta S., Muduli R., *Current Science* 91(10) (2006) 1409-1412.
- [19] Jha S. K., Chavan S. B., Pandit G. G., Negi B. S., Sadasivan S., *Environmental Monitoring and Assessment*. 76(2) (2002) 249-262.
- [20] Sharma R. K., Agrawal M., Marshall F. M., *Effects of waste water irrigation on heavy metal accumulation in soil and plants*, Paper presented at the National Seminar, Bangalore University, Bangalore 2004, India.
- [21] Patil D., *A lot's fishy about our creek and lake fish*. Daily Times of India. March 22, Mumbai 2009, India. Obtained through the Internet: <http://timesofindia.indiatimes.com/city/thane/Alots-fishy-about-our-creek-and-lake-fish/articleshow/4298566.cms> [Accessed 01/03/2010].
- [22] Adams W. J., Kimerle R. A., Barnett J. W. Jr., *Environ. Sci. Technol.* 26(10) (1992) 1864-1875.
- [23] Maher W., Batley G. E., Lawrence I., *Freshwater Biol.* 41(2) (1999) 361-372.
- [24] Clesceri L. S., Standard methods for the examination of Water and waste water. In Arnold, E., Greenberg, Eaton, A.D. (Eds.): *Collection and Preservation of Samples And Metals*, pp.1-27, pp.1-35, pp.3-1, pp.3-21, APHA, AWWA, WEF, Washington DC 1998.
- [25] Paar A., *Microwave Sample Preparation System –Instruction Handbook*, Anton Paar GmbH, Austria 1998, pp.128.
- [26] Sachdev S. L., West P. W., *Environmental Science & Technology* 4(9) (1970) 749-751.
- [27] Chen M., Ma L. Q., *Soil Science Society of American Journal* 65(2) (2001) 491-499.
- [28] *The Environment (Protection) Rules*. Available on Internet: cpcb.nic.in/GeneralStandards.pdf. Assessed on: November, 2011, 1986.
- [29] Tiwana N. S., Jerath N., Singh G., Ravleen (Eds.), *Heavy metal pollution in Punjab Rivers, in Newsletter Environmental Information System (ENVIS)*, Punjab State Council for Science and Technology, India, 3(1) (2005) 3.
- [30] Centre for Ecological Sciences., *IISc Environmental Hand Book – Documentation on Monitoring and Evaluating Environmental Impacts*, of Environmental Standards, Vol. 3, Indian Institute of Science, Bangalore 2001, (2001). Available at <http://wgbis.ces.iisc.ernet.in/energy/HC270799/HDL/ENV/START.HTM> (Accessed on 01 March 2010).
- [31] US EPA. 1993. *Wildlife Exposure Factor Handbook*. Vol. 1 EPA/600/R-93/187a
- [32] Horne M. T., Dunson W. A., *Archives of Environmental Contamination and Toxicology* 29(4) (1995) 500-505.
- [33] Taub Frieda B., *Fish 430 lectures (Biological Impacts of Pollutants on Aquatic Organisms)*, University of Washington College of Ocean and Fishery Sciences, Seattle 2004, WA.
- [34] Bradl H., *Heavy Metals in the Environment: Origin, Interaction and Remediation*, Elsevier/Academic Press, London 2005.

- [35] Wright D. A., Welbourn P., *Environmental Toxicology*, Cambridge University Press, Cambridge 2002, U.K.
- [36] Baldwin D. H., Sandahl J. F., Labenia J. S., Scholz N. L. *Environmental Toxicology and Chemistry* 22(10) (2003) 2266-2274.
- [37] ATSDR, *Toxicological Profile for Copper*. U.S Public Health Service. Agency for Toxic Substances and Disease Registry, Atlanta 1990, G.A.
- [38] Kabata-Pendias A., Pendias H., *Trace Elements in Soils and Plants*. 2nd ed. CRC Press, Boca Raton 1992, pp.365.
- [39] Vymazal J., *Algae and Element Cycling in Wetlands*. Lewis Pub., Boca Raton 1995, pp. 689.
- [40] Ezeonyejiaku C. D., Obiakor, M. O., Ezenwelu C. O., *Online Journal of Animal and Feed Research* 1(4) (2011) 130-134.
- [41] Alabaster J. S., Lloyds R., *Water quality criteria for freshwater fish*. Second edition, Butterworths publication, London 1982, pp. 361.
- [42] Hess R., Schmid B., *J. Paediatr. Haematol./Oncol.* 24 (2002) 582-584.
- [43] Sunderman F. W., *Arch Ind.* 20(1) (1959) 36-41.
- [44] Khunyakari R. P., Vrushali T., Sharma R. N., Tare V., *Journal of Environmental Biology.* 22 (2) (2001) 141-144.
- [45] Ruby S. M., Hull R., Anderson P., *Arch Environ Contam Toxicol.* 38(1) (2000) 46-51.
- [46] Pyatt A. J., Pyatt F. B., Pentreath V. W., *Invertebr Neurosci.* 4(3) (2002) 135-140.
- [47] Rozsa K. S., Salanki J., *Cell Mol. Neurobiol.* 14(6) (1994) 735-754.
- [48] Szucs A., Salanki J., Rozsa K. S., *Cell Mol. Neurobiol.* 14(6) (1994) 769-780.
- [49] Ember L., *Environ. Sci. Tech.* 9(13) (1975) 116-121.
- [50] Sharma D. C., Pant R. C., *Journal of Environmental Science and Health. Part A* 29(5) (1994) 941-948.
- [51] Ali N. A., Ater M., Sunahara G. L., Robidoux P. Y., *Ecotoxicology and Environmental Safety* 57(3) (2004) 363-374.
- [52] Dube B. K., Tewari K., Chatterjee J., Chatterjee C., *Chemosphere.* 53(9) (2003) 1147-1153.
- [53] Chatterjee J., Chatterjee C., *Environ. Pollut.* 109(1) (2000) 69-74.
- [54] Cervantes C., Campos-Garcia J., Debars S., Gutierrez-Corona F., Loza-Tavera H., Carlos-Tarres-Guzman M., Moreno-Sanchez R., *FEMS Microbiol. Rev.* 25(3) (2001) 335-347.
- [55] Rai U. N., Tripathi R. D., Kumar N., *Glaucozystis nostochinearum ltzigsohn. Chromosphere.* 25 (1992) 721-732.
- [56] AMAP. *Assessment report: Arctic pollution issues*. Arctic Monitoring and Assessment Programme, Oslo 1998.

- [57] Nordic Council of Ministers Cadmium Review, January 2003. Report no. 1, Issue no. 04.
- [58] WHO. Cadmium - environmental aspects. Environmental Health Criteria 135. World Health Organisation, International Programme on Chemical Safety (IPCS), Geneva 1992, Switzerland.
- [59] Dallinger R., Prosi F., Segner H., Back H., *Oecologia*. 73(1) (1987) 91-98.
- [60] Kehrig H.A., Malm O., Moreira I., *Sci. Tot. Environ.* 213(1-3) (1998) 263-271.
- [61] www.cleanestuary.org/publications/files/CEP_hg_CM.Jan 2006, Conceptual model of mercury for CEP.
- [62] Neff J. M., *Environmental Toxicology and Chemistry* 16(5) (1997) 917-927.
- [63] Cai L., Liu G., Rensing C., Wang G., *BMC Microbiology* 9(4) (2009), doi:10.1186/1471-2180-9-4.
- [64] Kazi T. G., Arain M. B., Baig J. A., *The Science of the Total Environment*. 407(3) (2009) 1019-1026.
- [65] Gbaruko B. C., Ana G. R. E. E., Nwachukwu J. K., *African Journal of Biotechnology* 7(25) (2008) 4737-4742.
- [66] Das H. K., Mitra A. K., Sengupta P. K., Hossain A., Islam F., Rabbani, G. H., *Environment International*. 30(3) (2004) 383-387.