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EDITORIAL

In the series of toxic metals viz arsenic, chromium and mercury covered in earlier issues of ENVIS newsletter, the present newsletter carries information on another toxic metal i.e. "CADMIUM". Cadmium is a naturally occurring minor element, one of the metallic components in the earth's crust ubiquitously found. It is a silvery-white lustrous tarnishable metal, first discovered in Germany in 1817 as a by-product of the zinc refining process. Its name is derived from the Latin word *cadmia* and the Greek *kadmeia*. Cadmium is an extremely toxic metal commonly found in industrial workplaces. Due to its low permissible exposure limit overexposures may occur even in situations where trace quantities of cadmium are found in the parent ore or smelter dust. Cadmium is used extensively in electroplating, although the mode of operation does not generally lead to overexposures. Cadmium is also found in some industrial paints and may represent a hazard when sprayed. Several deaths from acute exposure have occurred among welders who have unsuspectingly welded on cadmium-containing alloys or worked with silver solders. Cadmium emits characteristic brown fumes of cadmium oxide upon heating, which is relatively non-irritating, and thus does not alarm the exposed individual. Under normal conditions, adverse human health effects have not been encountered from general exposure to cadmium. Potential risks have been extensively studied and documented. Cadmium has no known beneficial function in the human body and is a cumulative toxin. It is transported in the blood bound to metallothionein and accumulates in kidneys and the liver. Acute toxicity of this metal was discovered in the early 19th century and its chronic effect on human health was recognized in late 1930s-1940s with the published report of pulmonary, bone and renal lesions cases found in industrial workers. In 1960 an outbreak of Itai-Itai bone disease took place in Japan which drew attention of regulatory & scientific bodies to gather information about this heavy metal.

In the general population, exposure to cadmium occurs primarily by eating certain foods grown in contaminated soil. Cigarette smoke is one of the major sources of cadmium exposure. Cadmium emission in the environment can be controlled by spreading awareness among people about impact of cadmium on the environment and its health implications.

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ODDS AND ENDS

Cadmium-induced neurological disorders: prophylactic role of taurine

Cadmium (Cd) is a cumulative heavy metal poison and does not play any constructive role in the human body. It is a noxious agent and has a very long biological half-life. Along with its compounds, it is extremely toxic even in low concentrations, and will bioaccumulate in organisms and ecosystems. Beside occupational exposure, it enters the body through food and drinking water as well as through inhalation.

The study was conducted to investigate whether the conditionally essential amino acid taurine could play any protective role against the potent neurotoxin cadmium (Cd)-induced oxidative impairment in mice brain. Cd administration in the form of CdCl₂ (at a dose of 4 mg kg⁻¹ body weight for 3 days, orally) increased the intracellular accumulation of metallic Cd, reactive oxygen species and superoxide radicals. The toxin also augmented the extent of lipid peroxidation, protein carbonylation and the levels of glutathione disulfide. Activities of the antioxidant enzymes and the levels of reduced glutathione as well as total thiols have been significantly decreased due to Cd exposure. In addition, the toxin also caused significant DNA degradation (as evidenced from DNA smearing and diphenylamine reaction). Oral administration of taurine (at a dose of 100 mg kg⁻¹ body weight for 5 days) was found to be very effective in the prevention of Cd-induced oxidative impairment in the brain tissue of experimental mice. In addition, taurine treatment could also prevent the reduction in the *in vivo* antioxidant power linearly up to a dose of 100 mg kg⁻¹ body weight. The preventive role of taurine against Cd-induced cerebral oxidative damage was supported by the observation under

scanning electron microscope as well as histological examination of brain segments. To validate the experimental results, a well-known water soluble antioxidant, vitamin C was used as the positive control in the study. In all, the results suggest that taurine plays a beneficial role against Cd-induced cerebral oxidative stress.

Journal of Applied Toxicology 2008, 28/ 8, 974 – 986.

Lead and cadmium in soft plastic toys

It is widely accepted that no level of lead or cadmium in the blood should be considered safe for children and hence every effort should be made to ensure that their environment remains free from any such toxic metals. Toys made of polyvinyl chloride (PVC) are potentially toxic to children as PVC contains both lead and cadmium. Lead or cadmium compounds act as stabilizers but they readily leach out. Moreover, they can also be used in pigments to impart bright colours to toys in order to attract children. Chewing and swallowing behavior of children is a common source of lead and cadmium exposure. The present study was undertaken to ascertain the levels of total lead and cadmium in soft plastic toys. A total of 111 non-branded toy samples, purchased randomly from three metropolitan cities of Delhi, Mumbai and Chennai, were analysed for levels of lead and cadmium. Lead and cadmium were found to be

present in all tested samples in varying concentrations.

Current science 2007, 93/ 6, 818-822.

Sub-lethal effects of heavy metals on biochemical composition and their recovery in Indian major carps

Studies were conducted to assess the effects of sublethal exposure of heavy metals cadmium, arsenic and zinc for 45 days on Indian major carps *Labeo rohita*, *Cirrhinus mrigala* and *Catla catla*. Heavy metal treatments in general showed significant reduction in carbohydrate and lipid content in muscles as well as in gills of all the three fish species. The order of reduction of muscle and gill carbohydrate and lipid content due to different treatments was Cd + As + Zn > Cd + As > As + Zn > Cd + Zn > Cd > As > Zn. When fish were transferred to metal free water for 30 days, the level of carbohydrate and lipid contents improved considerably in all the three fishes.

Journal of Hazardous Materials 2009, 163/2-3, 1369-1384.

Therapeutic efficacy of Picroliv in chronic cadmium toxicity

Cadmium (Cd), an industrial and environmental pollutant, is toxic to several tissues, most notably causing hepatotoxicity on acute administration and nephrotoxicity following chronic exposure. The therapeutic efficacy of Picroliv—a standardized fraction of *Picrorhiza kurroa*, was investigated in male rats treated with Cd as CdCl₂ (0.5 mg/kg, sc) 5 days/week for 24 weeks and Picroliv at two doses (6 and 12 mg/kg, p.o.) was given during the last 4 weeks. The Cd induced levels of malondialdehyde and membrane fluidity and decreased levels of non protein sulphhydryls and Na⁺K⁺ATPase activity of hepatic tissue, along with liver function serum

enzymes were restored to near normalcy on treatment with the higher dose of Picroliv. Enhanced excretion of urinary proteins, Cd, Ca and enzymes (lactate dehydrogenase and N-acetyl-beta-D-glucosaminidase) evident after 24 weeks of Cd exposure, indicated severe renal damage. Picroliv appeared less effective in causing restoration of these urinary parameters as well as oxidative stress indices in the renal tissue. Picroliv not only reduced the accumulated levels of Cd, Zn, Ca and Cd-metallothionein in liver, but also enhanced the bile flow and biliary Cd. The morphological alterations in liver caused by Cd appeared less marked on Picroliv treatment. However, the renal morphology remained uninfluenced. Our earlier data on 18 weeks of Cd and 4 weeks of Picroliv co-treatment showed significant amelioration of both hepatic and renal manifestations of Cd. The hepatic protection by Picrilov is clearly demonstrated in this study, while marginal lowering of urinary proteins and enzymes is a positive signal of renal protective efficacy of Picroliv, which could be augmented by adopting higher doses and extended regimen.

Food and Chemical Toxicology 2009, 47/4, 871-879.

Link between low level cadmium exposure and lung disease

Cadmium is one of the critical ingredients causing emphysema, and even low-level exposure attained through second-hand smoke and other means may also increase the chance of developing lung disease. The University of Michigan School of Public Health study suggests that higher cadmium levels in the body as much as double the risk of developing a pulmonary disease diagnosis such as emphysema or chronic bronchitis. Though some studies have linked high levels of cadmium with decreased lung function in occupationally exposed workers, this

is only the second known study to show that subjects with even slightly increased levels of cadmium had decreased lung function and the first known study to do so using repeated measures of lung function over time. "The study suggests that the critical ingredient in smoking that may be causing emphysema is cadmium, a well-known contaminant of cigarette smoke," said Howard Hu, professor at the U-M School of Public Health and principal investigator in the study. "The worry is if you are exposed to this (cadmium) through other sources you can also be at risk for emphysema." Non-smokers are exposed to cadmium when they eat contaminated food or inhale second-hand smoke, as well as through a host of occupational exposures. Cadmium is a metal that is difficult for the body to dispel, Hu said, because kidneys tend to retain Cadmium and it recycles into the body. Cadmium has received its share of media attention, and some consumer groups are concerned about cadmium in sludge and crop fertilizers. It is also widely used in batteries and pigments. "The big picture is, we keep learning more about the contributions of environmental toxins to the chronic diseases of aging for which we never suspected an environmental cause," said Hu, who is also Chairperson of the School of Public Health Department of Environmental Health Sciences and has an appointment with the Medical School. The study looked at 96 men randomly selected from within the Normative Aging Study, a project that began in 1961 and includes approximately 2,280 healthy, male volunteers from Boston, Mass. Researchers tested lung function using three different measures. Subjects with higher levels of urinary cadmium showed evidence of a reduced ability to exhale, irrespective of whether they smoked but with an effect that was greatest and clearest among current and former smokers. The next step is a

much larger, population-based study with more subjects and multiple measurements of cadmium exposure and lung function over time. Hu said. He also stated that with a larger population we will be able to better disentangle the independent effects of cadmium and smoking, and whether dietary cadmium or other non-cigarette sources may also influence lung function.

<http://www.medicalnewstoday.com/articles/118810.php>.

Association of environmental cadmium exposure with pediatric dental caries

The cross-sectional data, including urine cadmium concentrations and counts of decayed or filled tooth surfaces, from the Third National Health and Nutrition Examination Survey was analyzed. The logistic and zero-inflated negative binomial (ZINB) regression to estimate the association between urine cadmium concentrations and caries experience was used, adjusting these analyses for potential confounders including environmental tobacco smoke (ETS). Urine cadmium concentrations ranged from 0.01 to 3.38 ng/mL. Approximately 56% of children had experienced caries in their deciduous teeth, and almost 30% had been affected by caries in their permanent dentition. An interquartile range (IQR) increase in creatinine-corrected cadmium concentrations (0.21 µg/g creatinine) corresponded to a 16% increase in the odds of having experienced caries in deciduous teeth [prevalence odds ratio (OR) = 1.16 ; 95% confidence interval (CI) , 0.96–1.40]. This association was statistically significant in children with low ETS exposure (prevalence OR = 1.30 ; 95% CI, 1.01–1.67) . The results from the ZINB regression indicated that among children with any caries history in their deciduous teeth, an IQR increase in cadmium was associated with 17% increase in the number of decayed or filled

surfaces. It was observed that there is no association between cadmium and caries experience in permanent teeth. Environmental cadmium exposure may be associated with increased risk of dental caries in deciduous teeth of children.

Environmental Health Perspective 2008, 116/6, 821–825.

Toxicity of cadmium on the growth and yield of *Solanum melongena* L.



Heavy metal cadmium is biomagnified through food chain and causes Itai-Itai disease in human. The present investigation reports the results of the effect of cadmium on seed germination, germination relative index (G.R.I.), seedling growth, chlorophyll stability index (CSI) and yield of *Solanum melongena* L. cv Pusa uttam. Effect of different concentrations of heavy metal cadmium (CdCl_2) in Hoagland's nutrient solution ($10^{(-2)}$ M, $10^{(-4)}$ M, $10^{(-5)}$ M and $10^{(-8)}$ M) were employed for all seedling and physiological parameters of brinjal. Cadmium showed toxic effects at high concentrations $10^{(-2)}$ M but promotory at lower concentration ($10^{(-8)}$ M) with regard to growth and yield.

Journal of Environmental Biology 2008, 29/6, 853-7.

Biomonitoring of metal deposition by using moss transplant method through *Hypnum cupressiforme* (Hedw.) in Mussoorie



Metals Cu, Zn, Cd and Pb were surveyed at 14 sampling sites by using moss *Hypnum cupressiforme* through active monitoring technique. Samples were transplanted in all four directions of Mussoorie city and were harvested after exposure of four months (representing each season) to analyze metal precipitation and its trend at different sites during 2005. Bioaccumulation ability for metals was evaluated seasonally exhibiting maximum in summer followed by winter and minimum in rainy season. However, at some places Cu shows highly significant values in rainy season in comparison to winter. In case of Zn and Pb significantly different ($p < \text{or} = 0.05$) values were observed between summer and rainy season. Baseline concentration of Cu, Pb and Zn was significantly different at 5% in comparison to other transplant sites. Result indicates Dhanaulti as most polluted location might be due to higher tourist activity and vehicular load, whereas, same was found low at Chamba because the place was free from pollution sources or away from in the proximity of roads and have low human interference. The present study allows us to determine the extent of the area affected by metal precipitation load in different rural and urban areas and abundance of metals in order $\text{Zn} > \text{Pb} > \text{Cu} > \text{Cd}$.

Journal of Environmental Biology 2008, 29/5, 683-8.

Nano level detection of Cd(II) using poly(vinyl chloride) based membranes of Schiff bases

The construction and performance characteristics of polymeric membrane electrodes based on two neutral ionophores, 2,2'-(1Z,1'Z)-(1E,1'E)-(1,2-phenylenebis(methan-1-yl-1-ylidene))bis(azaan-1-yl-1-ylidene)bis(methylene)bis(azan-1-yl-1-ylidene)diphenol (L(1)) and 4,4'-(1E,1'E)-(butane-1,4-diybis(azan-1-yl-1-ylidene))bis(methan-1-yl-1-ylidene)dinaphthalen-1-ol (L(2)) for quantification of cadmium ions, are described. The influences of membrane compositions on the potentiometric response of the electrodes have been found to substantially improve the performance characteristics. The best performance was obtained with the electrode having a membrane composition (w/w) of (L(1)) (2.6%):PVC (31.6%):DOP (63.2%):NaTPB (2.6%). The proposed electrode exhibits Nernstian response in the concentration range $5.0 \times 10^{(-9)}$ to $1.0 \times 10^{(-1)}$ M $\text{Cd}^{(2+)}$ with limit of detection $3.1 \times 10^{(-9)}$, performs satisfactorily over wide pH range (2.0 - 8.5) with a fast response time (11s). The electrode has been found to work satisfactorily in partially non-aqueous media up to 40% (v/v) content of methanol, ethanol and acetonitrile and could be used for a period of 2.5 months. The analytical usefulness of the proposed electrode has been evaluated by its application in the determination of cadmium in cigarette samples. The practical utility of the membrane electrode has also been observed in the presence of surfactants.

Analytical Chimica Acta 2009, 634/1, 36-43.

Metallothionein protection of cadmium toxicity

The discovery of the cadmium (Cd)-binding protein from horse kidney in

1957 marked the birth of research on this low-molecular weight, cysteine-rich protein called metallothionein (MT) in Cd toxicology. MT plays minimal roles in the gastrointestinal absorption of Cd, but MT plays important roles in Cd retention in tissues and dramatically decreases biliary excretion of Cd. Cd-bound to MT is responsible for Cd accumulation in tissues and the long biological half-life of Cd in the body. Induction of MT protects against acute Cd-induced lethality, as well as acute toxicity to the liver and lung. Intracellular MT also plays important roles in ameliorating Cd toxicity following prolonged exposures, particularly chronic Cd-induced nephrotoxicity, osteotoxicity, and toxicity to the lung, liver, and immune system. There is an association between human and rodent Cd exposure and prostate cancers, especially in the portions where MT is poorly expressed. MT expression in Cd-induced tumors varies depending on the type and the stage of tumor development. For instance, high levels of MT are detected in Cd-induced sarcomas at the injection site, whereas the sarcoma metastases were devoid of MT. The use of MT-transgenic and MT-null mice has greatly helped define the role of MT in Cd toxicology, with the MT-null mice being hypersensitive and MT-transgenic mice resistant to Cd toxicity. Thus, MT is critical for protecting human health from Cd toxicity. There are large individual variations in MT expression, which might in turn predispose some people to Cd toxicity.

Toxicology Applied Pharmacology 2009, 238/3, 215-220.

Early kidney damage in a population exposed to cadmium and other heavy metals

Exposure to heavy metals may cause kidney damage. The population living near the Avonmouth zinc smelter has been exposed to

cadmium and other heavy metals for many decades. It was aimed to assess Cd body burden and early signs of kidney damage in the Avonmouth population. Dispersion modeling to assess exposure to Cd was used. Urine samples were analyzed from the local population ($n = 180$) for Cd (U-Cd) to assess dose (body burden) and for three biomarkers of early kidney damage [*N*-acetyl- β -*D*-glucosaminidase (U-NAG), retinol-binding protein, and α -1-microglobulin]. Information on occupation, intake of homegrown vegetables, smoking, and medical history by questionnaire was collected. Median U-Cd concentrations were 0.22 nmol/mmol creatinine (nonsmoking 0.18/smoking 0.40) and 0.34 nmol/mmol creatinine (nonsmoking 0.31/smoking 0.46) in non-occupationally exposed men and women, respectively. There was a significant dose-response relationship between U-Cd and the prevalence of early renal damage—defined as U-NAG > 0.22 IU/mmol—with odds ratios of 2.64 [95% confidence interval (95% CI), 0.70–9.97] and 3.64 (95% CI, 0.98–13.5) for U-Cd levels of 0.3 to < 0.5 and levels = 0.5 nmol/mmol creatinine, respectively (p for trend = 0.045). U-Cd concentrations were close to levels where kidney and bone effects have been found in other populations. The dose-response relationship between U-Cd levels and prevalence of U-NAG above the reference value support the need for measures to reduce environmental Cd exposure.

Environmental Health Perspective 2009, 117/2, 181–184.

Selective sorption of lead, cadmium and zinc ions by a polymeric cation exchanger containing nano-Zr(HPO₃S)₂

A novel polymeric hybrid sorbent, namely ZrPS-001, was fabricated for enhanced sorption of heavy metal ions by impregnating Zr(HPO₃S)₂

(i.e., ZrPS) nanoparticles within a porous polymeric cation exchanger D-001. The immobilized negatively charged groups bound to the polymeric matrix D-001 would result in preconcentration and permeation enhancement of target metal ions prior to sequestration, and ZrPS nanoparticles are expected to sequester heavy metals selectively through an ion-exchange process. Highly effective sequestration of lead, cadmium, and zinc ions from aqueous solution can be achieved by ZrPS-001 even in the presence of competing calcium ion at concentration several orders of magnitude greater than the target species. The exhausted ZrPS-001 beads are amenable to regeneration with 6 M HCl solution for repeated use without any significant capacity loss. Fixed-bed column treatment of simulated waters containing heavy metals at high or trace levels was also performed. The content of heavy metals in treated effluent approached or met the WHO drinking water standard.

Environmental Science & Technology 2008, 42/11, 4140-5.

Removal of Cd(II) from aqueous solutions using clarified sludge

Clarified sludge is a major waste generating during steel making process. In India and in most industrial countries, the use of clarified sludge as a road ballast and land filter has had a very long history. In this study, clarified sludge has been characterized and used for the removal of Cd (II) from aqueous solutions. The effect of pH, adsorbent dosage, adsorbate concentration, contact time and temperature on adsorption process was studied in batch experiments. Kinetics data were best described by pseudo-second order model. The effective diffusion co-efficient of Cd (II) is of the order of 10^{-11} m²/s. The maximum uptake was 36.23 mg/g. The adsorption data can be well

described by Langmuir isotherm. The result of the equilibrium studies showed that the solution pH was the governing factor affecting the adsorption. Mass transfer analysis was also carried out for the adsorption process. The thermodynamic studies indicated that the adsorption was spontaneous and exothermic in nature. The sorption energy calculated from Dubinin-Radushkevich isotherm indicated that the adsorption process is chemical in nature. Desorption as well as the application studies were carried out considering the economic viewpoint of wastewater treatment plant operations.

Journal of Colloid Interface Science 2008, 325/1, 48-56.

Kinetic and equilibrium studies on the removal of Cd²⁺ ions from water using polyacrylamide grafted rice (*Oryza sativa*) husk and (*Tectona grandis*) saw dust

The increase in the use of heavy metals has resulted in an increased flux of metallic substances into the aquatic environment which poses a danger to human health. The present work relates to the removal of cadmium ions by treatment with polyacrylamide grafted rice (*Oryza sativa*) husk/saguan (*Tectona grandis*) saw dust. The drinking water guideline value recommended by WHO for cadmium is 0.005 ppm. The adsorbent has been prepared by treatment of rice husk/saw dust with acrylamide. Removal has been studied at various pH values for different times of contact and adsorbate concentrations and is found to be pH-dependent, maximum removal occurs at pH 9 and at a contact time of 180 min for both the adsorbents. The results were found to be consistent with both the Langmuir and Freundlich isotherm models. The value of *n* (rate constant) determined at pH 9 has been found to be 1 (within experimental limits). This is further

substantiated by applying the Lagergren model. The intra-particle diffusion constants were determined by the Morris-Weber model. Continuous flow column studies have also been undertaken and the breakthrough characteristics were determined. Desorption has been affected with 0.5M HCl. The results suggest that both polyacrylamide grafted rice husk/saw dust can be used as efficient and cost effective adsorbents for cadmium ion removal.

Journal of Hazardous Material 2009, 163/2-3, 1338-44.

Cadmium: Toxic effects on the reproductive system and the embryo

The heavy metal cadmium (Cd) is a pollutant associated with several modern industrial processes. Cd is absorbed in significant quantities from cigarette smoke, and is known to have numerous undesirable effects on health in both experimental animals and humans, targeting the kidneys, liver and vascular systems in particular. However, a wide spectrum of deleterious effects on the reproductive tissues and the developing embryo has also been described. In the testis, changes due to disruption of the blood-testis barrier and oxidative stress have been noted, with onset of widespread necrosis at higher dosage exposures. Incorporation of Cd into the chromatin of the developing gamete has also been demonstrated. Ovarian Cd concentration increases with age, and has been associated with failure of progression of oocyte development from primary to secondary stage, and failure to ovulate. A further mechanism by which ovulation could be rendered ineffective is by failure of pick-up of the oocyte by the tubal cilia due to suboptimal expansion of the oocyte-cumulus complex and mis-expression of cell adhesion molecules. Retardation of trophoblastic outgrowth and development, placental necrosis and suppression of steroid

biosynthesis, and altered handling of nutrient metals by the placenta all contribute to implantation delay and possible early pregnancy loss. Cd has been shown to accumulate in embryos from the four-cell stage onwards, and higher dosage exposure inhibits progression to the blastocyst stage, and can cause degeneration and decompaction in blastocysts following formation, with apoptosis and breakdown in cell adhesion. Following implantation, exposure of experimental animals to oral or parenteral Cd causes a wide range of abnormalities in the embryo, depending on the stage of exposure and dose given. Craniofacial, neurological, cardiovascular, gastrointestinal, genitourinary, and limb anomalies have all been described in placentates, with axial abnormalities and defects in somite structure noted in fish and ventral body wall defect and vertebral malformation occurring in the chick. In this study, mechanisms by which Cd can affect reproductive health, and the use of micronutrients in prevention of these problems has been examined.

Reproductive toxicology 2008, 25/3, 304-315.

How plants cope with cadmium: staking all on metabolism and gene expression

Environmental pollution is one of the major problems for human health. Toxic heavy metals are normally present as soil constituents or can also be spread out in the environment by human activity and agricultural techniques. Soil contamination by heavy metals as cadmium, highlights two main aspects: on one side they interfere with the life cycle of plants and therefore reduce crop yields, and on the other hand, once adsorbed and accumulated into the plant tissues, they enter the food chain poisoning animals and humans. Considering this point of view, understanding the mechanism by

which plants handle heavy metal exposure, in particular cadmium stress, is a primary goal of plant-biotechnology research or plant breeders whose aim is to create plants that are able to recover high amounts of heavy metals, which can be used for phytoremediation, or identify crop varieties that do not accumulate toxic metal in grains or fruits. In this review the focus was on the main symptoms of cadmium toxicity both on root apparatus and shoots. The mechanisms that plants activate to prevent absorption or to detoxify toxic metal ions, such as synthesis of phytochelatins, metallothioneins and enzymes involved in stress response were elucidated. Finally new plant-biotechnology applications that can be applied for phytoremediation were considered.

Journal of Integrative Plant Biology 2008, 50/10, 1268 – 1280.

Phytoextraction of cadmium by *Ipomoea aquatica* (water spinach) in hydroponic solution: Effects of cadmium speciation



Phytoextraction is a promising technique to remediate heavy metals from contaminated wastewater. However, the interactions of multi-contaminants are not fully clear. This study employed cadmium, Triton X-100 (TX-100), and EDTA to investigate their interactions on phytotoxicity and Cd phytoextraction of *Ipomoea aquatica* (water spinach) in simulated wastewater. The Cd speciation was estimated by a chemical equilibrium model and MINEQL+. Statistic regression was applied to evaluate

Cd speciation on Cd uptake in shoots and stems of *I. aquatica*. Results indicated that the root length was a more sensitive parameter than root weight and shoot weight. Root elongation was affected by Cd in the Cd-EDTA solution and TX-100 in the Cd-TX-100 solution. Both the root length and the root biomass were negatively correlated with the total soluble Cd ions. In contrast, Cd phytoextraction of *I. aquatica* was correlated with the aqueous Cd ions in the free and complex forms rather than in the chelating form. Additionally, the high Cd bioconcentration factors of *I. aquatica* (375–2227 kg^{-1} for roots, 45–144 kg^{-1} for shoots) imply that *I. aquatica* is a potential aquatic plant to remediate Cd-contaminated wastewater.

Chemosphere 2008, 72/4, 666-672.

Effects of cadmium (Cd) and lead (Pb) on the structure and function of thyroid gland

The study was carried out to investigate the effects of two heavy metals, Cadmium (Cd) and Lead (Pb), on the structure and function of the thyroid gland. Wistar albino rats were chosen for experimental purpose. The animals were fed on diet mixed with given doses of Cd and Pb salts. Blood serum was taken from treated rats recorded decrease in the thyroxine (T4) and the 3, 3, 5-triiodothyronine (T3) levels with a concomitant rise in the Thyroid Stimulator Hormone (TSH) levels, while no change in glucose and cholesterol levels was shown. Microscopic examination of the cellular structure of the thyroid glands of treated rat's recorded changes in the follicular cells of the thyroid tissues in the rats exposed to Cd and Pb in a comparison to that of the control animals. Histological results were confirmed by the findings of the serum analyses that recorded inhibition on the production of the thyroid hormones in the presence of

Cd and Pb. This indicates that animals exposed to Cd and Pb may be at a risk of thyroid damage.

African Journal of Environmental Science and Technology 2009, 3/3, 78-85.

Milk trace elements in lactating cows environmentally exposed to higher level of lead and cadmium around different industrial units.

Investigation was carried out to assess the trace mineral profile of milk from lactating cows reared around different industrial units and to examine the effect of blood and milk concentration of lead and cadmium on copper, cobalt, zinc and iron levels in milk. Respective blood and milk samples were collected from a total of 201 apparently healthy lactating cows above 3 years of age including 52 cows reared in areas supposed to be free from pollution. The highest milk lead ($0.85 \pm 0.11 \mu\text{g/ml}$) and cadmium ($0.23 \pm 0.02 \mu\text{g/ml}$) levels were recorded in lactating cows reared around lead-zinc smelter and steel manufacturing plant, respectively. Significantly ($P < 0.05$) higher concentration of milk copper, cobalt, zinc and iron compared to control animals was recorded in cows around closed lead cum operational zinc smelter. Analysis of correlation between lead and other trace elements in milk from lactating cows with the blood lead level $> 0.20 \mu\text{g/ml}$ ($n=79$) revealed a significant negative correlations between milk iron and milk lead ($r = -0.273$, $P = 0.015$). However, such trend was not recorded with blood lead level $< 0.20 \mu\text{g/ml}$ ($n=122$). The milk cobalt concentration was significantly correlated ($r = 0.365$, $P < 0.001$) with cadmium level in milk and the highest

milk cadmium (>0.10 to $0.39 \mu\text{g/ml}$) group had significantly ($P<0.05$) increased milk cobalt. It is concluded that increased blood and milk lead or cadmium level as a result of natural exposure of lactating cows to these environmental toxicants significantly influences trace minerals composition of milk and such alterations affect the milk quality and nutritional values.

Science of the Total Environment 2008, 404/1, 36-43.

Phytoremediation of Hg and Cd from industrial effluents using an aquatic free floating macrophyte *Azolla pinnata*

The level of heavy metal pollution in Singrauli, an industrial region in India, was assessed and the phytoremediation capacity of a small water fern, *Azolla pinnata* R.BR (Azollaceae), was observed to purify waters polluted by two heavy metals, i.e., mercury (Hg) and cadmium (Cd) under a microcosm condition. *Azolla pinnata* is endemic to India and is an abundant and easy-growing free-floating water fern usually found in the rice fields, polluted ponds, and reservoirs of India. The fern was grown in 24 40-L aquariums containing Hg^{2+} and Cd^{2+} ions each in concentrations of 0.5, 1.0, and 3.0 mgL^{-1} during the course of this study. The study revealed an inhibition of *Azolla pinnata* growth by 27.0-33.9% with the highest in the presence of Hg (II) ions at 0.5 mgL^{-1} in comparison to the control. After 13 days of the experiment, metal contents in the solution were decreased up to 70-94%. In the tissues of *Azolla pinnata*, the concentration of selected heavy metals during investigation was recorded between 310 and 740 mgKg^{-1} dry mass, with the highest level found for Cd (II) treatment at 3.0 mgL^{-1} containing a metal solution.

International Journal of

Phytoremediation. 2008, 10/5, 430-439.

Bone resorption and environmental exposure to cadmium in women: a population study

294 women (mean age, 49.2 years) were randomly recruited from a Flemish population with environmental cadmium exposure. A 24hr urinary cadmium and blood cadmium as indexes of lifetime and recent exposure was measured respectively. The multivariate-adjusted association of exposure with specific markers of bone resorption, urinary hydroxylysylpyridinoline (HP) and lysylpyridinoline (LP) as well as with calcium excretion, various calciotropic hormones, and forearm bone density were assessed. In all women, the effect sizes associated with a doubling of lifetime exposure were 8.4% ($p = 0.009$) for HP, 6.9% ($p = 0.10$) for LP, 0.77 mmol/day ($p = 0.003$) for urinary calcium, -0.009 g/cm^2 ($p = 0.055$) for proximal forearm bone density, and -16.8% ($p = 0.065$) for serum parathyroid hormone. In 144 postmenopausal women, the corresponding effect sizes were -0.01223 g/cm^2 ($p = 0.008$) for distal forearm bone density, 4.7% ($p = 0.064$) for serum calcitonin, and 10.2% for bone-specific alkaline phosphatase. In all women, the effect sizes associated with a doubling of recent exposure were 7.2% ($p = 0.001$) for urinary HP, 7.2% ($p = 0.021$) for urinary LP, -9.0% ($p = 0.097$) for serum parathyroid hormone, and 5.5% ($p = 0.008$) for serum calcitonin. Only one woman had renal tubular dysfunction (urinary retinol-binding protein $> 338 \mu\text{g/day}$). In the absence of renal tubular dysfunction, environmental exposure to cadmium increases bone resorption in women, suggesting a direct osteotoxic effect with increased calciuria and reactive changes in calciotropic hormones.

Environmental Health Perspective

2008, 116, 777-783.

Assessing heavy metal hyper-accumulation and mobility in selected vegetable crops: A case study of organic farm, Gujarat, India

The heavy metals play an important role in the metabolic pathways during the growth and development of plants, when available in required concentration. The heavy metal concentration of cadmium (Cd), cobalt (Co), copper (Cu), iron (Fe), nickel (Ni), lead (Pb) and zinc (Zn) was analysed using Inductive Coupled Plasma Analyser (ICPA) in 20 vegetable crop plants and their parts along with the soil, collected from Shivam organic farm, Valasan, Anand, Gujarat, India. The vegetables selected for the present investigation were *Abelmoschus esculentus*, *Allium sativa*, *A. cepa*, *Anethum graveolens*, *Brassica oleracea*, *B. oleracea*, *Capsicum annum*, *Coccinia indica*, *Coriandrum sativum*, *Cucumis sativus*, *Cyamopsis psoralioides*, *Lagenaria vulgaris*, *Luffa acutangula*, *Lycopersicon esculentum*, *Mentha piperata*, *Momordica charantia*, *Raphanus sativus*, *Solatum melongena*, *Spinacia oleracea* and *Trigonellafoenum-graceum*. The concentration of heavy metals in vegetable crop plants, grown in organic farm, falls within the prescribed limit except Fe. The Accumulation Factor (AF) and Mobility Index (MI) were calculated for the assessment of accumulation and mobility of heavy metals from soil to various plant parts, i.e., roots, stems and leaves through different levels: Level 1 (soil-root), Level 2 (roots-stem) and Level 3 (stem-leaf) in the plants. The accumulation factor (AF) for Cd, Co and Pb metals was found greater in plant parts as compared to available levels in soil. On the other hand, the concentration of Cu, Fe, Ni and Zn was lower in plant parts than the soil. The mobility index (MI) of heavy metal in plant parts

was found to be greater compared to available metal concentration in soil. The results showed concentration dependent variables of heavy metal levels among vegetable crop plants. The lower and higher concentration gradient along with their mobility gradient was also determined. A perusal of data reflects that accumulation gradient of each plant component vary according to nature, properties and podsol climate of a particular plant. The data on accumulation factor and mobility index of the heavy metals from soil to leaves suggest that all the metals are highly mobile in the soil.

Nature, Environment and Pollution Technology 2008, 7/2, 203-210.

Long-term dietary cadmium intake and postmenopausal endometrial cancer incidence: a population-based prospective cohort study

Environmental pollutants mimicking the effects of estrogen are suggested

to contribute to the high incidence of hormone-related cancers, but supporting data are sparse. A potent estrogen-like activity of the pollutant cadmium, mediated via the estrogen receptor- α , has been shown *in vivo*. The association between cadmium exposure and incidence of postmenopausal endometrial cancer was examined. The Swedish Mammography Cohort is a population-based prospective cohort of 30,210 postmenopausal women free of cancer diagnose at baseline (1987) and who completed a food frequency questionnaire at baseline and in 1997. The dietary cadmium intake based on the questionnaire data and the cadmium content in all foods was estimated. During 16.0 years (484,274 person-years) of follow-up between the baseline and mid-2006, 378 incident cases of endometrioid adenocarcinoma were ascertained. The average estimated dietary cadmium intake was 15 $\mu\text{g}/\text{day}$ (80% from cereals and

vegetables). Cadmium intake was statistically significantly associated with increased risk of endometrial cancer in all women; the multivariate relative risk (RR) was 1.39 [95% confidence interval (CI), 1.04–1.86; $P_{\text{trend}} = 0.019$], comparing highest tertile versus lowest. Among never-smoking women with body mass index (BMI) of $<27 \text{ kg}/\text{m}^2$, the RR was 1.86 (95% CI, 1.13–3.08; $P_{\text{trend}} = 0.009$). There was a 2.9-fold increased risk (95% CI, 1.05–7.79) associated with long-term cadmium intake consistently above the median at both baseline 1987 and in 1997 in never-smoking women with low bioavailable estrogen (BMI of $<27 \text{ kg}/\text{m}^2$ and nonusers of postmenopausal hormones). The results support the hypothesis that cadmium may exert estrogenic effects and thereby increase the risk of hormone-related cancers.

Cancer Research 2008, 68/15, 6435–41.

DID YOU KNOW ?

- International Agency for Research on Cancer (IARC) has classified cadmium as a Group I human carcinogen.
- Cadmium accumulates in the kidney, liver, and bone and is excreted very slowly once it enters in the body.
- The largest potential source of cadmium exposure, besides battery and paint factories, are food and cigarette.
- Cadmium can be efficiently removed from source waters by lime softening; coagulation with ferric sulphate, alum; ion exchange & reverse osmosis.

CADMIUM TOXICITY AND ITS MANAGEMENT

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Immunotoxicology

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Introduction

Humans are exposed to cadmium (Cd), a ubiquitous metal with no known biological function, mainly through occupation, contamination and from cigarette smoke. It is considered as one of most toxic

substances in the environment due to its wide range of organ toxicity and long elimination half-life of 10-30 years. It is classified by IARC as Group I carcinogen to humans [1]. While animal studies support a role for cadmium-induced prostate cancer, inconsistent findings exist for its role

in human prostate, breast, testicular, and bladder cancers.

Cadmium exposure

Environmental exposure to Cd generally occurs through the consumption of foods grown in Cd-contaminated areas. Because Cd is

fairly common as an impurity in ores, areas where mining or refining of ores takes place are at highest risk for contamination. The 1950 epidemic of painful osteomalacia is a remarkable historical example of environmental Cd pollution which occurred in Japan. Called as itai-itai disease, it affected hundreds of people. The World Health Organization has shown that dietary Cd exposure has a very wide range: inhabitants of worldwide nonpolluted areas have a daily dietary intake of approximately 40-100 µg, while inhabitants of polluted areas may obtain 200 µg or more as an average daily intake. Environmental exposure also occurs in smokers, who have higher blood Cd than non smokers, probably as a result of Cd contamination of the soil in which tobacco is grown. It is quite likely that Cd and tobacco are synergistic causes of chronic pulmonary disease. Workers exposed to Cadmium oxide fumes and to dust are at risk of developing Cd toxicity.

Cadmium metabolism and mechanisms of toxicity

Cadmium metabolism and its mechanism of toxicity has been well documented in animal models. Cadmium after absorption, is quickly and preferentially taken up by the liver where it induces metallothionein, a cysteine-rich protein that functions as a natural chelating agent with a strong affinity for Cd. Slowly, hepatic stores of metallothionein/cadmium complex are released and circulating Cd-MT is then filtered by glomerulus in the kidneys. A significant amount is reabsorbed and concentrated in proximal tubular cells. Cadmium can also accumulate in bone, pancreas, adrenals, heart, lungs, testis and placenta, but less avidly. Approximately 50 percent of total body stores occur in liver and kidney. Acute Cd exposure causes liver damage[2].

Chronic exposure to Cd leading to renal proximal tubular cell injury and

dysfunction in both humans and animals is known to be reversible during early damage conditions but becomes irreversible at an advanced stage[3]. Glomerular damage and kidney stones have been encountered in those occupationally exposed to Cd. Studies of workers with cadmium-induced renal damage estimate 40-80 percent increased annual mortality risk as a result of Cd exposure and renal damage. Proteinuria is the most common clinical finding and correlates with proximal tubular dysfunction, which manifests as urinary loss of low-molecular weight proteins such as β_2 -microglobulin and retinol-binding protein. The mechanisms of cadmium-induced renal damage results from dissolution of cadmium/metallothionein complex in kidney, exposing the renal tissue to unbound cadmium. Cadmium/cell membrane binding, cellular apoptosis of renal proximal tubular cells, increased calcium loss in urine and increased protein excretion has been observed in animals given long-term doses of Cd or repeated doses of cadmium/metallothionein complexes. Studies have also indicated that when the kidney is able to induce adequate de novo synthesis of metallothionein, no membrane damage occurs. Cadmium-induced osteomalacia caused by abnormalities in calcium and phosphate homeostasis can also result from renal proximal tubular dysfunction. The reproductive toxicity of Cd in rodents is well established. Acute Cd exposure causes vascular destruction in the testis and induced hypoxia/ischemia produces a series of secondary effects, including seminiferous tubule necrosis and leydig cell damage. Apart from hepatic, renal and testicular toxicity, Cd is a potent immunotoxicant as studied in rodents. *In vivo*, Cd causes thymic damage including loss of thymus weight, T-cell depletion and thymic atrophy[4]. Divergent effects of Cd on the immune system have been demonstrated. Susceptibility

of different thymocyte subpopulation to Cd induced apoptosis reveal a marked decrease in CD4+/CD8+ ratio. The mechanisms of Cd toxicity are not completely understood, but some of the cellular effects are known. Fifty to sixty percent of exposed populations have been shown to have chromosomal damage. Cadmium is known to bind to mitochondria and is capable of inhibiting both cellular respiration and oxidative phosphorylation.

Some of the specific changes that lead to tissue damage and death in Cd exposure have been related to oxidative stress (generation of ROS, depletion of GSH, increased lipid peroxidation and altered antioxidant enzymes), modulation of apoptosis, oxidation of nucleic acids and inhibition of DNA repair enzymes [5-7]. Cadmium can substitute for zinc or selenium in metalloenzymes. Lowered levels of selenium as well as decreased activity of glutathione peroxidase (a selenium-dependent enzyme) has been observed in Cd-exposed workers. Cadmium's ability to generate free radicals leading to the release of inflammatory chemokines and cytokines and alteration in expression of several genes, notably regulated through redox sensitive pathways has been reported [8,9].

Management of cadmium toxicity

The management of Cd toxicity is challenging. Cessation of Cd exposure is the first intervention. The fast intracellular binding of Cd to metallothionein leads to more intractable problems as interval between exposure and diagnosis increases. Therefore, there is no clinical antidote available for Cd poisoning and symptomatic treatment is the only choice.

Synthetic antidotes, trace elements and natural plant derivatives have been attempted for the mitigation of Cd toxicity. Chelation in chronic Cd

4. Pathak N, Khandelwal S, 2007. Toxicology Letters, 169, 95-108.
5. Ercal, N, Gurer-Orhan, H Aykin-Burns, N, 2001. Curr. Top. Med. Chem., 1, 529-539.
6. Lee WK, Abouhamed M, Thevenod F, 2006. Am. J. Physiol. Renal Physiol., 291, 823-832.
7. Ye JL, Mao WP, Wu AL, Zhang C et al 2007. Environ. Toxicol. Pharmacol., 24, 45-54.
8. Pathak N, Khandelwal S, 2008. Biometals, 21, 179-187.
9. Beyersmann D, Hechtenberg S. 1997. Toxicol. Appl. Pharmacol., 144, 247-261
10. Khandelwal S, Agnihotri N, Tandon, SK, 1990. Clin. Chem. Enzym. Comms. 3, 55-60.
11. Tandon, SK, Singh S, Prasad, S, 1997. Environ. Toxicol. Pharmacol. 3, 159-165.
12. Liu J, 1995. J. Ethnopharmacol. 49, 57-68.
13. Sumathi R, Baskaran G, Varalakshmi P, 1996. Jpn J Med Sci Biol. 49, 39-48
14. Nomiyama K, Nomiyama H, 1993. J. Trace Elem. Exp. Med. 6, 171-178
15. Khandelwal S, Shukla LJ, Shanker R, 2002. Indian J. Exp. Biol. 40, 564-570.
16. Yadav N, Dogra RKS, Khan MY, Khandelwal S, 2005. Hum. Exp. Toxicol. 24, 529-536.
17. Yadav N, Khandelwal S, 2006. Hum. Exp. Toxicol. 25, 1-11.
18. Yadav N, Khandelwal S, 2009. Food Chem. Toxicol. 47, 871-879.
19. Yadav N, Khandelwal S, 2008. Food Chem. Toxicol. 46, 494-501.
20. Pathak N, Khandelwal S, 2006. Biochem. Pharmacol. 72, 487-497.
21. Pathak N, Khandelwal S, 2007. Eur. J Pharmacol. 576, 160-170.
22. Pathak N, Khandelwal S, 2009. Environ. Toxicol. Pharmacol. 28, 52-60.

CURRENT CONCERNS

Cadmium is used in the manufacturing of variety of consumer products such as batteries, pigments, coatings and platings, stabilizers for plastics, nonferrous alloys, photovoltaic devices etc. It

provides long service lives to the products which makes it irreplaceable so from environmental point of view the recovery of cadmium from cadmium products would ensure that cadmium would be kept out of the

waste stream and out of the environment, but it will also conserve limited resources of cadmium. It is therefore necessary to encourage the industries to collect and recycle cadmium-containing products, which would contribute to the sustainable and safe use of cadmium.

REGULATORY TRENDS

The certain standards sets by Indian agency for cadmium are:

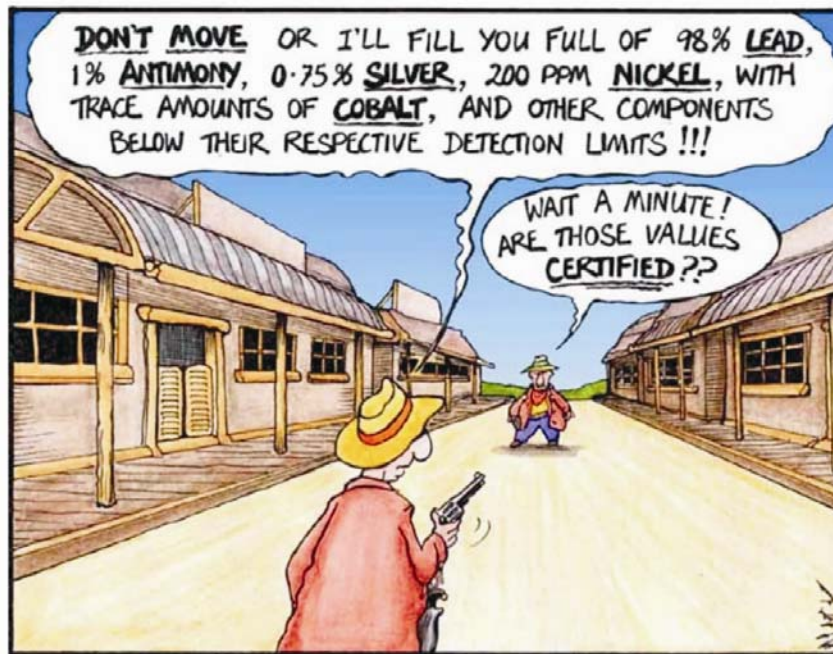
- Bureau of Indian Standards (IS 10500) sets standard for cadmium in drinking water. The desirable limit is 0.01 mg/l.
- Under Schedule II of the Environment (Protection) Act, 1986, standards for discharge of effluent into Inland surface water, Public Sewer & Marine coastal areas are set to 2.00 mg/l.
- Under schedule I of the Environment (Protection) Act,

1986 industry specific standards have been notified for small scale industries, dye and dye intermediate industries electroplating industries, inorganic chemical industry (wastewater discharge) & bullion refining. It's limits is 2.00 mg/l.

- The Prevention of Food Adulteration Act, 1954 limits the cadmium content 1.50 ppm by weight for all food items. The Ministry of Health and Family Welfare Government of India and the State Health Directorate are

responsible for implementing this regulation. Similarly, under Export (Quality Control and Inspection) Act, 1963 the Maximum Residual Limits (MRLs) for heavy metals in fish and fishery products have been promulgated. However, if the MRL fixed by the importing country are more stringent than these prescribed limits, the standard prescribed by import countries need to be complied.

ON THE LIGHTER SIDE



<http://www.mrseiler.org/cartoons.html>

ON THE WEB

<http://www.atsdr.cdr.gov/Toxprofiles/phs5.html>

The Agency for Toxic Substances and Disease Registry was created pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act of

1980 which established a congressional mandate to "remove or clean up abandoned and inactive hazardous waste sites and to provide federal assistance in toxic emergencies.

www.cadmium.org/ - 9k -

Cadmium and the future. Legislation.

www.osha.gov/SLTC/cadmium/recognition.html - 36k -

This links aid in recognizing cadmium and its compounds and the health effects associated with them.

CONFERENCES

3rd International Perspective on Current & Future State of Water Resources & the Environment

5 to 7 January 2010, Chennai, Tamil Nadu, India

Website: <http://content.asce.org/>

conferences/india2010/index.html

Indian Environment Summit 2009

14 to 16 September 2009, New Delhi, India

Website: <http://iesummit.net>

ISA-RC-24- International Conference on Water, Environment, Energy and Society

28 to 30 June 2009, Firozabad, Agra, U.P., India

Website: <http://www.environment-societyisa.org>

BOOK STOP

Cadmium

By Allan Cobb

Publisher: Marshall Cavendish, 2007

ISBN 0761426868, 9780761426868

Introduces the element cadmium, discussing its physical and chemical properties, where it is found, and how it is used.

Environmental Science and Tech-**nology in India**

By Arvind Kumar

Publisher: Daya Books

ISBN 8170355419, 9788170355410

MINI PROFILE OF CADMIUM SULFIDE

SYNONYMS: Cadmium Golden 366; Cadmium Lemon Yellow 527; Aurora Yellow; Cadmium Orange; Capsebon; Greenockite

CAS RN: 1306-23-6**MOLECULAR FORMULA:** Cd-S**MOLECULAR STRUCTURE:****MOLECULAR WEIGHT:** 144.48

PROPERTIES: Light-yellow or orange-colored cubic or hexagonal crystals; melting point: Sublimes at 980 °C, very slightly soluble in ammonium hydroxide; soluble in water (18 °C): 0.13 mg/100 g; oxidized to the sulfate, basic sulfate and eventually to oxide on heating in air to 700 °C, especially in the presence of moisture; decomposes with evolution of H₂S in concentrated or warm dilute mineral acids; readily decom-

poses in moderately dilute HNO₃.

USES: As a pigment; color for soaps; coloring glass yellow; coloring textiles, paper, rubber; in printing inks, ceramic glazes, fireworks; in phosphors and fluorescent screens; in scintillation counters, semiconductors, photoconductors.

TOXICITY DATA:Oral-Rat:LD₅₀:080 mg/KgOral-Mouse:LD₅₀:1166 mg/kg

Route	Symptoms	First Aid	Target Organ
Inhalation/ Ingestion	Irritation, chest pain, cough difficulty in breathing, sore throat, 'metal fume fever' shivering, sweating, body pains, headache dizziness, weakness, nausea, vomiting, diarrhea, abdominal pain, burning sensation, salivation, muscle cramps, vertigo, shock, unconsciousness and convulsions.	Allow the victim to rest in a well ventilated area. Do not induce vomiting. Loosen tight clothing such as a collar, tie, belt or waistband. If the victim is not breathing, perform mouth-to-mouth resuscitation. Seek immediate medical attention.	Respiratory tract and gastrointestinal
Contact	Irritation, skin eruptions and pruritus may occur.	Gently and thoroughly wash the contaminated skin with running water and non-abrasive soap. Be particularly careful to clean folds, crevices, creases and groin. Cover the irritated skin with an emollient. If irritation persists, seek medical attention.	Eye & Skin

PERSONAL PROTECTION: Wear splash goggles, lab coat, gloves & dust respirator.

HANDLING AND STORAGE: Keep container dry. Keep in a cool place. Ground all equipment containing ma-

terial. Carcinogenic, teratogenic or mutagenic materials should be stored in a separate locked safety storage cabinet or room.



MAY WE HELP YOU

To keep abreast with the effects of chemicals on environment and health, the ENVIS Centre of Indian Institute of Toxicology Research, deals with:

Maintenance of toxicology information
database on chemicals

Information collection, collation and dissemination

Toxic chemical related query response service

Preparation of monograph on specified chemicals of current concern

Publishing Abstract of Current Literature in Toxicology

for further details do write to

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