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Environmental Health Effects of Chromium - A Modeling Approach to Find Environmental Impacts

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Abstract: The present study has done to present the complete research investigations pertaining to environmental health effects of chromium and its compounds used in many chromium based industries such as cotton chrome roller ginning industries chromium leather tanneries, textiles, dyeing industries, ferrochrome industries, pigments, metallurgical industries, chemical industries and photography etc. A heath survey was carried out to characterize and assess chromium pollution from these industries. A model has also been presented to assess the potential impacts due to the unsafe chromium in these industries on human. Research investigations are provided pertains to sources of chromium, health effects of chromium and cotton dust, leather processing technology, chrome tanning process, risk assessment, air pollution monitoring and cotton textile processing is presented. A specific case study on cotton roller ginning industries is also presented.

Keywords: Environmental Health Effects, Chromium, Environmental Impacts

1.Introduction

Chromium

Chromium is the 21 st element in earth crust in relative abundance. It belongs to *Group VI B* (Atomic No.24). It is a hard white metallic transition element occurring naturally as chromate. It is a salt in which an ion contains both chromium and oxygen. It is a biologically *inert* metal.

2. Sources

Chromium occurs in the *Valence States of +2, +3 and +6.* The latter two the most common. The +2 and +3 states are basic whereas +6 is acidic, forming are ions of the type chromate and dichromate. Chromium and its compounds are exclusively used in may industries such as leather tanning, textiles, ferrochrome, pigments, and electroplating, photography and CCLC roller ginneries. Chromate is the only economically important salt of chromium and almost all chromium based industries originate from chromate. Chromium based industries generate considerable quantity of pollutants containing *toxic hexavalent* chromium *(Cr^{+6}).*

3. Health effects

Exposure to chromium can occur through the three major routes, via. absorption

through the skin, by direct ingestion and by inhalation of chromium containing particles. The oral LD_{50} of $Na_2Cr_2O_7$ (sodium chromate) in humans has been reported to be 50 ppm (National Institute of Occupational and Safety Hazard, *NIOSH, 1992*).

The absorption of chromium is largely dependent on the oxidation state of the metal and the physical characteristics of the compound itself. Cr^{+6} compounds can penetrate the skin more readily than trivalent forms, and uptake is enhanced with increases in the pH of the chromium containing substances. The absorbed Cr^{+6} is quickly reduced to Cr^{+3}, which binds strongly to the skin and its proteins. The absorption of Cr^{+6} may be increased through broken skin or local actions (dermatitis), as occurs frequently with workers bearing chromium induced dermal ulcerations.

Some specific effects of higher blood levels of chromium include lung cancer, skin ulcers, brain damage, encephalopathy symptoms, necrosis-localized death of a cell or cells in the living body tissues, pneumoconiosis-a lung disease caused by the inhalation of dust particles around the lungs, organoleptic problems, emphysema, bronchitis, careinoma.

Chromium compounds are responsible for a wide range of respiratory effects. Regarding *carcinogenicity* of chromium, several Cr^{+6} compounds like chromate are quite potent carcinogens. The incidence of lung cancer increases due to long term exposure to low to high levels of sodium chromate (Morton Lippman, 1991). Cr^{+6} has been found carcinogenic and is well documented by experiments with animals (Sujana, M.G., et.al.1997). There is some evidence to establish that the insoluble portion of Cr^{+6} is more responsible for cancer incidence (Sujana, M.G. et.al.1997). Cr^{+6} in high dose has been implicated as the cause of *digestive tract cancer*. There is an increase in risk of lung cancer for workers, who are exposed to high levels of chromium (Snell-Etre.1970). Cr^{+6} is highly corrosive and cause chronic ulceration of skin surfaces and perforation of the nasal septum. It has been reported that large doses of Cr^{+3} in impure chromium compounds cause short term *mutagenic damages* (Timmy Katyal, 1979). In addition, the effects of Cr^{+3} are the causative factor through easy reducibility of Cr^{+6} by organic tissues in the body. The size of Cr^{+6} ion radius is in the range as the perforation size in human cell walls. After entering the cells, the ion is reduced to Cr^{+6} . Since, Cr^{+3} being higher radius is retained in cell walls. These degenerative responses occur rapidly and are dependent of the dose and any *hypersensitivity* reactions.

Physiological disorders arising out due to prolonged exposure of Cr^{+3} have been reported (Morton Lippman, 1991). Contact dermatitis occurs as a result of exposure to both Cr^{+3} and Cr^{+6} although *ulceration* is exclusively related to Cr^{+6}. *Systemic toxicity* may occur with both of the oxidation states (mainly from increased absorption of chromium through the broken skin), resulting in renal toxicosis, liver failure, and eventually death (Legard, S., 1992). Cases of *acute systemic poisoning* are rare; however, they may follow deliberate or accidental ingestion. *Chromium poisoning* effects include skin disorders, liver damage, gastric distress, olfactory sense impairment, nosebleeds and yellowing of the tongue and teeth.

The *tetrogenicity* of Cr^{+3} has been demonstrated in animal studies (Morton Lippman, 1991). This includes malformations and open eyelids as well as increased evidence of skeletal defects. *Genotoxicity and mutagenicity* occur due to Cr^{+6} and Cr^{+3}. This is powerful in most prokaryotic and eukaryotic cell systems.

4. Acute toxic effects

The lethal oral dose for soluble chromium compound is considered to be 50-ppm body weight. Short duration exposures to airborne, liquid or solid chromium compounds lead to the clinical symptoms of acute toxicity on human which are vomiting, diarrohea, haemorrhagic diathesis and blood loss into the gastrointestinal tract causing cardiovascular shock. Other diseases are allergic contact dermatitis, skin ulcers, nasal membrane inflammation, nasal ulceration, glycosuria, liver, necrosis, nephritis, aortic plagues,

atherosclerosis, serum albumin, glubulin, necrosis and desquamation of the epetheliums in the kidney, diarrhea, cyanosis, tail necrosis and gastric ulcers (Morton Lippman, 1991).

5. Chronic toxic effects

Prolonged exposure to airborne or solid, liquid chromium compounds lead to chronic toxic effects on humans. The diseases are nasal septum perforations, ulceration's of skin surfaces, rhinitis, liver damage, pulmonary congestion, edema, nephritis, intestinal lung and gastric cancers, irritation of gastro-intestinal mucosa, chronic tortal parental nutrition, (Symptoms like weight loss, hypoglycemia), respiratory effects, congestion and hyperemia, chronic rhinitis, congestion and hyperemia, chronic rhinitis, congestion of larynx polyps of the upper respiratory tract, inflammation of lungs, emphysema, bronchitis, bronchopneumonia, lung tumors, malignant tumour, cutaneous and nasal mucous, membrane ulcers (Morton Lippman,1991). Table-2.1 gives *chromium toxicity effects on human organs.* (See the table at the end of our paper)

Occupational exposures occur during the various stages of production in chromium based industries. Because chromium can be used for many purposes, there is the potential for exposure in a variety of industries. The risk of exposure is through inhalation of chromium bearing aerosols. Additionally, there are wide variations in the possible aerosol characteristics such as the relative proportions of the major oxidation states of the chromium bearing particles as well as varying solubility within these fractions. Cr^{+6} is readily absorbed from the respiratory system, whereas Cr^{+3} is not. Under normal exposure conditions (that are atmospheric chromium), absorption of chromium from the respiratory tract has been estimated to be less than 1 g/day. Absorption from the lung is dependent on the characteristics of the aerosol, including the size, shape, hygroscopicity, and overall electric charge of chromium containing particles. Other factors that may influence the absorption of these particles include ambient temperature, solubility in body fluids and reactions with other air-borne agents. In the *gastrointestinal tract*, only 1% of an ingested dose of Cr^{+3} is absorbed, whereas absorption of Cr^{+6} is 3-6 %. The poor absorption of the Cr^{+6} may be related to its rapid reduction to the trivalent form by components of saliva and gastric juice. Once chromium has entered the bloodstream, it is reduced to Cr^{+6} by components normally present in the blood such as *ascorbic acid* and *glutathione*. The oxidation state of chromium is the determining factor for its transportation *via.* the blood stream. Cr^{+3} is mainly transported *via.* the serum, bound to the iron-binding *transferrin* and *β-globulin* fraction of serum proteins; however at high concentrations Cr^{+3} binds to *serum albumin* or *α1- or α2-globulin*. In contrast to $Cr^{+3,}$ Cr^{+6} can readily cross the erythrocyte membrane and bind to the glubulin portion of *hemoglobin* following oxidation of the *heme* group (Morton Lippman, 1991). Inside these cells, Cr^{+6} is reduced to Cr^{+3} by *glutathione* and then becomes trapped intracellularly. Consequently, the degradation products of *erythrocytes* may explain, in part, the high concentration of chromium found in the spleen and the slow excretion of chromium from the body. Urinary excretion is generally less than 10 g chromium/day in the absence of excess exposure.

The *genotoxicity* of chromium compounds has been documented (Kirth othmer, 1960). The Cr^{+6} ion is readily taken up into *eukaryotic* cells by *anion*-carrying proteins, after which it is reduced to Cr^{+3} by a number of cytoplasmic reducing agents. The final cellular form of Cr^{+3} becomes trapped *intracellularly* because it has low cell membrane permeability. This shift from Cr^{+6} to Cr^{+3} allows a concentration gradient to be established such that a continual influx of Cr^{+3} ions raises intracellular chromium levels until the metal burdens become lethal.

Most Cr^{+6} salts were *mutagenic* and Cr^{+3} salts are relatively *non-mutagenic*. chromium to be capable of inducing *chromosomal aberrations* and *enhancing cells transformation*. Although both the valence states of chromium are able to interact with

DNA, Cr^{+3} ions are responsible for decreasing the fidelity of DNA replication. In addition, both Cr^{+3} and Cr^{+6} exhibit a *clastogenic potency*; however, Cr^{+6} possesses the greater activity and is powerful mutagen in many prokaryotic and eukaryotic cell systems. These properties of Cr^{+6} support the claim that Cr^{+6} compounds are likely to be active carcinogens, although it is more likely that the ultimate species responsible for the carcinogenic/mutagenic effects observed in *vivo* is the intracellularly derived Cr^{+3} form (Winslow, H. 1972).

Particulate from *coal-fired power plants* has been shown to contain chromium in the range of 2.3-31 ppm; however, these levels are reduced to 0.19-6.6 ppm by fly ash collection processes. Chromium in ambient air originates from industrial source (that is steel manufacturing and cement production) and the combustion of fossil fuels; the content in coal and crude oil varies from 1- 100 g/l and 0.005 to 0.7 g/l respectively. In rural areas, chromium levels in the air are usually less than 10-ng/ m^3, whereas the concentration varies from 10-50 ng/ m^3 in industrial cities (James, M.Sontag, 1986).

Epidemiological studies of the *incidence of cancer* in occupationally exposed individuals have indicated that cancer mortality rates in the workers were 5-40 times higher than expected. An excess incidence of lung cancers had been reported in workers in the chromate plants. Cancer of the nasal cavities as well as of the larynx is reported in a small exposure population. A survey of chromate plants showed an increased risk of cancer in two of the plants (Probability of 0.66). An increased gastric cancer has been observed among metal finishers. There were five incidences instead of three cases expected (Morton Lippman, 1991).

Overwhelming epidemiological evidence shows a considerable excess of chromium-compounds-induced occupational cancers, with a pre-dislocation to the lung, nasal cavity, sinuses, and larynx. It is found that 70% of the cases had lent periods between 11 and 30 years. The table 2.2 illustrating the degree of cancer hazard of chromium exposure to humans. (See the table at the end of our paper)

6. Sources and Health Effects of Particulate Matter

Sources

Particulate matter in the air usually refers to small solid particles of material found in the atmosphere in addition to gases. The particles of organic or inorganic composition that is suspended may be individual elements and / or compounds. *Particle size diameter* (PSD) may be used to classify the types of sources. For example, particles having PSD less than 1 μm are mostly products of condensation and combustion. Large particles having PSD above 10 μm result from physical actions, such as wind erosion and grinding or spraying operations. Those particles between 1 and 10 μm of PSD tend to be fugitive dusts, process dusts and combustion products. Suspended particulate matter (SPM) in the air refers to particles, which are too fine to have an appreciable falling velocity and therefore tend to stay suspended in atmosphere for a considerable time. Metallic fumes, fibrous materials, heavy metals, all micro-organisms, various allergens and many organic carcinogens are present in air in the form of SPM having the PSD up to 100 μm. Air contaminant dust in the size-range of 0.25-10.0 μm is called respirable suspended particulate matter (RSPM). RSPM of different range series are absorbed forcibly against any surface through the principle of impaction.

7. Health effects

The SPM is the dust entirely consisting of coarse particulate, which are quickly eliminated in the respiratory system and hence are not very harmful. The coarser dust collected as part of the SPM creates nuisance, soiling of surfaces and some visibility problems but is unlikely to contribute significantly to respiratory and other health effects associated with air pollution.

Generally, health effects are caused primarily by *RSPM* in the PSD range of 0.25-10 microns emissions from industries. Particles having PSD less than 0.25 microns are retained in lung alveolar regions. Particles greater than 10 microns are lodged in the upper tract and do not reach the bronchi. Various air pollution sources produce *lung damaging size range particles*.

8. Heath effects of RSPM cotton dust

The dangerous dust from the cotton worker's point of view falls under the category of RSPM. This invisible fine dust enters the air passages and produce physiological changes leading to the *byssinosis* (Shirley-IV, 1982).

Particles size analysis carried out show that the PSD of the *cotton dust* particles varies between 1 to 40 microns and higher. Particles with PSD varying from 5 to 50 microns result in respiratory troubles, as human nose cannot effectively filter the particles and lead to allergic symptoms like asthma. *Byssinosis* is a disabling lung disease caused due to inhalation of cotton *fibre dust* over long periods of time (Shirley-IV, 1982). It is accompanied by chest tightness with respiratory troubles.

9. Health effects of leather dust

The *leather dust* is the carrier of chromium in all its forms and makes it mobile and dangerous. The fine leather dust whose size varies from less than one micron to higher values is easily absorbed through skin and is inhaled. The fibres are usually from 10 to 120 microns in length and 0.5 to 50 microns in diameter. It is of insidious nature and fibrous minute particles being powdered during the ginning process report to cause lung cancer, mesothelioma, chest pain, cough and asthma (Rao, M.N., 1995).

10. An Environmental System Modeling Approach for Finding Health Impacts

Principle of System Diagram Modelling

An approach for explaining the combined *synergistic (augmentative)* health effect is given in this formal model.

The model is a simplified formulation that mimics real-world phenomena so that complex situations can be comprehended and prediction made (Odum, E.P., 1976). It is designed to simulate some real situation for decision making regarding impact of the natural environment on man. In simplest form, models may be verbal or graphic, that is, consisting of concise statements or picture graphs. There are informal and formal models. Formal models are such formulations going to play an increasing role in decision-making problems.

11. Procedure

In its formal version a working model of a *synergistic* and health effects of dust-producing, grinding CCLC roller ginnery situation would, in most cases, have four components, as listed below (with certain technical terms listed in parentheses).

1.Properties (*state variables)*

2.Forces (*forcing functions*), which was outside energy sources or casual forces that drive the system

3.Flow pathways, showing where energy flows or materials *transfers* connect properties with each other and with forces

4.Interactions (*interacting functions*) where forces and properties interact to modify, or amplify or control flows.

On this model were shown six properties P_1, P_2, P_3, P_4, P_5 and P_6 which *interacted* at 'I' to produce or affect the fourth property P_7, when the system was driven by forcing function, 'E'. Flow pathways were shown with F_1, F_2…. and so representing input and output flow-paths receptively for the system overall.

P_1 was chromium , P_{11} as Cr^{+6} and P_{22} as Cr^{+3}

P_2 was cotton dust

P_3 was CCLC powder

P_4 was micro dust including light trash

P_5 was pesticides and insecticides applied to seed-cotton in the field

P_6 was other foreign matter

Where, P_1, P_2, P_3, P_4, P_5 and P_6 were the six pollutants emitted from the dust-producing, grinding CCLC rollers during the ginning process. Under the driving force of four factors namely sunlight (photo energy), temperature, humidity and air movement, these interacted to *produce 'chrome specific dust'* 'P_7'. In this, the interacting function 'I' was a synergistic or augmentative one, which was more serious as a pollutant than P_1, P_2, P_3, P_4, P_5 and P_6 acting independent of each other. The figure-2.7 represents systems diagram modelling showing four components of synergistic (augmentative) health effects in cotton ginning and textile mill plant.

12. Health Study

1. To study the health effects and socio-economic impact of environmental pollution in ginneries and textile mills, an endeavor was made. Local ESI centers, State Govt. hospitals, private clinics and nursing homes were contacted to assess physiological disorders and chromium based diseases.

2 Field studies and rough estimates of the morbidity rates, hospital admissions and absenteeism with normal values, ESI beneficiary records and workers health record have shown that there was high risk of health damaging potential in and around the ginneries. This was readily gauged from the fact that there were large number of private clinics and nursing homes in and around the above towns. The incidence of chrome-based diseases and related health effects on the people around this factory was noticeable. Field studies shown that the ginneries have employed women in the age group 21 to 40 years and workers in the age group of 18 to 50 years.

3. Ginneries were located in and around seed-cotton growing areas, and employed women for menial jobs. The women often came along with their children for performing their jobs, like (i) feeding kapas into the gins, (ii) collecting the lint, seed and floor sweeping, (iii) cleaning and grading the kapas and (iv) light activities. The children were exposed directly with CSD. Plate-2.14 shows (i) working mothers exposed to chrome specific dust and cotton dust and (ii) unsafe chromium contamination and pollution from cotton ginning industries.

The percentage incidence of chromium based diseases and related health effects on the people around this factories was noticeable. They were noted based on the field study made on chromium based diseases as per epidemiological evidences as given in Table 2.3. Plate- 2.15 shows pala house beating of lint cotton. (See the table at the end of our paper)

4. Identified the workers engaged in cotton ginneries and textile mills. Prepared case history and monitored chromium in work environment.

5. Field sampling and estimation of chromium in blood/urine samples in chromium affected workers were attempted.

13. Conclusion

The present study has done to present the complete research investigations pertaining to environmental health effects of chromium and its compounds used in many chromium based industries such as cotton chrome roller ginning industries chromium leather tanneries, textiles, dyeing industries, ferrochrome industries, pigments, metallurgical industries, chemical industries and photography etc. A heath survey carried out on characterization and assessment on chromium pollution from these industries shows that there is adverse health effects on occupational and non-occupational exposures .

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Table- 1
Chromium Toxicity Effects on Human Organs

Symptoms	Affected organs
Prolonged exposure to Cr^{+6} causes ulcers. Skin irritation is reported with prolonged exposure to Cr^{+3}. Disease namely allergic dermatitis, perforation of nasal septum occurs after exposure to Cr^{+6} or chromite dust.	• Skin
Air borne Cr^{+6} causes corrosion of bronco-pulmonary tract, irritation of mucous membrane, lung cancer.	• Nose • Lung • Lung
Tubular necrosis, renal insufficiency Necrosis Damage	• Kidney • Liver • Brain
Hyperchlorhydria	• Gastrointestinal Tract
Chromosomal aberrahos	• Mutagenecity

Source: (Sujana, M.G., et.al.1997)

Table-2
Lung Cancer Among Chromium Exposed Workers

Country	Morbidity ratio		Lung cancers/100000	
	Expected	Observed	General population	Chromium exposed workers
United States	1.9	31.9	16.7	470.8
England	1.3	7	--	--
Germany	1	43	--	--
India	5.0	150	57	3500

Table-3
Study of Chromium Based Diseases as per Epidemiological Evidences

ACUTE EFFECTS	CHRONIC EFFECTS
Allergic contact dermatitis, skin ulcers, nasal membrane inflammation, nasal ulceration, glycosuria, liver necrosis, nephritis, aortic plagues, death in man, athrosclerosis, serum albumin, α_1^- or α_2^- globulin, necrosis and desquamation of the epithelium in the kidney, diarrhea, cyanosis, tail nacrosis, gastric ulcers	Nasal septum perforations, Ulcerations of skin surfaces rhinitis, liver damage, pulmonary congestion, edema, nephritis, intestinal lung & gastric cancers, irritation of gastro-intestinal mucosa, chronic total parenteral nutrition (symptoms : weight loss, hypoglycamea), respiratory effects, congestion & hyperemia, chronic rhinitis, congestion of larynx polyps of the upper respiratory tract, inflammation of lung, emphysema, bronchitis, bronchopeumonia, lung tumors, malignant tumour, cutaneous & nasal mucous membrane ulcers