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HEAVY METALS IN THE INK INDUSTRY

By

Carla Maria Vidal

A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

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ABSTRACT

HEAVY METALS IN THE INK INDUSTRY

By

Carla Maria Vidal

The objective of the present study was to determine how ink manufacturers for the packaging industry were affected by the Model Toxics in Packaging Legislation developed by the Coalition of Northeastern Governors (CONEG).

The study was based upon six heavy metals: copper, zinc, cadmium, chromium (VI), and mercury. Health and environmental effects of the six metals were also summarized.

A survey of ink manufacturers was conducted. Ninety percent of the ink industries responding limit total heavy metal content to less than 100 ppm to comply with CONEG types of regulation. Sixty two and forty one percent of the respondents said cost and price, respectively, were increased by the reduction of heavy metal content in their inks by an average of 9% and 8.8% respectively. Heavy metals are expected to be replaced totally in 5 to 20 years. Organic inks are the usual substitutes for heavy metal based pigments.

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I am indebted to my parents for their continued support throughout my education. Without them, I could never have accomplished all that I have.

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CHAPTER 1

INTRODUCTION

Clogged landfills are a highly visible symptom of our nation's solid waste problem, and, as such, governmental and consumer pressure to reduce the packaging portion are intensifying. As a result, an industry-wide effort to minimize the environmental and health impact of packaging is in full force (Fearncombe, 1995).

One way to reduce the solid waste problem is source reduction applied to objectionable materials, such as heavy metals. The model toxics in packaging legislation as developed by the Coalition of Northeastern Governors (CONEG) focuses on environmental and health issues regarding heavy metal content in packages and packaging components. The CONEG types of regulation definitely ban the intentional introduction of cadmium, hexavalent chromium, lead and mercury in packaging.

The following study will be based upon health and environmental effects of the four heavy metals addressed by the CONEG types of regulation, and additionally copper and zinc. The main purposes of including copper and zinc in this investigation are:

1. Increasing concern about zinc chromate as a lung carcinogen, as evidenced by cases of concern about zinc chromate as a cause of dermatitis through skin contact and lung cancer among ink manufacturing workers.
2. Increasing health and environmental concern about copper and zinc in gold and metallic inks. Exclusion of copper and zinc would eliminate all of the metallic printing presently used in package design, severely reducing the palette of colors available to package designers (Rusterholtz, 1992).

A survey of ink manufacturers was conducted. The main purpose of the questionnaire was to record the effects of the CONEG types of legislation on ink manufacturers in terms of heavy metal content limitation, certification of compliance, price and cost increases, and pigment reformulation.

History

Rusterholtz (1992) states that the concern about heavy metals in flexo ink started in 1989 due to the increased awareness of post-consumer waste, recycling, and resource conservation. According to Czarnecki (1992), the first heavy metal that was considered for elimination was lead. Lead chromates and lead molybdates were removed at once. Czarnecki (1992) and Lusting (1990) listed six heavy metals whose contents in ink were limited by the industry in 1989. This list includes cadmium, arsenic, mercury, antimony, lead and selenium (CAMALS). This limitation caused the elimination of certain chrome yellow, molybdate orange and cadmium red pigments.

According to Rusterholtz (1992), due to the current concern over waste generation and disposal, nine additional metals (aluminum, barium, cobalt, copper, lithium, manganese, molybdenum, silver, and zinc) will be added to the CAMALS list. Package users are taking extreme actions about heavy metals, because such metals are not easily disposable due to their environmental hazards. The inclusion of additional metals will eliminate some blue, green, and white colorations, and would decidedly constrict the list of available red pigments. Many ink companies have entirely eliminated the use of lead pigments in their inks. It seems that companies have added metals to their list at random, without regard to the possible technical consequences to printing.

Sources of Heavy Metals in Inks

Rusterholtz (1989, 1992) referred to metal-containing pigments (colorants) as the main source of metals in printing ink. Heavy metals are included in inks as:

1.- Molecular constituents of some organic pigments such as copper phthalocyanine blue and green, barium red 2B, and calcium lithol rubine, and inorganic pigments such as lead chromate, iron oxide, chrome green and cadmium sulfide.

2.- Insolubilizers of rosin on the pigment surface. Rosin is used to treat the surface of the pigment during manufacturing to improve dispersability.

3.- Inorganic extenders to promote standard color strength in the manufacture of organic, dry color pigments.

Regulations

Fishman and Adelsky (1992) discussed governmental and ink consumers' requirements about heavy metals in printed packaging. The limitations applied to manufacturing, use, and disposal of printed packaging. Ink manufacturers have been pressured by the packaging industry to control metal content in their inks. This action is in response to the proliferation of government regulations. There are a variety of regulations and policies that have impact, including: Resource Conservation and Recovery Act (RCRA), Clean Water Act, federal regulations originated by the Consumer Product Safety Commission (CPSC), Resource Conservation and Recovery Act (RCRA), Occupational Safety and Health Administration (OSHA), American Standards Association, Toy Manufacturers Specifications and Fast Food Industry Guidelines, and different state regulations originating in State Sewerage Guidelines, Proposition 65 and the Coalition of Northeastern Governors (CONEG).

According to Abel (1992), in Europe, limitations on the amount of heavy metals in cosmetics, toys and graphic instruments, and proposed limitations on heavy metals in coatings which are used for food packaging, even where there is no direct contact due to dust, are a substantial problem in many industries. Hickman (1987) stated that the highly colored dusts associated with pigments are pelletised and low-dust forms of pigment are now widely used to improve the working environment. The United Kingdom has banned the inclusion of lead chromate and cadmium in pigments since 1972.

The Coalition of Northeastern Governors (CONEG)

In 1990, nine state governors came together to draft an act to reduce heavy metals in the packaging industry .As a result, It was written the Model Toxics in Packaging Legislation was written as a model for packaging legislation that can be adopted by any state (Johnston, 1992).

The proposed legislation applies to packages, and to package components such as inks, ointment, dyes, etc., and bans the **intentional** presence of four heavy metals: lead, mercury, cadmium and hexavalent chromium. In addition, this legislation limits the **non-intentional** (incidental) presence of the four metals as trace contaminants (Fishman and Adelsky , 1992; and Renson, 1992). The Model Toxics in Packaging Regulation is a result of the increasing awareness of the overall solid waste stream. Heavy metals included in packaging are likely to be present in emissions or ash when packaging is incinerated and in leachate when packaging is landfilled (Fishman and Adelsky, 1992) .

The incidental presence of these metals was limited to 600 ppm one year after enactment of the legislation, 250 ppm two years after, and 100 ppm four years after enactment of the state legislation. The proposed legislation has become effective in eleven states: Connecticut, Iowa, Massachusetts (1992), Maine (April 1, 1992), Minnesota (August 1, 1993), New Hampshire, New York (January 1992), Rhode Island, Vermont, Washington (July 1992), and Wisconsin, and has been proposed in eight additional states : California, Delaware, Georgia, Illinois, Maryland, New Jersey, Pennsylvania, and Oregon. It makes little difference which new states follow the lead of states that have already passed the proposed legislation. Since most goods are sold in many different states, the effective date in any one state effectively imposes limits on heavy metal content in packaging of those goods for all states. Therefore, today, many packagers and their

converters are expecting their ink suppliers to certify to maximum heavy metal content of 100 ppm (Johnston, 1992 and Renson, 1992) .

Certificates of Compliance

According to Fishman and Adelsky (1992), the proposed regulation stipulates that "not later than two years from the adoption of this statute, a Certificate of Compliance of packaging components should be issued by its manufacturer." Johnston (1992) stated that such certificate of compliance needs to be supplied to purchasers of packaging or packaging components, such as ink manufacturer customers, and the converters need to provide this certificate to contractors that package products to be sent to retailers.

A specific analytical method for testing was not specified by the proposed regulation. The suppliers, therefore, are allowed to used different methods. Two types of analytical methods are used for measurement of heavy metal content.

1. Measurement of soluble or leachable metals. Soluble heavy metals refer to the portion of a heavy metal that may leach into water and enter into a large water aquifer. Because landfill disposal is a issue, this test is commonly requested. Weak acid-extraction data is specified under EPA RCRA guidelines, and the strong acid-soluble data is specified in the voluntary toy standards, most fast food standards, the ASTM guidelines, and the European EN71 standard.
2. Measurement of the total metals present. This category of analysis is the one most often used to satisfy the CONEG proposed guidelines.

Within these two types of tests, there are different kinds of sampling or sample preparation (Rusterholtz, 1992). Renson (1992) evaluated batch-to-batch testing as uneconomic and time consuming testing. Random sampling is the most realistic way of sampling. The proposed regulation does not require analysis of every batch, but asks for

random sampling on a reasonable statistical basis. According to Czarnecki (1992), an ASTM standard should be followed to produce reproducible and accurate results.

Substitutes

Reduction of heavy metal content in pigments promoted the development of new pigments that may, to a certain degree, substitute for the present heavy metal based inks. In spite of their low degree of brilliance, relatively poor flow properties and high price, organic substitutes are most commonly used in the ink industries, because they are the closest substitutes existing in the market (Larson, 1992). According to Abel (1992), heavy metal pigments are still a strong competitor against organic pigments for high-performance engineering thermoplastic components used in durable goods.

Johnston (1992) states that substitutes such as hansa yellows, DNA orange, and AZO reds are used widely because they comply with the current legislation.

Hill (1979) mentioned co-precipate pigments as a good substitute for chrome yellow. Co-precipate pigments have almost the same physical properties as chrome yellow. Other substitutes for this pigment are azo couplings, arylide and diarylide. Two disadvantages of these replacements are opacity and high cost.

Disposal

According to Flower et al. (1978), sanitary landfills have been demonstrated to be the least expensive environmentally acceptable means of waste disposal, purportedly possessing attributes of neatness and safety in addition to relatively low cost. However, many landfills were (are) used improperly; illegal dumps of hazardous waste or insufficient disposal facilities are not unusual (Donker, 1994).

One of the potentially dangerous sources of chemical release at waste sites is leachate. Landfill leachate carries a wide range of heavy metals and organic micropollutants. In general, landfills are originated by dumping domestic waste and clean industrial wastes into excavations. Because of their operation, magnitude, and large number, landfills represent an important threat to groundwater resources. Only starting during the 1980's in the U.S. and in a few European countries have new landfills been equipped with liners and other facilities to prevent soil and groundwater contamination (Donker, 1994).

Flower et al. (1978) stated that the rapid urban and suburban development in the United States has caused many once remote dumping grounds to now be close to developed areas. As such, they provide an attractive source of land for many reasons. Conversion to recreational areas or other nonstructural applications has long been considered an acceptable end for completed landfill sites.

Increasingly stringent federal and state laws have made dumping harder for the printing industry, and landfills are closing at a rapid clip because older dumps cannot meet stringent EPA regulations. In the past, the printing industry bought a barrel of oil for $20 and then needed to pay $300 a barrel to get rid of oil mixed with ink in landfills. Landfills are no longer accepting oily waste at all, so those who buy ink need to figure out how to dispose of it (Fitzgerald, 1987).

According to Rusterholtz (1989), heavy metal content in inks is a concern since two-thirds of the total municipal waste in printing substrates. Municipal waste is composed of paper and paperboard (41%), metals (8.7%), glass (8.2%), plastic (6.6%), rubber and leather (8.1%), food waste (7.9%), grass, leaves and plants (18%) and miscellaneous (inorganic waste) (1.5%). Note that the first four categories are all packaging material and also are all printing substrates. Because packaging is disposable, one-third of landfilled waste originates from packaging applications. The number of landfills accepting solid waste has shrunk from 30,000 in 1968 to 6000 in 1989. Once the

packaging is incinerated, the resulting inorganic ash contains heavy-metal oxides or sulfates. Such incinerator ash requires disposal.

We can reduce the amount of heavy metals in the substrates to reduce pollution. Carcinogenic heavy metals such as lead, cadmium, chromium, arsenic, mercury, antimony, selenium and silver are worrisome when they appear in drinking water and consumer products that are, or might be, ingested or absorbed. In the future, legislation is going to continue to grow on local, state, and federal levels. The industry as a whole has to become more proactive in legislation or they will drown in the aftermath of legislation (Johnston , 1992).

CHAPTER 2

ENVIRONMENTAL EFFECTS ASSOCIATED WITH COPPER

Introduction

Copper has been recognized at 210 out of 1177 National Priorities List (NPL) hazardous waste sites in the United States (U.S. Department of Health and Human Services, 1990 a).

According to Nriagu (1979 b), there are three main sources of pollutant copper in the atmosphere :

1. Copper production and handling (46% of the total anthropogenic emissions)
2. Iron and steel production (l0%)
3. Fossil fuel combustion (11%)

Nriagu (1979 b) estimated the total flux of copper to the atmosphere as 75 tonnes/year, 75% of which comes from anthropogenic sources. The primary anthropogenic emission sources of copper are waste incineration (5.3 tonnes/year) , copper production (20.84 tonnes/year), and wood combustion (11.5 tonnes/year).

In the printing industry, the presence of "soluble copper" has been limited for some time; but it appears that limits on total copper may come under consideration. If this comes about, the continued use of copper phthalocyanine ink, blue and green, would be in question (Benemelis, 1991). The phthalo blue has one atom of copper in every molecule of phthalo blue pigment. In this pigment form, the copper is completely insoluble and, in fact, phtalo blue is one of the most stable of all pigment types (Rusterholtz , 1987).

Hyland (1990) and Rusterholt (1987) noted that the printing industry finds itself in an usually difficult position because it is difficult to find an acceptable pigment that does not include the phthalo blue chemistries. Copper gives the inks the diverse shades of blue. This metal affects the lay of the ink, and attributes related to ink runnability and postpress performance. When copper-based pigments are replaced with other pigments, it requires a lot of money and effort to get color similar to copper-based pigments.

It is significant to note that a non-copper-based phthalo blue has been presented on an experimental basis by the pigments division of Sun Chemical Corp. in an effort to maintain the attributes of phthalo-blue chemistry without the disadvantages of copper. Although progress in ink reformulation is coming along slowly, inks, in the next few years, will have lower levels of copper than they do today (Rusterholtz, 1987, and Hyland, 1990).

Sewage Treatment Plants

Since copper is a widely used material, there are many actual or possible sources of copper pollution. Considerable levels of copper may be found in municipal sewage. Perhaps the most dramatic portrayal of the mobility of copper contamination is the concentration of 24-690 ppm found in sewage sludge. The majority of this copper originates from industrial discharges which find their way into sewage works. Sewage

sludge is frequently applied to land, both as a method of disposal and as a soil ameliorant providing organic matter and the nutrients nitrogen and phosphorus (Nriagu, 1979b).

According to Minear et al. (1981), the heavy metals, including copper, that are loaded to Publicly Owned Treatment Works (POTW's) are of interest because of the possible inhibitory impact on the biological treatment process, the effluent levels that can be reached as a function of concentration, and the practice of land dumping of waste sludge where the total metal load to the treatment plant is of interest.

Around the United States, there are a few states which have had problems with the high concentration of copper in the environment. Leh and Lak (1974) cited high concentrations of copper, ranging from 4.5 to 16.3 ppm, in soil samples from the Grand Rapids, Michigan area. The Federal Government limited toxic discharges of copper and mercury from New York's 14 sewage treatment plants. The metals build up in the tissue of shellfish. New York Harbor shellfish have some of the highest levels of heavy metal contamination in the nation (Gols, 1990).

Rusterholt (1987) stated that limitations on certain metal contents in municipal sewage systems have existed for some time. As more and more testing was undertaken by various state and local authorities, it became evident that the test methods used are an important factor. The standard methods that have evolved for evaluating sewage discharges utilize acid digestion as part of the process. Under these conditions, insoluble suspended solids are chemically broken down, converting all material to soluble form. Under such conditions, it is possible to obtain extraordinary high parts per million values of many chemicals.

Hyland (1990) remarked that there are circumstances where box plants are close to closed because the local sewage authority is saying that the copper levels in their effluents are too high.

Many companies are now closely examining the use of water and realizing that one area where economies can be made is in effluent discharge. Rather than pay water

authorities to manage effluent through public sewers, many companies with effluent discharge problems are now processing their own liquid waste themselves. And where in-house effluent plants were formerly considered to be a refinement which only large-scale industry could afford to invest in, many smaller companies are now concluding that installing their own waste plant need not be as expensive or as disruptive as they originally thought (Environmental Data Services, 1980).

Water Pollution

The median concentration of copper in natural water is 0.004-0.010 ppm. It is primarily in the Cu (II) state. Most of it is complexed or tightly bound to organic matter; little is in the free (hydrated) or readily exchangeable form. Of special concern is copper that gets into drinking water from the water distribution system. When the system has not been flushed after a period of disuse, copper concentration in tap water may exceed the EPA copper water limit of 1.3 ppm. Levels greater than 10 ppm can develop depending on the plumbing system, pH and hardness of water (U.S. Department of Health and Human Services, 1990a). Copper is released to water as a result of natural weathering of soil and emissions from industries and sewage treatment plants. Most of this copper is attached to particulate matter (Nriagu, 1979b).

Soil Pollution

The EPA (1990a) estimated that 97% of copper released into the environment is applied to soil. The forms are primarily tailings and overburden from copper mines and tailings from mills. Other releases to land consist of municipal refuse and sludge from publicly owned treatment works (POTWs). The copper content of municipal solid waste is 0.16 percent. Much of the sludge is landfilled directly or as a residue from incineration.

The addition of sewage sludge to agricultural soil improves physical aspects and acts as a valuable source of nutrients to growing crops. However, there is increasing concern over the use of sewage sludge which has been polluted by heavy metals. Until recently, studies have concentrated on the uptake and transport of heavy metals into the food chain via crops. Now, however, there is growing evidence of an adverse effect on microbial processes related to nutrient cycling in these types of soils (Donker et al., 1994).

Most of the copper in soil is apparently tightly bound to soil components and may not be accessible for uptake. While investigations show that copper does not leach significantly from soil, levels of copper as high as 2.8 ppm have been found in some groundwater (U.S. Department of Health and Human Services, 1990a).

Air Pollution

Copper is discharged into the air naturally from windblown dust and volcanoes, and from anthropogenic sources. The mean concentration of copper in the atmosphere is 5-200 ng/m^3 . The amount of copper and other pollutants in wind blown dust from a waste site is of some concern. In one study, the amount of airborne copper and other heavy metals accumulated near a large refuse dump that received municipal and industrial

waste and sewage sludge was determined. First measuring the amount of the metal accumulated was measured. The deposition rate was then determined and compared with that for an agricultural control area. The mean copper deposition rate was twice as high near the dump as in the control area (U.S. Department of Health and Human Services, 1990 a).

Effect of Copper Pollution on Terrestrial Animal Life

According to Nriagu (1979b), excessive intake of copper can have significant effects on animals. Depending on the species involved, growth rates and food intake may be cut down, anemia can develop, and considerable damage may be done to the liver, kidneys, brain, and muscle, often resulting in death. Luckily the tolerance to copper of most domestic and laboratory animals is relatively high. Copper poisoning is a much less serious problem in animal husbandry than is copper deficiency. It is often necessary to increase dietary intakes of copper by 20- to 50-fold over normal levels before any damages caused by copper toxicity develop. However, this is not invariably the case. With some species, copper poisoning may occur quite readily, even under natural conditions.

Up to 15 ppm of copper is generally recognized as safe (GRAS) by the FDA in livestock feed. Cattle can tolerate mineral mixtures and feeds with added copper . In contrast, sheep are susceptible to the toxic effects of added copper. Adding excess copper to sheep, swine, and poultry feeds may create a hazard for the consuming public because the metal accumulates in the animal's liver (National Research Council, 1977). Pigs are much more tolerant of copper than are ruminant animals and under normal situations would be considered unlikely to suffer from copper poisoning (Nriagu, 1979b).

Effects of Copper Pollution on Aquatic Life

Baatrup (1991) explained that the increasing discharge of wastes from industrial and agricultural activities has dramatically changed the conditions of aquatic life. The dissolved toxicants act directly and continuously on aquatic animals. This continuous exposure to chemical pollution, even at low sublethal doses, may cause serious damage to aquatic life processes.

Copper in water is exceedingly toxic to aquatic biota, in contrast to its low toxicity to mammalian consumers of water. Concentrations as low as 5 to 25 ug/l are lethal within 4 days to some invertebrate and fish species. The suggested standard for public water supplies, based on palatability, is 1,000 ug/l. The extreme sensitivity is a consequence of high surface-volume ratios of algae. High respiratory water flows, plus an extensive, highly permeable gill surface area that facilitates rapid uptake of large amounts of copper, makes invertebrates and fish sensitive. The sensitivity to waterborne metals is analogous to mammalian sensitivity to airborne metals as a result of the rapid and continuous respiratory uptake. Ingested copper is available only in limited amounts since freshwater biota generally do not drink water, and fecal material vies with the stomach and intestinal tract for absorption of ingested copper (Nriagu, 1979 b).

Copper Toxicity to Aquatic Invertebrates

Some concentrations of copper have been found to be acutely toxic to invertebrates. Sensitivity to copper differs greatly from species to species. A portion of the variation in toxicity among species is related to the nature of the body covering. In general, larvae or younger stages are more sensitive to copper than are adults. Adaptation to high copper concentrations in water or sediment appears to occur but is probably due to genetic selection of hardy individuals rather than acclimation. In addition to reduction

in survival rate, growth, and reproduction, copper also causes histopathologically observable tissue damage, decreased oxygen consumption, or distress conduct in invertebrates. The mode of operation of copper in invertebrates is not well known, although impairment of osmotic and ionic regulation may be one possible cause of death (Nriagu, 1979 b).

Copper Toxicity to Fish

Ashraf et al. (1992) declared that fish are known for their ability to gather trace metals from seawater and sediment. The processes leading to trace metal mobilization and resorption by sediments are known specifically to influence the persistence, toxicity, uptake and transport in fish. Relationships between tissue concentrations and the mechanisms involved in the bioaccumulation of trace metals in fish have evidenced a strong dependency between trace metal and characteristic of each type of species.

Copper is an essential metal, an integral part of several enzymes. However, at elevated levels, copper is highly toxic to fish, especially in fresh water because of the higher contents of ionic copper (Baatrup, 1991).

According to Nriagu (1979b), many factors affect lethal copper toxicity to fish through changes in the availability of copper to the fish, and the sensitivity of the fish to a given amount of copper taken up.

Lethal effects may be observed over a wide range of concentrations (23 to 10,200 ug/l); the variations are primarily a result of the effects of water hardness, organic complexing capacity, and species sensitivity. Numerous histological, physiological, and enzymatic responses of fish to near-lethal copper exposures suggest that osmoregulatory failure during exposure to the metal is the probable cause of death. Sublethal effects of copper in fish occur at concentrations up to the lethal level. The initial signs of toxicity, however, can be observed at concentrations less than 160 ug/l. The behavior and growth

of salmonids (fish of the family Salmonidae) are the most sensitive parameters. Avoidance, activity, appetite, growth, and migration are affected by copper concentrations between 4 and 10 ug/l. Physiological responses, including inhibition of gill ATPase (adenosine triphosphatease), plus short term corticosteroid elevation, were also observed between 5 and 10 ug/l. Threshold effects on reproduction, growth, and mortality, and hence fish production, were observed between 10 and 20 ug/l for salmonids, catfish, and walleyes, between 18 and 40 ug/l for fathead minnows, and between 40 and 160 ug/l for bluegills. Finally, disease was induced in eels, salmonids, suckers, and cyprinids at 30 to 60 ug Cu/l. These data indicate that fish populations are adversely affected at copper concentrations well below the lethal level, so that some species could disappear without direct observable mortality (Nriagu, 1979b).

According to Baatrup (1991), the toxicity of copper seems to result from its interaction with cellular membranes. Increased free radical formation and lipid peroxidation may lead to severe cellular stress.

Fish depend on their external and internal senses for mediating behavior such as food search, predator recognition, communication, orientation, and migration. Stimuli are perceived by specialized sensory structures and converted into electrical signals, which are conducted to the central nervous system. Here, neural information is integrated and appropriate behavioral responses are generated. Unfortunately, the nervous system is one of the most vulnerable parts of the animal body, and injuries to its elements may strongly influence the behavior and survival of the organism. In fish, both acute and chronic injuries to the nervous system can occur. Dissolved toxicants, including copper, act directly on superficial sense organs, including olfactory and taste organs, which are not protected by "external barriers" or internal detoxifying systems. Pollutants may disrupt normal chemosensory function by masking or counteracting biologically relevant chemical signals, or they may cause direct morphological and physiological damage to the receptors. In addition, pollutants absorbed across the skin or the gills may enter the blood

stream and thereby reach internal sense organs and other nervous tissues, including the brain (Baatrup, 1991).

A study by Curby et al. (1976) showed that freshwater adapted fish used in copper pollutant tests were less able to adjust to new experimental situations than test fish taken from a saltwater tank. The freshwater adapted fish appeared to be the most affected by the metal insult. They were the most sluggish, did not eat when fed or seem to sense the food in the water; they were the first to die in the adverse conditions. This may mean, in a larger sense, that even fish in the wild that appear to be adapted to slightly deleterious conditions are less able to withstand any subsequent change in the environment. Such fish would be more susceptible to a lower level of pollutants in the water than would fish in a "healthy" environment.

Effects of Copper Pollution in Plants

A study by Madry (1978) shows that suppression of growth and reduced productivity of plants by air pollution are due to the way in which contaminants affect the physiology of the plant itself. These injuries interfere unfavorably with some or all of the normal plant processes such as photosynthesis, respiration, transpiration, cell permeability, root development, etc. , all of which are essential for plant growth. Copper and its various salts are highly toxic to lower forms of life such as algae, bacteria, etc. In protein synthesis and photosynthesis, excesses of copper, zinc, and other metals may be substituted in these processes, replacing iron, magnesium, manganese, or other desirable metals. Excesses of the element in higher plants have also become toxic. It is apparent that uncontrolled use and undesirable accumulation of copper must be avoided.

Donker et al. (1994) concluded in their study that contaminants were accumulating on the leaf surfaces and entering the plants through the stomates. At the same time, some of the metals were entering the plant through a root system that had been damaged by the metals or a myriad of other factors. Both sources contributed to the excess in plant accumulation of the metals and suppression of growth. At increasing metal concentrations in the environment, heavy metal-sensitive species disappear. However, a few angiosperms can still be found on soils with toxic heavy metal concentrations (Zn up to 60,000 mg/kg, and Cu 7,000 mg/kg).

CHAPTER 3

HEALTH EFFECTS ASSOCIATED WITH COPPER IN HUMANS

Introduction

In 1977, the National Academy of Sciences stated that the shortage of literature on ill effects caused by exposure to copper and its compounds in industry suggests that copper is not a particularly hazardous industrial substance. However, if workers are subjected to excess concentrations of the metal in any of its forms, unwanted health effects can result.

According to Venugopal and Luckey (1983), the range between deficiency and toxicity of Cu is ample for mammals. In humans, manifestations of copper toxicosis do not occur until much higher levels have been reached than in other animals. The copper content of a 70 kg (154 lb) human adult is between 80 and 150 mg, and the average daily dietary intake by a normal adult is about 4-5 mg. The range of requirement of copper by human beings is 1.0-3.8 mg/day (Lee, 1972).

The Copper and Brass Research Association (1947) pointed out that several factors determine whether damaging health effects will occur and what the type and severity of those health effects will be, if a person is exposed to copper. These factors include the dose, the duration, the route or pathway of exposure (breathing, eating,

drinking, or skin contact), other chemical exposure, and individual features such as age, sex, nutritional status, family traits, life style, and state of health.

The following section analyzes two factors: large dose ingestion, and fumes and dust exposure .

Large Quantities of Copper Ingested

Accidental ingestion of large amounts of copper salts has been discussed by Friberg (1977), the National Academy of Sciences (1977), Carson and Ellis (1986), and Dillon (1991). They defined the consequences of overdoses of copper ingested by humans. The chain of effects is as follows:

1. An instantaneous metal taste.
2. Gastrointestinal disturbances such as epigastric burning, nausea, vomiting and diarrhea.

These manifestations generally protect the patient from serious systemic effects such as hemolysis, liver and renal damage, oliguria, azotemia, hemoglobinuria, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, or death.

After suicidal ingestion of a large quantity of copper sulfate, jaundice and renal harm have occurred at average copper concentrations in blood of about 8,000 mg/l, whereas at a copper level of about 3,000 mg/l only gastrointestinal disturbances were seen (Friberg, 1977).

Copper Fumes and Dust Toxicity

Industrial exposure to copper dust or fumes has been frequent, but health surveys of workers engaged in the processing of copper have not revealed signs of chronic illness (Friberg, 1977).

Dusts and fumes from copper and its compounds usually have an intolerable taste, a warning that tends to cause humans to limit exposure before serious toxic intake can occur. However, metal fume fever from exposure to copper can happen (National Academy of Sciences, 1977).

The Occupational Safety and Health Administration (OSHA) has assigned a limit to protect workers of 0.2 mg/m^3 of copper fumes and 1 mg/m^3 copper dusts and mists in occupational air during an 8-hour work shift. The National Institute for Occupational Safety and Health (NIOSH) suggests that the concentration in workroom air be limited to 0.1 mg/m^3 for copper fumes and 1 mg/m^3 for copper mist, averaged over an 8 hr work shift (U.S. Department of Health and Human Services, 1990 a).

The U.S. Department of Health and Human Services (1990a), discussing several reports of human exposure to airborne copper, stated that three reports addressed effects different from metal fume fever. One study examined 100 factory workers over a period of 4 years. The workers sieved copper dust, the purity of which was 99.9%. The reported copper levels in the air were 464 mg Cu/m^3 in the first year, 132 mg Cu/m^3 in the second, and 111 mg Cu/m^3 during the third. The concentration of copper in the air was not reported for the fourth year, but was assumed to be less than 111 mg Cu/m^3. Effects of exposure were found on the respiratory system, liver, gastrointestinal system, reproductive system, nervous system, and sella turcica.

Exposure to copper dust can produce several reactions in the human body:

1. Metal Fume Fever
2. Respiratory Effects
3. Anemia and Hemolytical Effects
4. Dermal / Ocular Effects
5. Gastrointestinal Effects
6. Neurological Effects
7. Reproductive Effects
8. Other Systemic Effects
9. Heart Disease

Metal Fume Fever

Inhalation of copper fumes or fine copper dust may cause so called metal fume fever. Boyce (1961) found that metal fume fever is caused by inhaling rather heavy concentrations of dust or fumes. The most potent particle size range appears to be less than 0.6 microns. According to Carson and Ellis (1986) and Friberg (1977), metal fume fever is an influenza like syndrome with symptoms which dissipate after 24 hours. Fume fever symptoms of respiratory irritation, chills and aching muscles were discussed by Baselt (1988). Chronic copper poisoning in industry was related to anorexia, nausea, vomiting, nervous manifestations and hepatomegaly (enlargement of the liver). Serum copper concentrations ranged from 0.8 to over 2 mg/l in such cases. Boyce (1961) stated that a worker recuperating from fume fever is fairly immune and generally can experience another inhalation without experiencing a second attack.

Gleason (1968) reported that metal fume fever appeared after exposure to about 0.1 mg/m^3 of fine copper dust. A number of workers who acquired copper fume fever had serum copper levels that averaged 1.26 mg/l.

Copper fever has been found among men handling copper oxide powder in a paint factory, and copper acetate dusts have caused complaints of sneezing, coughing, digestive disorders, and fever. Visible metal fume fever has also been reported in three men who were exposed to dust produced during the polishing of copper plates (National Academy of Sciences, 1977).

Respiratory Effects

In humans, copper is a respiratory irritant. Plant employees exposed to copper dust experienced mucosal irritation of the mouth, eyes, and nose (U.S. Department of Health and Human Services, 1990a). Venugal and Luckey (1983) showed that inhalation of dusts and fumes of metallic Cu and its salts produces congestion of nasal mucous membranes, ulceration and perforation of the nasal septum, and pharyngeal congestion.

The National Academy of Sciences (1977) and Friberg (1977) reported that pulmonary copper deposition and fibrosis happened in the lungs of some vineyard workers after years of exposure to a pesticide with a high content of copper. Later granulomas and malignant tumors appeared in these laborers' livers and lungs.

Anemia and Hemolytic Effects

Decreased hemoglobin and erythrocyte levels have been observed in workers exposed to airborne copper levels of 0.64-1.05 mg/m^3. Results of hair analysis showed that the workers were also exposed to iron, lead and cadmium (U.S. Department of Health and Human Services, 1990a). Mild, possibly hemolytic, anemia has been observed in workers exposed to copper in the air at levels at or below the TLV (Carson and Ellis, 1986).

Hepatic Effects

Hepatomegaly (big liver) was observed in factory workmen exposed to copper dust (U.S. Department of Health and Human Services, 1990a). In two autopsied cases of so called "vineyard sprayers lung", hepatic granulomas were found (U.S. Department of Health, 1990).

Dermal / Ocular Effects

Gafaber (1967) found that brass dust and slivers may cause dermatitis by mechanical irritation, and Boyce (1961) added that skin conditions due to handling copper powder can make trouble during hot humid weather. Some individuals seem to be more sensitive than others and raw copper powder seems to cause considerably more trouble than after it has been blended with a stearate lubricant.

Mucosal irritation of the eyes has been seen in factory workers exposed to copper dust (U.S. Department of Health and Human Services, 1990a). The National Academy of Science (1977) affirmed that presence of a particle of metallic copper in the eye may result in loss of the eye, sunflower cataracts, or visible deposits of copper in the cornea known as Kayser-Fleischer rings.

Gastrointestinal Effects

Anorexia, nausea, and occasional diarrhea were described in factory workers exposed to copper dust. A portion of the copper in the air was probably ingested; thus, the gastrointestinal effects were probably the result of oral exposure to copper (U.S. Department of Health and Human Services, 1990a).

Neurological Effects

Headache, vertigo, and drowsiness were reported in factory workers exposed to copper dusts (U.S. Department of Health and Human Services, 1990a).

Reproductive Effects

Sexual impotence was reported in 16% of inspected factory workers exposed to copper (U.S. Department of Health and Human Services, 1990a).

Other Systemic Effects

Seven cases of enlargement of the sella turcica, nonsecretive hypophyseal adenoma, accompanied by obesity, arterial hypertension, "and red faces" in factory workers exposed to copper dust were reported (U.S. Department of Health and Human Services, 1990a) .

Heart Disease

A high concentration of copper in the blood may be a risk component for coronary disease, along with raised blood cholesterol and cigarette smoking. According to Webb (1991), Frei and Gaziano (1993), Chiu, Jeng and Shieh (1994), and Lamb and Leake (1994), the copper appears to work in combination with cholesterol to further atherosclerosis, the thickening of the arterial wall, a high level of low-density lipoprotein (LDL) cholesterol in the blood, a high level of copper, and a low concentration of selenium.

CHAPTER 4

ENVIRONMENTAL AND HEALTH EFFECTS ASSOCIATED WITH ZINC

Environmental Effects Associated with Zinc

Animal Toxicity

Inhalation

Lam et al. (1985) found functional, morphologic, and biochemical changes in the respiratory tract of guinea pigs exposed to 5 mg/m^3 zinc oxide for 3 hours/day for 6 days. Amdur et al. (1982) reported on the pulmonary response of guinea pigs to zinc oxide fumes. The animals were exposed to approximately 1 mg/m^3 of freshly formed zinc oxide. Conner et al. (1982,1985) studied the irritancy potential of a combination of zinc oxide and sulfur dioxide. Guinea pigs were exposed to 6 mg/m^3 zinc oxide mixed with 1 ppm sulfur dioxide, for 3 hours a day for 6 days. Total lung capacity, vital capacity, functional residual volume, alveolar volume and diffusing capacity were decreased following exposure and had not returned to normal 72 hours after exposure. Similar but more severe changes were seen after a single 3-hour exposure of 25 mg/m^3 zinc oxide and sulfur dioxide. Hilderman and Taylor (1974) reported a case of acute emphysema in cattle exposed to zinc oxide fumes emitted during oxyacetylene cutting and welding of

galvanized pipe. Three heifers were severely affected, and died within a short time. The animal autopsy showed severe changes in lungs with edema, emphysema, and hemorrhages. In this case, a galvanized material was implicated, but the extremely severe condition caused by the fumes indicated either that cattle are highly sensitive to zinc oxide fumes, or that other metals, such as cadmium, may have been involved. Harding (1957) administered, 50 mg of zinc stearate to rats by intratracheal instillation. Approximately 50 percent were dead after dosing. Beeckmans et al. (1963) described the effects of zinc oxide fumes on rats. 132 rats were exposed to 400 to 600 mg/m ^{3} of zinc oxide fumes for 10 to 20 minutes. 16 rats died during the experiment. Other rats showed a marked fall in body temperature, which was increased when zinc oxide was irradiated by ultraviolet light for 50 seconds. In sacrificed animals, 1,100 - 5,500 mg of zinc for each gram of lung tissue was found.

Oral

Sampson et al. (1942) reported the effect of ingestion of zinc lactate in pigs. Pigs were fed 17.5 g of zinc lactate for 9.5 months. They began losing their appetite after only a few weeks on the diet. Symptoms of stiffness and lameness were also found. Autopsy findings revealed pathologic lesions in the joints and an increased liver zinc content. Brink et al. (1959) and Hill et al. (1983) also studied the arthritic condition by feeding pigs with 500 to 8000 mg/kg zinc. In addition to the arthritic condition characterized by swollen joints, at feeding levels of 2000 mg/kg and above, test animals exhibited depressed weight gain and food consumption. Brink et al. (1959) also found dosage - related increase in deaths. Postmortem examination revealed extensive hemorrhaging in axillary spaces and intestine and marked gastritis with some ulceration.

Smith and Embling (1984) reported the effect of zinc ingestion in sheep. Animals were administered 240 mg zinc/kg as zinc oxide or zinc sulfate, three times a week for 4

weeks. This produced pancreatic damage in all exposed animals. Animals ingesting zinc sulfate also experienced severe diarrhea which commenced after a week of dosing and persisted throughout the experiment. All animals in the zinc sulfate group died after 13 days. Postmortem examination revealed a reduction of the papillation of the rumen wall and edema of the fundic folds of the abomasum. The liver had a finely mottled surface and an orange brown color.

Dewar et al. (1983) studied the effects of excessive dietary zinc oxide in chicks and hens. Chicks were maintained on a diet containing 2,000, 4,000, or 6,000 mg/kg for 42 days or 1,000, 2,000, or 4,000 mg/kg for 28 days while hens received 10,000 or 20,000 mg/kg for 4 days. Mortality was high in chicks receiving 4,000 and 6,000 mg zinc/kg. Postmortem examination revealed macroscopic abnormalities of the alimentary tract. In five chicks in the 6,000 mg/kg group, there was internal hemorrhaging from the descending aorta or the thoracic aorta. Histological examination revealed gizzard and pancreatic lesions in all exposed groups. There was no mortality reported for the exposed hens; however gizzard and pancreatic lesions were found in both exposed groups.

Carcinogenicity

Leonard and Gerber (1989) studied small mammals and showed that zinc is cocarcinogenic with 4-mitroquinoline-N-oxide on oral cancer and with N-ethyl-N-nitrosourea on brain cancer. There is conclusive evidence that repeated intratesticular injections of zinc salts can induce testicular sarcomas in birds and rats. Zinc and zinc compounds are not conclusively carcinogenic except when injected directly into the testes; no field or experimental evidence exists showing zinc to be tumorigenic through any other route. Zinc is essential for the growth of rapidly proliferating cells such as tumors.

Accordingly, growth of animal tumors is stimulated by zinc and retarded by zinc deficiency .

Under conditions of high gonadal activity, the injection of zinc salts into testes of fowl has induced testicular tumors. Nriagu (1980) also reported that seminomas, interstitial cell tumors, and teratomas occurred in rats after testicular injection of zinc salts.

Teratogenicity

No conclusive evidence has shown that excessive zinc produces any teratogenic effect in mammals (National Association of Sciences, 1979; Dawson et al., 1988; Leonard and Gerber, 1989). However, Dawson et al. (1988) showed that excess zinc is teratogenic to frog and fish embryos, possibly by inhibition of DNA synthesis.

Reproduction

Samanta and Pal (1986) and White (1955) reported that excessive dietary zinc may adversely affect fertility in humans and lower animal forms.

Zinc Hazards to Fishes

Most of the zinc introduced into aquatic environments is eventually partitioned into the sediments. According to Skidmore (1967), zinc bioavailability from sediments is enhanced under conditions of high dissolved oxygen, low salinity, low pH, and high levels of inorganic oxides and humic substances.

Several documents report the toxic effects of zinc salts on the gills of experimental fishes. Skidmore (1967) reported on epithelial damage of gills which decreased the permeability of the gills to oxygen. Skidmore and Tovell (1972) reported on the effect of acute exposure of rainbow trout to 40 mg/l of zinc sulfate. This caused an acute inflammatory reaction in the gill with a separation of the epithelium outward from the pillar cells. This was followed by circulatory breakdown, tissue destruction, respiratory collapse, and death. Matthiessen and Brafield (1973) demonstrated the effects of dissolved zinc on the gills of the stickleback. Detachment of the epithelial cell upon exposure to zinc was found. Burton et al. (1972) confirmed an earlier hypothesis that the major physiological change preceding death in acute toxicity studies with zinc was tissue hypoxia. The hypoxia was directly related to gill tissue damage which disrupts normal gas exchange at the gill surface

Other experiments also showed that fish eggs and fry are extremely sensitive to zinc poisoning. Pickering and Vigor (1965) indicated very great sensitivity to zinc of eggs and fry of the fathead minnow. He found median tolerance limits (TL_{50}) of 3.92 to 3.98 mg/l for 1 day old eggs. The TL_{50} values continued to drop until on the 12th day the level had fallen to 1.97 to 1.69 mg/l. Newly hatched fry exposed for 2 days to zinc sulfate had a TL_{50} of 0.95 mg/cc. Skidmore (1966, 1967) studied the effects of zinc sulfate on zebra fish eggs. The result showed that sensitivity to zinc sulfate increased during the course of embryonic development. Later embryonic stages of 2 to 4 days and newly hatched fry 4 to 13 days old were highly susceptible to the toxic effects of zinc. Birge and Just (1975) reported that zinc appeared to be more toxic to late embryonic stages and newly hatched fry.

Lloyd (1960) studied the cause of death of fish in solutions of zinc sulfate. The reason was not from the precipitation of mucus on the gills but probably from zinc-induced damage to gill epithelium. Crandall and Goodnight (1962) found that guppies reared in zinc solutions experienced stunted growth, had a higher mortality rate, and

showed less sexual dimorphism. The report also showed many abnormalities among the internal organs. The liver had degenerated, the pancreas was undersized, the kidneys were distorted and hemorrhaged, and the skeletal muscles were underdeveloped.

Bengeri and Patil (1986) showed that signs of zinc poisoning in fish included hyperactivity followed by sluggishness; before death, fish swam at the surface. They were lethargic and uncoordinated, showed hemorrhaging at gills and base of fins, shed scales, and had extensive body and gill mucus. Eisler and Gardner (1973) indicated that acute exposures to high lethal concentrations of zinc caused tissue damage of epithelia lining the oral cavity. Reed et al. (1978) studied the acute toxicity effects of varying concentrations of zinc on certain fishes native to Illinois. Fourteen-day bioassays were performed with bluegill fry, channel catfish fingerlings, and largemouth bass fingerlings in waters relatively high in alkalinity and the salts of calcium and magnesium. The result indicated that the 14-day median tolerance limit at $20^{o}C$ was 11.0 mg/l soluble zinc for blue gill, 8.2 mg/l soluble zinc for the channel catfish, and 8.0 mg/l soluble zinc for the largemouth bass.

Plant Toxicity

Brenchley (1941) reported that the normal levels of zinc range from 10 to 100 mg/kg in most crops and pasture plants. Zinc was early established as essential for the growth of higher plants. However, there are some reports indicating the toxicity of zinc in plants. That is mostly seen in the areas close to some emission source. Sensitive terrestrial plants died when soil zinc concentrations were more than 100 mg/kg.

Toxicity of Mixtures of Zinc and Copper

Several reports indicate the toxicity of mixtures of zinc and copper. Sprague (1986) suggested that mixtures of zinc and copper are generally acknowledged to be more-than-additive in toxicity to a wide variety of aquatic organisms. Venugopal and Luckey (1973,1975) reported that zinc toxicity can be alleviated by Cu and Fe. Klevay (1973) reported that an imbalance between zinc and copper is an important factor in the production of hypercholesterolemia in rats. He also suggested that animal fat ingestion and the zinc: copper ratio in milk may be important in the etiology of cardiovascular disorders in man. These hypotheses have not yet been fully tested and are not generally accepted. Samman and Roberts (1988) reported that high levels of administered zinc limits copper uptake in humans and certain animals. Saxena et al. (1989) suggested that excessive zinc in humans interferes with copper absorption from the intestine, resulting in copper deficiency and eventually in cardiovascular discases; high zinc intakes also decrease iron bioavailability, leading to a reduction of erythrocyte life span by 67%.

Health Effects Associated with Zinc in Humans

Exposure

According to the EPA (1987b), humans are exposed to zinc through the inhalation of air and the ingestion of food and water. Zinc levels in air are generally less than 1 mg/m^3. Assuming that an individual inhales 20 m^3 of air per day with an average zinc concentration of 1 mg/m^3, the daily zinc contribution from this source would be 20 mg.

The EPA (1980b) reported that zinc dietary intake is 18 to 18.6 mg/day for males age 15 to 20 years old , whereas the dietary intake by girls 12 to 14 years old was 10 mg. Nriagu (1980) reported daily zinc dietary intakes of 14 to 80 mg.

Inhalation Toxicity

According to EPA (1987b), Prasad (1993) and Baselt (1988), in certain occupational settings, the inhalation of zinc oxide fumes as well copper fumes, as mentioned before, produces a disease known as metal fume fever. The disease is initiated by inhalation of zinc oxide fumes. Venugopal and Luck (1978) stated that zinc oxide fumes will accumulate in the lungs before being absorbed into the blood. Zinc is found in high concentrations in bone, skin, prostate gland, choroid of the eye, and serum. It is distributed to all organs and tissue of mammals. Metal fume fever usually occurs within a few hours after inhalation and has a short duration, about 6-48 hours. Fume fever symptoms from zinc oxide fume inhalation differed from copper fume inhalation.

Distinctive manifestations of zinc oxide fume fever are headache, leukocytosis and sweating and of copper fume fever are chills, anorexia, vomiting, nervous manifestations and enlargement of the liver. Metal fume fever generally strikes at the beginning of the work week when the worker has not been exposed for two days, and so it has been called "Monday Fever". Further repeated exposure does not cause any new symptoms, suggesting some type of adaptation.

Carcinogenic and teratogenic effects

Excess zinc may have carcinogenic effects. Kok et al. (1988) reported data on mortality from cancer and cardiovascular disease correlated with zinc and copper status, in a population from the Netherlands.

The EPA (1987b) concluded that many human studies have documented the level of zinc in both cancerous and noncancerous tissues, and the zinc content has been found to be both high and low with no definite pattern. Carson and Ellis (1986) tested laboratory animals which were injected with zinc salt, inducing testicular tumors. However, zinc appears to be indirectly involved, since zinc is required for tumor growth. Pounds (1985) has described metal carcinogens causing genetic damage in short term tests for mutagenicity in bacteria, yeast or mammalian cell-culture. The evidence showed the types of tumors caused by zinc excess include leydigioma, seminoma, chorionepithelioma and teratoma.

Recent research on the effects of zinc excess in animals has shown positive teratogenic effects. A report, by Luo et al. (1993), demonstrates that Zn^{2+} is strongly teratogenic for *Xenopus embryos,* south African frog.

Risk of progression to AIDS

High intakes of zinc may be monotonically and significantly associated with an increased rate of progression to acquired immunodeficiency syndrome (AIDS). Tang et al. (1993) investigated the different levels of dietary intake of micronutrients by a group of 281 HIV-1 seropositive homosexual/bisexual men. Participants completed a self-administered semiquantitative food frequency questionnaire as baseline. The data shows increased intake of zinc associated with risk of developing AIDS.

CHAPTER 5

ENVIRONMENTAL EFFECTS ASSOCIATED WITH HEXAVALENT CHROMIUM

According to the Environmental Criteria and Assessment Office (1984), the industries that employ chromium are metallurgy (329,000 metric tons/year), manufacture of refractories (97,000 metric tons/year), and the chemical industry (114,000 metric tons/year). The primary chromium chemicals are sodium chromate and sodium dichromate, which are converted to other compounds including pigments and catalysts. Pigments consume approximately 29,000 metric tons/year and chromate catalysts less than 2,000 metric tons/year of chromium.

Nriagu and Nieboer (1988) and Elias et al. (1989) classified chromate pigments in two categories: corrosion inhibition and color pigments. Corrosion inhibition pigments based on zinc chromates, strontium chromates and barium chromates, are used in coatings to prevent the corrosion of metals. Chromate color pigments are mostly lead chromates, which include the yellows (lemon, primrose, and medium yellows), chrome oranges, molybdate orange, chrome green, and Guinet's green. They are used in printing inks, rubber, paper, etc.

The Lead Chromate Committee (1987) described chrome yellow and molybdate orange as valuable color pigments with technical and economic benefits over most alternative pigment types. They impart bright and stable colors to printing inks, as well as surface coatings and plastics. Their bright colors, opacity, light-fastness, good heat resistance, and complete freedom from bleed make replacement with other colors difficult and costly.

According to Sullivan (1969), chromic phosphate is utilized for green pigments, chromic potassium sulfate is used in the manufacture of inks, chromic sulfate is used in the manufacture of green inks, lead chromate (VI) is used as a pigment in oil and water colors; basic lead chromates are used as pigments (colors from brown-yellow to red); potassium dichromate (VI) is used in printing photolithography and pigment prints; sodium dichromate (VI) is used in the manufacture of chrome pigments and dyes; and ammonium dichromate (VI) is used in lithography and photoengraving.

OSHA (1979) stated that chromate pigments are used in colored plastics (polyesters, vinyls, thermosets, polystyrenes) employing chrome yellow, chrome orange, chrome green (lead chromate), molybdate orange and zinc chromate. Chromate pigments are also employed, in a limited amount, for colored elastomers (urethanes and nonsulfur vulcanized elastomers) using chrome yellow and chrome green.

Landfill

According to the U.S. Department of Health and Human Services (1993), a higher level than normal of chromium can be expected near landfill sites with chromium-containing wastes, and near industrial facilities that manufacture or use chromium and chromium-containing compounds.

Kapil and Keogh (1990) cite a case in Hudson County, New Jersey in the 1960's and 1970's where slag containing chromium in carcinogenic forms and in acutely toxic concentrations was used as landfill in residential, commercial, and recreational settings in over 100 locations. Community exposure from this fill occurred in a variety of ways: wind, soil erosion, chromium compounds leached by rainwater, and children playing in areas where slag was used as fill.

Nriagu and Nieboer (1988) discussed groundwater contamination by chromium in industrialized areas. Typically, chromium-containing wastes have been disposed of by discharge to surface impoundments or lagoons. Leakage from these lagoons into groundwater has been relatively common. Almost all reported incidence of chromium-related groundwater contamination are of industrial origin.

Air

According to Towill et al. (1977), the total chromium atmospheric emissions are approximately 11,000 to 16,000 tons/year in the United States. Nriagu and Nieboer (1988) estimated that emissions from anthropogenic sources were about 5,000 tn/year in 1978. According to the U.S. Department of Health and Human Services (1993), anthropogenic emissions decreased to 2,700-2,900 tn/year by 1993.

The U.S. Department of Health and Human Services (1993) estimated U.S. atmospheric chromium emissions from anthropogenic sources as : combustion of coal and oil 1,723 tn/year (0.2% Cr(IV)), chromeplating 700 tn/year (100% Cr (IV)), sewage sludge incineration 133 tn/year (less than 0.1%Cr (VI)), municipal refuse incineration 2.5 tn/year (0.3% Cr (VI)) and chromium chemical manufacturing 18 tn/year (67% Cr (VI)).

The Environmental Criteria and Assessment Office (1984) reported an air concentration of total chromium of 0.005 ng/m^3 at the South Pole. In the United States,

reported values were between 0.005 and 0.157 ug/m^3. The total yearly deposition of chromium in urban areas may vary from 0.12 ug/m^2 to 3 ug/m^2 . In general, urban areas have higher total deposition than rural areas. No federal or state ambient air chromium standards have been proposed.

Water

Shepherd and Jones (1971) reported concentrations of chromium in seawater of 0.05 ug/ l. In one survey, the chromium content of 24 municipal water supplies was found to be from 1 to 40 ug/l. The chromium content of marine animals generally falls between 200 and 1,000 ug/l.

The Environmental Criteria and Assessment Office (1984) described the origin of chromium in surface waters as surface runoff, deposition from air, and release of municipal and industrial wastewaters. Most of the chromium present in surface water was hexavalent

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The U.S. Department of Health and Human Services (1993b) has assigned the maximum level of Cr (III) and Cr (VI) allowed in drinking water that is not expected to cause effects that are harmful to health: 1,400 ug chromium/l for 10 days of exposure for children, 240 ug chromium/l for longer-term exposure for children, 840 ug chromium/l for longer-term exposure for adults, and 120 ug chromium/l for lifetime exposure of adults. In contrast, Kapil and Keogh (1990) mentioned a EPA recommended concentration for drinking water of 50ug/l Cr (VI) because of the toxic effects of Cr (VI) and possibility of oxidation of Cr (III) to toxic Cr (VI).

Soil

Chromium in the soil exists predominantly in the trivalent oxidation state. The main origin of chromium that contaminates the soil is fallout and washout from the atmosphere. Another source is sewage sludge when it is used as a fertilizer (Towill et al., 1977). Eisler (1986) observed that extremely high levels of Cr (VI) in sludge components may have serious effects on wildlife when the sludge is applied to croplands.

The chromium quantity in the soil, according to the Environmental Criteria and Assessment Office (1984), varies with soil origin and degree of contamination from anthropogenic sources. Tests on domestic soil have shown chromium concentrations ranging from an average of 14-70 ppm. Because the amount of chromium in food and food plants is relatively low, and because chromium does not appear to accumulate in mammalian systems, bioaccumulation in the soil-plant-animal system does not appear to be a significant exposure source.

Effects of Chromium on Vegetation

According to Krupa et al. (1982), plants, in general, tolerate soluble chromium in amounts of 0.06-50 ppm on average. The most sensitive plants are tobacco, maize and oats. The highest accumulation of chromium in plant organs appeared in the roots. Chromate taken up into leaves caused changes in the content of plastid pigments and lipoquinones similar, in general, to those in senescent plants.

Sullivan (1969) related dependency of chromium effects on plant type, chromium concentration in the soil, and availability of the chromium to plants. In soils less acidic than pH 4, very little chromium is available to plants. When small amounts of chromium are available, the growth of oats and barley is stimulated. However, even a smaller amount is toxic to wheat. Toxic effects may be expected in plants when the levels of available chromium in the soil exceed 7-10 ug Cr (VI) /g of soil. Nevertheless, some plants may tolerate as much as 100,000 ug Cr (VI)/g.

Anderson (1982) associated effects of chromium with alterations in the plant roots and symbiotic soil microorganisms. Such impairment impedes the transport of nutrients to plant tissues and therefore reduces plant growth and viability. Inhibition can occur at concentrations as low as 10 ppm.

The National Academy of Sciences (1974) gave examples for water plants where hexavalent chromium at 0.03-64 ppm inhibited the growth of algae, whereas lower concentrations stimulated growth in some cases. Hexavalent chromium at 1-5 ppm in seawater reduced photosynthesis of giant kelp macrocystic pyrifera by 20 to 30 percent after 7 to 9 days, 5 ppm produced 50 percent inactivation of photosynthesis within 4 days and in the case of land plants, the effects on growth of adding chromium to the soil depended on the amount of chromium naturally present in the soil.

The National Research Council (1974) provided more examples. Although chromium at 75 ppm in soil was not harmful to orange seedlings, the addition of Cr at 150 ppm was toxic. Chromic sulfate stimulated the growth of corn seedlings in culture solutions containing chromium at 0.5 ppm, but at 5 ppm and above it inhibited growth. The growth of tomatoes, oats, kale, and potatoes was reduced by chromium (as chromate) at 16 ppm. Chromium at 5 and 10 ppm in nutrient solutions produced iron chlorosis in oat plants, and at 15-50 ppm it was toxic. Chromium at 8 and 16 ppm produced iron chlorosis in sugar beets, and at 5 ppm (as chromate) it was toxic to tobacco and at 10 ppm toxic to corn. In some instances, toxicity has been associated with the chromium concentration in

plant tissues. For example, tobacco leaves grown on serpentine soil, which normally has a high chromium concentration (possibly several percent), may contain chromium at 14 ppm (dry weight) without toxic signs; but at 18-34 ppm, toxic effects were visible. Concentrations of 175 ppm (dry wt) in the roots were without harm; but at 375-410 ppm, toxic symptoms were present. In fruits, vegetables, and grain, no harmful evidence was found with concentrations from traces to about 14 ppm (dry tissues); but toxic symptoms appeared in corn when the leaves contained 4-8 ppm and in oats when the leaves contained 252 ppm.

Witter (1989) stated that chromium in sludge is present as Cr (III) in which form it is non-mobile, and in which it remains in the soil. Toxic effects of chromium on plants of chromium have only been noted when added as Cr (VI). Chromium is relatively non-zootoxic and no negative food chain effects can be expected because of the extremely low plant uptake of Cr. Field and pot experiments show no negative effects on plant growth and non-significant or minimal (less than 1%) Cr uptake when sludge containing chromium was applied at rates of 110 kg Cr/Ha.

Terrestrial Animals

The U.S. Department of Health and Human Services (1993b) noted harmful effects on the respiratory system and a lower capability to fight disease, if animals breath high levels of chromium.

Eisler (1986) reviewed the effects of Cr (VI) in dogs and chickens. Cr (VI) was deadly to dogs when exposed for 3 months to food with 100 ppm of Cr (VI). Tissue accumulations (especially in the brain) were significant in dogs exposed to drinking water concentrations of 11.2 ppm Cr. Male domestic chickens fed diets containing up to 100 ppm of Cr (VI) for 32 days showed no adverse effects in survival, growth, or food utilization efficiency. However, teratogenic effects were documented in chicken embryos

after eggs had been injected with Cr (VI). Chromium is an animal carcinogen. In the only animal study demonstrating a carcinogenic effect of an inhaled chromate, adenocarcinomas were reported in lungs of mice exposed throughout life to $CaCrO_4$ dust at 13 mg/m^3 for 35 hours weekly.

Aquatic Organisms

Towill et al. (1977) stated that chromium is considerably toxic to fish and many other aquatic organisms, especially in its hexavalent form. This toxicity varies depending on their sensitivity to the element. The lethal level for some invertebrates is 0.05 ppm, while tests on other organisms, including fish, indicate that several tens of ppm can be tolerated. Maximum permissible concentrations must be set based on the more sensitive organisms.

Eisler (1986) compared different factors that determine sensitivity to chromium (VI). Younger life stages are more sensitive than older organisms. The organisms most sensitive to Cr (IV) were freshwater crustaceans, rotifers and marine crustaceans.

Invertebrates

Invertebrate species are generally more sensitive to Cr (VI) than fish species. Eisler (1986) reported acute levels of Cr (VI) for six freshwater invertebrate species from five families. These ranged from 67 ug/l for a scud to 59,900 ug/l for a midge.

Shepherd and Jones (1971) and Eisler (1984) associated Cr (VI) with unfavorable effects in invertebrates of amply separated taxa: reduced survival and fecundity of the cladoceran Daphnia magna at a concentration of 10 ppb and exposure for 32 days; growth inhibition of the protozoan Chilomonas paramacium at 1,100-3,000 ppb during exposures

of 19-163 hours at temperatures of 10- 30 degree celsius; abnormal movement patterns of larvae of the midge Chironomus tentans at 100 ppb for 49 hours; and a temporary decrease in hemolymph glucose levels in the freshwater prawn Macrobrachium lamarrei surviving 1,840 ppb Cr (IV) for 96 hours. The acute toxicity of Cr (VI) in 20 saltwater vertebrate and invertebrate species ranges from 2,000 ug/l for polychaete annelids and a mysid shrimp, to 105,000 ug/l for the mud snail. Polychaetes and microcrustaceans are the most acutely sensitive taxa. Chronic toxicity was observed at Cr (VI) concentrations of 25 and 123 ug/l for polychaetes and amysid shrimp, respectively.

Shepherd and Jones (1971) found, in a 2-year study of oyster mortalities in water containing 0.01 ppm chromium, that toxicologic effects may be cumulative even at these low concentrations.

Fish

Anderson (1982) revealed that the growth and reproductive capacity of fish can be adversely affected at concentrations of Cr(VI) as low as 10 ppb.

Nriagu and Nieboer (1988) said that Cr (III) seems to be more poisonous to fish than Cr(VI). The mean 96-hour LC_{50} for Cr(III), pooling data for all species and water conditions, was 22.0 mg/L, significantly lower than the 100.7 mg/l for Cr (VI). The chronic and/or sublethal effects of Cr(VI) and Cr(III) to fish include histophatological damage, altered blood parameters such as hematocrit, serum protein levels, and blood glucose levels, decreased enzyme activity, and impaired respiratory and locomotory activities. Generally, reproduction and larval survival seem to be the most sensitive indicators of chronic toxicity. As well, the coldwater salmonids appear to be the most sensitive family of fish to chromium toxicity; while the warmwater minnows, carps, and livebearers appear to be the most tolerant families of fish to chromium toxicity, both acute and chronic.

Shepherd and Jones (1971) referred to a toxic concentration of about 20 ppm Cr (VI) for minnows and rainbow trout. The growth of chinook salmon was reduced at a measured concentration of 16 ppb of Cr (VI). Eisler (1986) stated that Cr (VI) concentrations of 16 to 21 ppb in the medium resulted in reduced growth of rainbow trout during exposure of 14 to 16 weeks and altered plasma cortisol metabolism after 7 days.

Anderson (1982) reported the result of long-term tests with brook trout and rainbow trout. Both presented chronic effects at 265 ug /l, which is much lower than the 1,990 ug/l for the fathead minnow. In chronic tests with brook trout, rainbow trout, and fathead minnows, a temporary adverse affect on growth occurred at low concentrations.

Eisler (1986) stated that Cr (VI) concentrations of 16 to 21 ppb in the medium resulted in reduced growth of chinook fingerlings during exposure of 14 to 16 weeks.

Anderson (1982) indicated a tolerance level of 45 ppm for blue gills exposed 20 days in hard water, and of 200 ppm (using $K_2Cr_2O_7$) for mummichogs exposed in seawater for 1 week.

The toxicity of chromium to Channa punctatus was described by Sastry and Tyagi (1981). Exposure of Channa punctatus for 30 days to a sublethal concentration of chromium produced some marked changes in blood and tissues. The blood glucose and lactic acid levels were elevated. Liver glycogen was depleted.

Regulations

According to the Lead Chromate Committee (1987), wastes are judged hazardous by the Resource Conservation and Recovery Act (RCRA) only if they do not pass the tests for ignitability, corrosivity, reactivity, or Toxic Characteristic Leaching Procedure (TCLP formerly EP) toxicity outlined in the regulation. For this purpose, lead chromate pigments were tested. Results indicated that, with few exceptions, the toxic characteristic leaching toxicity exceeds the 5 mg/l maximum for lead, and encapsulated lead chromates

as currently produced are closer to the 5 mg/l limit but still above it. However, all of the colored lead chromate pigments tested were well below the 5 mg/l maximum for chromium. In conclusion, any offgrade or contaminated chrome yellows or molybdate oranges must be treated as a hazardous waste. Waste sludge containing pigment would not necessarily be hazardous based on lead chromate content unless leaching tests indicated 5 mg/l of lead or chromium could be extracted. Handling properties of lead chromate pigments are improved by granulation, resin encasement, surface treatment with materials such as teflon fibers, and electrostatic charge neutralization.

Finley et al. (1992) point out that the EPA is developing an inhalation reference concentration (RfC) for Cr (IV) and Cr(III). Inhalation reference concentration (RfC) is an estimate of continuous exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Cr (VI) is considered by the EPA to be a known inhalation carcinogen. For that reason the EPA in 1991 proposed to promulgate an inhalation RfC of 0.002 ug/m^3 for both Cr(VI) and Cr (III). The proposed RfCs are several orders of magnitude less than threshold limit values and other occupational exposure limits.

CHAPTER 6

HEALTH EFFECTS ASSOCIATED WITH HEXAVALENT CHROMIUM

Introduction

According to the Environmental Criteria and Assessment Office (1984), depending on the oxidation state and type of chromium compounds, we can determine the risks and benefits to human health. In the natural environment, chromium appears in two oxidation states, hexavalent [Cr(VI)] and trivalent [Cr(III)]. Chromium (VI) is toxic to animals and plants, whereas Cr (III) is considered to be less toxic (Nriagu and Nieboer, 1988).

Cr (VI) has a high penetration power. According to OSHA (1979), hexavalent chromium can be absorbed through the skin, lungs, or intestinal tract by sorption through the membranes and tissues. Then, Cr (VI) is able to attach itself to protein, nucleic acids, and hemoglobin. The new complexes which are formed may interfere with the regulation of cellular activity, and may exert a carcinogenic action on the cells (Sullivan, 1969).

Towill (1977) recommended that exposure to Cr(VI) compounds be controlled, especially in the occupational environment. Among the three standards recommended by NIOSH for the control of worker exposure to chromium, there are two standards that pertain to Cr (VI). One standard pertains to occupations and workplace where there is

exposure to Cr (VI) material associated with an increased incidence of lung cancer. This standard allows concentration of Cr (VI) in the airborne workplace not greater than 25 ug/m^3 (determined as a time-weighted average (TWA) exposure for up to a 10 hr-workday, 40 hr workweek) and not greater than 50 ug Cr (VI)/m^3 of breathing zone air (determined by any 15-min sample).

Two years ago OSHA issued a new standard which reduced the permissible exposure limit even further. The level of chromic acid and chromic (VI) compounds in the workplace air should not be higher than 100 ug Cr/m3 for any period of time (U.S. Department of Health and Human Services, 1993b).

Cancer

Chromium compounds are among the few established causes of work or environment-related cancer that have been known for more than 100 years. Levy and Venitt (1986) stated that no one has determined which kinds of hexavalent chromic salts (chromates or dichromates) are responsible for the increased risk of lung cancer. As stated before, Cr (VI) induces cancer because of its wide solubility. Therefore, chromates with aqueous solubility are more active in inducing tumors. The potential carcinogenicity of Cr (VI) compounds will depend to a large extent on chemical composition and those physical properties which determine the absorption, distribution and retention of a sufficient concentration of chromate ions at the target location.

Only certain Cr(VI) compounds are chemical carcinogens. Anderson (1982) identified some potential chromium carcinogens as chromates of calcium, lead, and zinc. The U.S. Department of Health and Human Services (1993b) mentioned calcium chromate, chromium trioxide, lead chromate, sodium dichromate, strontium chromate, and zinc chromate as known carcinogens.

The Lead Chromate Committee (1987) discussed an animal study conducted at the University of Aston, England in which calcium chromate and strontium chromate produced a high incidence of tumors, while zinc chromate, which is somewhat less soluble, produced a lower number of bronchial carcinomas. Among the seven lead chromate-type pigments tested, Lead chromate, Primrose chrome Yellow, LD Chromate Yellow, and Medium Chrome Yellow, each produced one case of carcinoma out of a population of 100 rats. The remaining pigments (Molybdate Chrome Orange, Light chrome Yellow, and Silica-encapsulated Medium Chrome Yellow) did not produce any cases of carcinoma. In addition, no tumors were found in the barium chromate group. Neither the highly soluble (chromic acid or sodium dichromate), nor the insoluble compounds (barium chromate) can be considered to be carcinogenic. Only chromates of medium solubility (strontium, calcium and zinc chromate) are carcinogenic.

In humans, chromium compounds produce cancers only in the respiratory system and only via respiration. Sullivan (1969) defined the average duration of exposure to chromium before death from respiratory tract cancer as 18 years.

The U.S. Department of Health and Human Services (1993) believes that chromium (VI) is primarily responsible for the increased lung cancer rates observed in workers who were exposed to high levels of chromium in workroom air.

According to NIOSH (1975), Gross and Kolsh in 1943 reported the first study of cancer in the chromate pigments industry in Germany. Three firms which manufactured lead chromate (chrome yellow) and zinc chromate (zinc yellow) pigments participated in this study . 8 deaths from lung cancer were reported. Five of these deaths occurred among workers producing zinc chromate; the other three deaths occurred among workers in plants producing chromates of both zinc and lead. Four of the deceased were only 33-37 years of age . Seven of the deceased had worked in the industry 5-17 years. The authors suggested that zinc chromate was the prime causative agent and should be considered to be a potent carcinogen.

Langard (1993) mentioned a reported issued by Letterer et al. which reported two cases of lung cancer in workers 36 years old, after 7 and 20 years of work in a chromium pigment plant. One had been exposed to zinc chromate only.

NIOSH (1975) and Langard (1993) referred to a very famous report published by Langard and Norseth in 1975. This study examined a Norwegian zinc chromate company. 133 male workers were observed from 1953 to 1972. Zinc chromate was the main exposure, but a small number of the workers were exposed to lead chromate between 1948 and 1952. Of these a cohort of 24 were derived comprising those who were employed for more than 3 years before December, 1972. Three cases of lung cancer occured in this group between 1951 and 1972.

The Lead Chromate Committee of the American Dry Color Manufacturers' Association (1987) in 1974 sponsored a study of three typical United States chromate pigment production plants. The final report showed a level of lung cancer slightly higher than expected, and a lower level than was seen in the Norwegian population. The authors concluded that lead chromate is a carcinogen. A five year follow-up study through the end of 1979 found no evidence to support an association between lead chromate and lung cancer, but did find evidence to tie increased lung cancer to exposure to zinc chromate.

According to Nriagu and Nieboer (1988), three chromate pigment plants in Great Britain were studied in 1984. An elevated risk of lung cancer was found only in the plants that produced both zinc and lead chromate, and was absent in workers who were employed only in the manufacture of lead chromate. Langard (1993) also referred to the same study stating that a nonsignificant excess of lung cancer deaths was seen in a small subgroup of workers exposed to lead chromate only, as well as in a small group of lead chromate workers who had previously suffered from lead poisoning. These findings remained consistent with the hypothesis that lead chromate might be noncarcinogenic. Zinc chromate is a more potent human carcinogen by inhalation than are other Cr (VI) compounds.

Dermal Diseases

Stern et al. (1993) referred to Cr (VI) as a potent source of allergic contact dermatitis. Chromium is the second most common skin allergen, the most common sensitizer, and the most important cause of occupational dermatitis. An EPA inhalation reference concentration (RfC) based on noncancer toxicity is pending.

The National Academy of Sciences (1974) classified chromium skin exposure as a corrosive reaction, including ulcers and stigmata (scars), and sensitization reaction, including eczematous contact dermatitis (allergic reaction).

Anderson (1982) and Stern et al. (1993) stated that short-term exposure can cause allergic contact dermatitis (skin ulceration or eczema). Allergic dermatoses are associated with the handling of chromium-containing materials. These ulcerations result from contact with chromium acid, sodium or potassium chromate or dichromate, and ammonium dichromate at levels high enough to provoke an immediate response in normal healthy workers. The dermatoses can continue for long periods of time. This dermatitis is described as localized swelling and inflammation, wrythema , papules and vesicles, followed by dryness, scaling, and fissuring, on breaks in the skin or where abrasion of the workers' bodies in contact with chromium compounds exists, but exposure of the skin to chromic vapors, fumes and/or dusts may contribute to these effects.

Sullivan (1969) described the location of these ulcerous dermatoses in the skin, usually on the hands and forearms, and occasionally on the feet, ankles, face, and back. Other specific sites like roots of the fingernails, knuckles, eyelids, edges of the nostrils and throat were mentioned by the National Academy of Sciences (1974). The U.S. Department of Health and Human Services (1993b) pointed out that corneal vesication result from a crystal of potassium dichromate or a drop of potassium dichromate in the

eyes. Sullivan (1969) stated that these ulcers in some cases have perforated to the bone. Over 50% of a group of chromate workers showed signs of either active or healed chrome ulcers. On a few workers who are apparently hypersensitive to chromates, the dermatitis is an immune system response to an allergen absorbed into the skin and appears as an allergy. Sensitization may develop, resulting in typical asthmatic attacks, which recur on later exposure even when exposure is to much lower concentrations.

Nriagu and Nieboer (1988) alluded to the report of Parkhurst in 1925 where a risk of chromate dermatitis was identified in the printing industry. Pirila and Kilpio in 1954 reported chromate sensitivity in 27% of 149 cases of dermatitis among printing workers. Of 76 cases of dermatitis reported among lithographers, 17 patch-tested positively to potassium dichromate. Spruit and Malton in 1975 demonstrated high chromium levels in the materials handled by three men working in an offset printing factory who developed dermatitis and chromate sensitivity. The use of potassium dichromate in the printing industry has declined following suitable substitution by other agents.

OSHA (1979) recommended that during production operations, where splashes of liquid pigment can occur or dry powders can be inhaled, workers must be protected. Drying, grinding, blending, and packaging are operations where chromate exposure is likely to occur because chrome dust is very fine, and once it gets into the workplace air it remains a long time.

Respiratory Effects

Bencko (1985) reported a concentration of chromium in lungs of individuals living in industrialized areas of 70 ug, the liver 270 ug and the kidneys 90 ug per 1 kg fresh tissue. Considerably lower levels were found in persons residing in non-industrialized areas of the country.

According to the U.S. Department of Health and Human Services (1993b) and Anderson (1982), long-term breathing of concentrations of chromium (VI) greater than 2 ug/m^3 can induce inflammation of the thin mucous membrane layer covering the nasal septum producing irritation to the nose, such as runny nose, sneezing, itching, and nosebleeds. Longer exposure can cause ulcers and perforation or holes in the nasal septum. Kartz and Salem (1993) and Anderson (1982) pointed out that this perforation can be present without any awareness by the workers. If the exposure continues, the perforations get deeper and become very painful.

The National Academy of Sciences (1974) remarked about additional effects due to exposure to high concentrations of chromium: cough, headache, dyspnea, substernal pain, and bronchospasm. The bronchospasm is likely to be due to chemical irritation of the air passages, and not to "bronchitis", although it is commonly so diagnosed. A long-term inhalation of chromate dust causes chronic irritation of the respiratory tract and results in such symptoms as congestion and hyperemia, chronic catarrh, congestion of the larynx, polyps of the upper respiratory tract, chronic inflammation of the lungs, emphysema, tracheitis, chronic bronchitis, chronic pharyngitis, and bronchopneumonia. Some German doctors affirmed that a typical pneumonoconiosis resulted from exposure to some chromates. Two incidents of acute pulmonary complications involving the deeper pulmonary structures after inhalation of massive amounts of chromic acid mist have been delineated. The estimated chromic acid concentration in the mist was 20-30 mg/m^3. The manifestations included cough, chest pain, some dyspnea, pleural effusion, and loss of weight. In another study, atrophic rhinitis was reported in 5-10% of a group of workmen exposed to the mist of a 5% chromic acid solution. Hyperemia, swelling, congestion, and nasal catarrh occurred in some of these persons.

Mutation

According to Bianchi (1980), evidence has accumulated to show that compounds of chromium possess the ability to cause transformation and mutation. Cr (VI) increases the frequency of sister chromatid exchanges (SCEs) and increases the frequency of chromosome aberrations, in mammalian cells, only at extremely high concentrations. Gennart (1993) reported an increased frequency of chromosomal aberrations and/or sister-chromatid exchanges (SCE) in lymphocytes of workers exposed to Chromium (VI) compounds.

CHAPTER 7

ENVIRONMENTAL EFFECTS ASSOCIATED WITH CADMIUM

Introduction

According to Dorn (1979) and Dobson (1992), cadmium is used mainly in the production of electroplating for protective plating on steel, pigments and chemicals in plastics and glass, plastic stabilizers for polyvinyl chloride, alloys and solders, electrode material in nickel-cadmium batteries, semi-conductors and photocells, and pesticides.

Stansley et al. (1991) included smelter emissions, wastewater discharges, landfill application of sewage sludge fertilizers, and fossil fuel combustion as anthropogenic sources of cadmium. If these sources of emission continue, cadmium in the environment will reach a dangerous level. Osuna and Edds (1982) state that the concentration of this metal in humans has already increased, with the kidney the main organ affected.

Plastics

Cadmium is used as a pigment and a stabilizer in the polymer industry. According to Dobson (1992), these materials held 22% and 12%, respectively, of the consumption of cadmium around the world in 1985. Cadmium pigments' share of the market remains constant. In contrast, the use in stabilizers for plastics is decreasing, due to manufacturers replacing cadmium by other cheaper compounds. For instance, liquid barium-zinc compounds are used in PVC instead of cadmium. This compound costs less and performs better.

Monks (1990) stated that ABS is the plastic that most commonly use cadmium as a pigment. ABS accounts for 35% (200 tons) of the total cadmium use in plastics, followed by HDPE (25%), polypropylene (15%), polystyrene (10%) and LLDPE (10%). 45% (90 tons) of cadmium used in ABS finds its way into municipal landfills and garbage incinerators.

Plastics are an important source of cadmium in solid waste. Monks (1990) ranked cadmium in plastics as the second largest contributor of this metal to the total waste in 1986. The amount discharged in 1986 in the U.S. was 564 tons. This represents 28% of the total cadmium released.

Monks (1990) discussed the increasing concern of additives makers and users about pigments and PVC heat stabilizers based on cadmium. This concern is a result of the surge of regulations proposed by CONEG, OSHA, and the Clean Air Act for the metal's use in plastics.

These regulations have emerged due to cadmium contamination of air and groundwater resulting from the incineration of cadmium-containing plastics. Cadmium containing pigment makers are planning to phase out their use because they are pushed to

replace cadmium by the pigment users. However, the replacements for cadmium pigments do not provide the same palette diversity as pigments made with cadmium.

Plasticizers

The British Department of the Environment (1980) described cadmium plasticizers as inhibitors of degradation by heat or light in PVC. This degradation causes darkening, toughening and embrittlement. Plasticizers that contain cadmium in amounts of 1 to 20% are used. These plasticizers are used in small quantities in the polymer (1 to 3%).

Pigments

The Department of the Environment (1980) reported the total amount of cadmium used in Great Britain as a pigment as 550 tonnes in 1978. A great percent (70%) of cadmium pigment was employed in the coloring of plastics such as polyvinyl chloride (PVC), polystyrene (PS) and polypropylene (PP). The rest of the cadmium pigment was used in the coloring of rubber and, to a lesser extent, industrial and car paints, ceramic decorative materials, iridescent and colored glasses, enamel, and printing inks.

Cadmium pigments can provide us with a whole diversity of colors from lemon-yellow, through orange, to deep maroon.. In addition, cadmium pigments deliver colors such as khakis, browns and even greys (Department of the Environment , 1980).

The kind of cadmium compounds used as pigments were described by the Commission of the European Communities (1981) and the Department of the Environment (1980). Cadmium sulfide and selenide are used especially for printing ink, rubber, plastics, coated fabrics and leather. Properties of these two compounds include brightness, opacity, heat resistance up to 1200 Celsius, light stability, good covering power, insolubility in organic solvents and insolubility in water. However, these

compounds are expensive, are darkened by atmospheric sulfur, and are toxic when are used in toys or food containers. Cadmium oxide or carbonate are used for coloring enamels, glazes and glass.

According to Monks (1990), the closest alternatives for cadmium pigment substitutions are organic compounds and inorganic compounds such as iron oxide and a host of nickel titanates blended with antimony. However, it is difficult to get the same shades of color as cadmium provides. Iron oxide mutes to a reddish-brown color and nickel titanates with antimony have poor color dispersion. In cheap plastics and paints, cadmium pigments have already been replaced. Due to the heat resistance property of cadmium pigment, it is difficult to find replacements with the same color shades as cadmium when the pigment is exposed to high temperatures.

Polyethylenes, PVC and polystyrene wraps, containers and household objects include in their manufacturing pigments derived from cadmium compounds. Preda et al. (1983) studied the release of this metal from the plastic. They observed pathological changes in the tissues of white rats exposed to extracts of such plastics.

Water

Concentrations of cadmium in water are low and constant. Such concentration is below the drinking water standard (0.01 mg/l). The World Health Organization recommended that the concentration in drinking water needs to be kept below 0.005 mg Cd/l (Dorn, 1979).

Soluble cadmium is persistent and highly toxic. Taylor (1983) found lethal effects of soluble cadmium at levels greater than 0.002 mg/l. in fresh water and greater than 0.095 mg/l in marine water. The EPA established in 1980 an allowable concentration of

cadmium in water equivalent to 0.0063 mg/l in fresh water and 0.059 mg/l in marine waters.

Webb (1975) mentioned smelting and refining of zinc and lead ores, and the dumping of sewage sludge and of waste plastics as the major industrial contaminants. Major packaging-related industries that dump great amounts of sewage sludge and waste are pulp and paper mills, rubber processing, and paint and ink plants. The recovery of cadmium from industrial wastes is uneconomic due to low levels of cadmium.

Air

Nriagu (1990) estimated the emissions around the world of cadmium as 7570 tonnes. 90% of this number is accounted for by anthropogenic sources. Smelting of base metal ores is the main source of cadmium, with a amount of 5,299 tonnes/year, followed by refuse incineration (about 750 tonnes/year), coal combustion (about 530 tonnes/year), cement production (about 270 tonnes/year), and fertilizer production (about 170 tonnes/year).

Atmospheric concentrations of cadmium in urban locations are below 10 ng/m^3. Cadmium released anthropogenically is transmitted to the food chain. Incineration of wastes containing cadmium, such as plastics, increases the level of cadmium in the air. Exposure to this level is predicted to increase human cancer risk in urban or industrial areas (EPA, 1985).

Soil

Nriagu (1990) listed the main cadmium pollutant sources in soil as atmospheric fallout, urban refuse disposal, dumping of fly ash, use of sewage sludge as a source of nutrients or organic amendments for improving soil physical properties, disposal of agricultural and animal wastes, and the application of fertilizers.

The Commission of the European Communities (1981) estimated a range of concentration of cadmium in the soil of 0.01- 2.5 mg/kg. Commonly the level is less than 1mg/kg. According to Hattori (1989), when these levels are exceeded, the effects are decrease of vegetation due to plant toxicity.

Sewage Sludge and Land Disposal

Soils improved by sewage sludge contain a high concentration of cadmium. The Commission of the European Communities (1981) described the treatment of sewage sludge before it is included in the soil. 30% of the cadmium in the raw sewage is separated by sedimentation. Then, 60 to 70% of the cadmium is separated by activated sludge treatment. In total, 90% of the cadmium in raw sludge is removed. However, the remaining 10% is added to the soil. This amount will increase the level of cadmium in agricultural soil. Dorn (1979) mentioned a fertilizer called "Milorganite" which is a result of municipal sewage converted to organic fertilizer. The use of this fertilizer may increase the content of cadmium in soil.

Jones et al. (1987) pointed out that the increase in this metal may have an adverse effect on human health. Food based on plants is one of the major source of dietary cadmium. The World Health Organization's (WHO) allowable intake of cadmium by an adult is 51-71 ug/day, except for beef, canned fish and canned tomato sauce, which should

be limited to 100 ug/kg, and kidney and canned kidneys, which would be limited to 500 ug/kg. The present average daily intake of cadmium in Europe is 20-40 ug/day. This amount will increase in the long term, reaching WHO's allowable limits of 51-71 ug/day. Most of the increase in dietary cadmium results from grain and cereal intake.

Wolf and Baker (1979) emphasized the limitation of cadmium in soil to no more than 3 pounds/ acre as what they considered safe. Sewage containing more than 50 ppm is not beneficial to plants due to cadmium accumulation in the soil.

Considerable leaching of cadmium from soil to ground water can occur only in extreme contamination, low pH or both. This contaminated ground water can contaminate aquatic systems. Cadmium concentration in soil solutions may be 0.1-1 ug/l (Nriagu and Sprague, 1987).

Animal Toxicity

According to Webb (1975), concentrations of cadmium present in the environment may be a great hazard for sensitive animals. Such low levels can produce adverse effects in domestic animals. Stansley et al. (1991) reported a case of accumulation of high amounts of cadmium in the liver and kidneys of wild moose and deer.

Birds

Eisler (1985) noted that aquatic birds are highly tolerant to cadmium. He mentioned adult drake mallards have survived a dose of 200 ppm of cadmium given for 90 days. Terrestrial birds are more tolerant than aquatic birds. Effects on terrestrial bird toxicity include growth retardation, anemia, and testicular damage.

Aquatic Organisms

According to Taylor (1983), international covenants (Oslo, London, Paris, Rhine, and Barcelona Conventions and the EEC Directive on the Discharge of Dangerous Substances) include cadmium and its compounds on their black lists. The reason behind this decision was to prevent aquatic pollution. Cadmium presents all the selective factors: persistence, toxicity and bioaccumulation.

Eisler (1985) found a decrease in growth, respiratory disruption, molt inhibition, shortened life span of next generation crustaceans, altered enzyme levels, and abnormal muscular contractions in marine organisms exposed to cadmium in a range from 0.5 to 10 ppm. Sea water organisms can resist higher levels of cadmium than freshwater organisms. In freshwater biota, high mortality rates, reduced growth and inhibited reproduction were present at concentrations exceeding 10 ppb. Mortality increased as time of exposure increased, water hardness decreased, and the organism's age decreased. Concentration of cadmium in marine organisms (particularly zooplankton, mollusks and other filter-feeders) was 10^{3} or 10^{4}. Shellfish also accumulate Cd; the brown meat of the edible crab, for example, consistently contains around 5-15 mg/kg of Cd.

Fish

The Commission of the European Communities (1981) classified fishes according to their sensitivity. Salmonid fish and some species of invertebrates, such as Daphnia magna and Gammarus fossarum, were the most sensitive. In fish species that are particularly sensitive to cadmium, it accumulates mainly in the liver, gills, and kidney. Webb (1975) pointed out that cadmium accumulates in gills as water passes through, and in the body when water is ingested.

Lowe-Jinde and Niimi (1984) stated cadmium toxicity signs in fish are growth reduction, testicular injury, impaired gill function, hematological changes, and disturbances in osmotic-ionic balance and carbohydrate metabolism. Lower concentrations of Cd than those that are toxic to adult fish affect reproduction and are toxic for the larvae. According to Dobson (1992), toxicity decreases as salinity increases, temperature decreases, and oxygen content increases. The Commission of the European Communities (1981) noted that accumulation of cadmium in muscle fish tissue varies to levels 1000 times higher than in the seawater (less than 0.1 ug of cadmium in a liter of seawater).

Metallothionin (MT) is a protein that regulates the detoxification of heavy metals. Gagne et al. (1989) mentioned that cadmium reduces the synthesis of this compound, decreasing the detoxification ability of fish.

Studies of 30 to 60 days duration with three comparatively sensitive species of freshwater fishes demonstrated that cadmium, in a range of concentration from 1 to 3 ppb in water of low alkalinity caused reductions in growth, survival, and fecundity of brook trout, the most sensitive species tested. Under conditions of increasing alkalinity, the maximum allowable cadmium concentration range for brook trout is 7 to 12 ppb (Eisler 1985).

Invertebrates

Nriagu and Sprague (1987) described cadmium toxicity in three groups of freshwater invertebrates. The most sensitive group was crustaceans, which manifested effects at an average of 62 ug/l. Within this group, the cladocerans were the most affected (19 ug/l), copepods the most tolerant (250 ug/l), and the amphibious intermediate (62 ug/l Cd). Insect larvae were the most tolerant, exhibiting a broad range of tolerance from 840 to 233,000 ug/l. The gastropod mollusks were intermediate, ranging between 300 and

8,400 ug/l. According to Dobson (1992), toxicity increases as temperature and salinity increase.

Plant Toxicity

The content of cadmium in plants fluctuates depending on differing growing conditions and on natural variations in other factors (Commission of the European Communities, 1981). Eisler (1985) mentioned cadmium toxicity in freshwater biota, in general, at a range of cadmium concentration from 0.47 to 5.0 ppb. The effects of this exposure were decreases in standing crop, decreases in growth, inhibition of reproduction, immobilization, and population alterations.

Dobson (1992) and EPA (1980) pointed out growth reduction as the main toxic effect in plants. Cadmium reduced the length of shoots and roots, depending on the concentration. Cadmium in soil is bound; therefore, this cadmium is not available to the plant. However, cadmium applied to the soil in nutrient solutions is more available to plant absorption. Cadmium solutions are applied to the soil frequently as fertilizer. Due to the accumulation property of cadmium, cadmium soil content increases. Effects on plant growth were seen only when cadmium concentration reached a high level. Webb (1975) mentioned roots as the part of the plant that contains the greatest concentration of cadmium.

Cadmium, among all the toxic metals released in the environment, is the only one that can be stored in the human food chain in considerable quantities. Cadmium accumulates mainly in certain kinds of plants such as food crops, root crops, leafy vegetables and tobacco plants (Nriagu, 1990).

Hallenbeck (1979) mentioned the long biological half-life of cadmium, which is about 16 to 33 years, its mobility, and efficiency of deposition as the main causes of cadmium accumulation. The accumulation of cadmium in human bodies reaches a peak at about age 50 and then remains constant.

CHAPTER 8

HEALTH EFFECTS ASSOCIATED WITH CADMIUM

Introduction

According to Thun et al. (1989), approximately 100,000 employees in the United States are exposed occupationally to cadmium. Waalkes et al. (1992) mentioned that cadmium is a very widely known potent occupational metallic toxicant for an ample range of tissues and organ systems, whose toxicity depends on the concentration in the organ, although this varies widely with the tissue in question. Zettergren et al. (1991) and Mueller (1993) added that cadmium's effects on different organ systems include damage to the tissues of the reproductive system; induction of testicular interstitial cell cancer; structural and functional damage to liver, kidney, lung and nervous system; and disturbance of calcium metabolism, which can lead to conditions such as osteoporosis and osteomalacia.

Mueller (1993) and Shaikh et al. (1987) pointed out that cadmium stays in the body for a long period, having a half-life of 10 to 30 years. This metal is stored in the liver and kidneys bound to metallothionein (MT). For this reason, if the exposure is

chronic, the accumulation of cadmium increases the body burden, causing nephrotoxicity years later even when exposure has ceased.

Acute Effects

Hammens et al. (1978) described the symptoms of acute inhalation of cadmium as a metallic taste in the mouth; headache; shortness of breath, chest pain, cough with foamy or bloody sputum, abnormal pulmonary rales, and physical signs which mimic the flu; weakness and leg pains; pulmonary edema, which may lead to death or may gradually improve over several days; pneumonic consolidation; and later liver damage.

Murray et al. (1981) and Waalkes et al. (1992) mentioned that in a case of brief exposure to a high dose of cadmium, the organs affected are the lungs in the case of inhalation, and the gastrointestinal tract in the case of oral exposure. The lung is the initial site of damage in high level inhalation exposure. The EPA (1989) observed that lung irritation results from inhalation of air with a concentration of 1 mg/m^3 of cadmium.

Barnhart and Rosenstock (1984) mentioned that acute inhalation of cadmium fumes may cause metal fume fever and chemical pneumonitis in workers only if the workplace is poorly ventilated. The symptoms of metal fume fever are fever, general malaise, and chest tightness. Chemical pneumonitis can result in death if ventilation is not improved.

Chronic Effects

Barnhart and Rosenstock (1984) stated that long-term exposure to low levels of inhaled cadmium may cause emphysema, pulmonary fibrosis, renal insufficiency, and maybe cancer. According to Waalkes et al. (1992) and Mueller (1993), the kidneys and lungs are the critical organs affected. The manifestations of lung damage are pulmonary emphysema and bronchitis. De Silva and Donnan (1981) and EPA (1989) mentioned that long term exposure at 0.02 mg/m ^{3} of cadmium in the air, posed a relatively small risk of lung or kidney damage but at 0.1 mg/m ^{3} of cadmium in the air the risk of emphysema formation and kidney damage (proteinuria) increased. The signs of damage may not appear until many years after the last exposure to cadmium.

Peereboom and Copius (1981) reported early effects of chronic exposure to cadmium as emphysema, renal lesions and proteinuria and also anemia, anosmia and yellow coloring of the teeth, at cadmium concentrations of 0.5-5 mg/m ^{3} in air during 10 years or longer for 8 h/day, which were found in several factories.

Exposure Limits

Peereboom and Copius (1981) stated exposure to 40-48 mg/m^3/hr of cadmium in air causes adverse effects in humans. According to their data the lethal dose of cadmium for man is 5 mg/m ^{3} for 8 hours exposure.

The American Conference of Governmental Industrial Hygienists set a Threshold Limit Value (TLV) at 200 ug/m ^{3} for Cd dusts and 100 ug/m^3 for Cd fume (Peereboom and Copius, 1981; Lauweys et al., 1974; EPA, 1993). Because breathing

cadmium may cause lung cancer, the National Institute for Occupational Safety and Health (NIOSH) wants workers to breathe as little cadmium as possible.

OSHA (1992) established three limits of exposure in the workplace. The permissible exposure limit (PEL) is a Threshold Limit Value of 5 ug/m ^{3} of all cadmium compounds (dust or fumes), which must not be exceeded in the air during any 8-hour work shift of a 40-hour work week. The action level is half of the PEL, 2.5 ug Cd/m ^{3}, and the separate engineering control air limit (SECAL) is 15 or 50 ug/m ^{3}. The higher value is used where it is not possible to achieve the PEL limit through engineering and work practices alone.

Exposure Effects

Death

The U.S. Department of Health and Human Services (1993a) reported inhalation exposure to cadmium as a possible cause of death in humans. Pulmonary edema and chemical pneumonitis resulting from exposure to cadmium can lead to death from respiratory failure. The lowest measured concentration of cadmium in the lungs of men dying from cadmium inhalation is 1.5 ug/g wet weight. The exposure to 1-5 mg/m ^{3} for 8 hours could cause some deaths among exposed humans.

Musculoskeletal Effects

The U.S. Department of Health and Human Services (1993a) reported the possibility of developing calcium deficiency, osteoporosis or osteomalacia in workers after long-term occupational exposure to high levels of cadmium. According to

Fleisher et al. (1975), osteoporotic disease ("Itai-Itai") developed among Japanese who ingested cadmium from contaminated drinking water.

Immunological Effects

According to the U.S. Department of Health and Human Services (1993), cadmium inhalation causes a decrease in the generation of reactive oxygen species by leukocytes compared to unexposed controls.

Developmental Effects

The U.S. Department of Health and Human Services (1993) reported a decrease in birth weight of Russian babies whose mothers were occupationally exposed to cadmium at concentrations ranging from 0.02 to 35 mg/m^3.

Genotoxic Effects

Tang et al. (1990) reported studies that proved the existence of chromosomal aberrations in peripheral lymphocytes, resulting from cadmium exposure.

Reproductive Effects

Mason (1990) discussed a study which observed high tissue cadmium levels and some histological changes in testicular autopsy samples from men who had suffered severe cadmium fume poisoning.

Respiratory Effects

The U.S. Department of Health and Human Services (1993a) and Waalkes et al. (1992) reported typical symptoms presented due to inhalation of long- term high levels of Cadmium oxide fumes or dust exposure as severe tracheobronchitis, pneumonitis, and pulmonary edema. These can lead to death if the exposure continues, and those who survive may have impaired lung function several years later.

According to Waalkes et al. (1992) and the U.S. Department of Health and Human Services (1993a), low-level long-term exposure to cadmium fumes or dust produces chronic bronchitis, progressive fibrosis of the lower airways, alveolar damage resulting in dyspnea and emphysema, chronic rhinitis and impairment or loss of the sense of smell due to chronic irritation or necrosis of the nasal membranes. Exposure duration and level will define the severity of the disease.

To evaluate respiratory effects due to cadmium exposure, Piscator (1981) considered concentration in the workplace, particle size and type of compound. Small particles such as cadmium oxide deposit mainly in the lower part of the respiratory tract and the alveoli and are relatively easily absorbed. Large particles, e.g. cadmium sulfide, deposit in the upper respiratory tract and have a relatively low solubility and absorption.

Cardiovascular Effects

Spieker et al. (1987) defined cadmium as a substance that significantly alters the vasopresor-induced reactivity and the stress strain characteristics of the blood vessel wall. This suggests that long-term exposure to cadmium might be associated with hypertension. Bhattacharyya and Chaudhuri (1988) studied the role of cadmium in the genesis of hypertension. They found that 34.1% of hypertension patients had

increased levels of cadmium in blood, urine or kidney tissues, causing cardiomegaly and neurological disturbances. They reported more cases of positive cadmium in male patients and persons residing in urban areas.

Renal Effects

Waalkes et al. (1992) and Piscator (1981) classified the kidney as the main organ affected by chronic exposure to cadmium. Piscator (1981) and Shaikh et al. (1987) estimated that a long-term exposure at the critical concentration of 200 ug of cadmium / kg of wet weight in the human kidney might cause damage in renal tubes.

According to Waalkes et al. (1992), Peerebom and Copius (1981), Murray et al. (1981) and Thun et al. (1989), tubular disfunction is shown as proximal tubule necrosis and chronic nephritis. Nephropathy causes an acquired Fanconi syndrome, an increase in low molecular weight proteinuria, aminoaciduria, glucosuria, azotemia (secondary to chronic interstitial scaring), and changes in calcium metabolism (hypercalciuria). This altered calcium metabolism results in an increase in calcium excretion and the formation of renal calculi. Peereboom and Copius (1981) mentioned an elevated level of renal stones among industrial cadmium workers. Waalkes et al. (1992) reported women suffering with Itai-Itai disease who developed severe osteomalacia and osteoporosis.

De Silva and Donnan (1981) described a company that manufactures cadmium selenosulphide and cadmium sulphide pigments in England. They studied only the cadmium in the color production section, in which exposure was high in many locations. Cadmium carbonate and cadmium sulphide were produced by precipitation, then filtered and dried. After drying, the carbonate and sulphide were crushed, milled, blended with selenium and sulphur, and calcined to produce red and yellow pigments, respectively. The drying stage is the one which produced the highest amount of dust.

The cadmium concentration was ten times higher than 0.05 mg/m^3 which is the hygiene standard recommended by the British Occupational Hygiene Society. They observed signs of renal damage in the 6 workers who had worked in the production plant for 7 years or more. In addition, two of these men exhibited exertional dyspnea, and respiratory obstruction with mild symptoms.

Cancer

The U.S. Department of Health and Human Services (1993) and Waalkes (1992) described experimental results that relate cadmium exposure to risk of lung cancer as conflicting. Confounding factors such as exposure to other metals and smoking may explain observed increases in cancer rates. The data did not show enough evidence to confirm that an increased risk of lung cancer in humans followed prolonged inhalation exposure to cadmium.

According to Kjellstrom et al. (1979), four prostatic cancers were observed among 248 workers with a minimum of 1 year's exposure to cadmium oxide. The expected rate was 0.58 prostatic cancers.

The IARC concluded that occupational exposure to cadmium in some form increases the risk of prostate cancer in man. Heavy exposure to cadmium therefore causes an excessive death rate from prostatic cancer (Piscator, 1981).

Sarcomata at injection sites in rats have been found after subcutaneous or intramuscular injections of low doses of cadmium in the form of metal powder and sulfide. Higher doses produced testicular tumors (Piscator, 1981).

CHAPTER 9

ENVIRONMENTAL EFFECTS ASSOCIATED WITH MERCURY

Introduction

The Agency for Toxic Substances and Disease Registry (1992) classified mercury in three groups: elemental mercury, inorganic mercury salts, and organic mercury. Elemental mercury is volatile when it is heated. The EPA (1987) considers mercury as a hazardous air pollutant.

According to Leonard et al. (1983), the electrical apparatus industry uses 25% of the mercury produced; the chlorine-alkali industry uses 20%; the ship-bottom paints industry uses 15%; the industrial and control instruments industry uses 10%; the dental preparations industry uses 5%; and laboratories, pharmaceuticals, amalgams and catalysts use 15%.

Catalysts

Kew (1980) and Nriagu (1979a) emphasize the importance of mercury as a catalyst used in the synthesis of vinyl chloride and vat dyes. These catalysts are employed for converting acetylene into acetaldehyde, vinyl chloride and vinyl acetate; the last two being the starting materials for the production of polyvinyl chloride (PVC) and polyvinyl acetate (PVA). The catalysts are commonly mercuric chloride (vinyl chloride) and mercuric sulfate (vinyl acetate). Carbon pellets impregnated with mercuric chloride are the major source of contamination. In the dyestuff industry, mercuric sulfate is used as a catalyst in the production of anthroquinone derivatives of dyes. Organomercurial salts have also been used as catalysts in the production of urethane and urethane resins. The disposal of spent catalysts to the land is the major source of release of mercury in this category. Only 100 kg/year is released to air and 200 kg/year to water. Because recovery of mercury from mercurial catalysts is not economical, mercury is being effectively replaced by other materials.

Air

The Agency for Toxic Substances and Disease Registry (1989) reported a concentration of mercury in the atmosphere of 20 ng Hg/m^3. The anthropogenic releases of mercury to the atmosphere have been estimated to be 2,000-3,000 metric tons/year, mostly from the mining and smelting of mercury ores, industrial processes involving the use of mercury, and combustion of fossil fuel.

Water

The Agency for Toxic Substances and Disease Registry (1989) estimated a concentration of mercury in fresh water of 0.025 ug Hg/l. The level of inorganic mercury in rivers, lakes, and streams is limited to 144 ug/l of water, and all releases of more than one pound of mercury metal need to be reported. The FDA level of mercury in bottled water is no more than 2 ug/l, and the EPA's limit is 0.021 milligrams of inorganic or organic mercury per day in food or in water.

Mercury may also be released to surface waters in effluents from a number of industrial processes including ink manufacturing. Kew (1980) mentioned levels of 0.2-85 ug/l of mercury in urban runoff, and 0.2 ug/l to 0.6 ug/l in stormwater and combined sewer runoff in 11 cities across the U.S.

The Agency for Toxic Substances and Disease Registry (1992) noted an elevated mercury level in 25% of groundwater and surface-water samples from 2,785 hazardous waste sites tested. Industrial processes that may result in mercury-containing effluent include chloride and caustic soda production , mining and ore processing, metallurgy and electroplating, chemical and ink manufacturing, paper milling, leather tanning, textile manufacturing, and pharmaceutical production.

Soil and water microorganisms methylate mercury. The resulting substance is methyl mercury, which accumulates rapidly in fish and other aquatic organisms. This compound accounts for 70 to 90% of the total mercury detected in fish. The mercury concentration in fish at the top of the food chain is typically biomagnified up to 100,000 times the concentration in surrounding waters. Predacious fish can have more than 50 times the average mercury concentration found in most other fish. Fish and fish products have concentrations of mercury from 0.1 to 0.22 mg Hg/kg fish. This amount is below the

FDA limit for edible fish of 0.5 mg/kg (Agency for Toxic Substances and Disease Registry, 1992).

Minamata Bay in Japan was contaminated with mercury in the 1950's by effluent discharged from a factory that used a mercury catalyst. Fish consumed from this bay poisoned hundreds of people, causing 41 deaths. Studies of methylmercury concentrations in the blood of newborns showed a significant correlation with maternal blood levels. Damage to the fetal nervous system and derangement of developmental processes such as neuronal migration and neuronal cell division were seen in newborns whose mothers were exposed to contaminated fish (Agency for Toxic Substances and Disease Registry, 1992).

Landfills and Soil

According to Kew (1980), the sources of release of mercury in land areas are landfilling and lagooning of industrial and municipal sludge, flyash disposal, and agricultural applications. Mercury migration to groundwater probably happens only in poorly operated landfill sites. However, we need to consider that over one-half of the U.S. landfill sites did not comply with regulatory requirements. Little information is available concerning mercury contamination through sludge and fertilizer application to agricultural sites. Mercury in the sludge is assumed to be less available for biological uptake and leaching. Accumulation in the soil surface is likely for mercury applied in this form.

Animals

Taylor (1979) mentioned that since mercury is a well-known aquatic pollutant, it is included in the black list of all the international conventions such as the Oslo, Paris and Barcelona Conventions and the EEC Directive on the discharge of dangerous substances.

Eisler (1987) listed early developmental stages as the most sensitive in all organisms tested, and methylmercury as more toxic than inorganic forms. Lethal concentrations of total mercury to sensitive organisms varied from 0.1 to 2.0 ug/l of medium for aquatic fauna; from 2.2 to 31 mg/kg body weight (acute oral) and 4 to 40 mg/kg (dietary) body weight for birds; and from 0.1 to 0.5 mg/kg body weight and 1 to 5 mg/kg body weight for mammals.

Mercury is a known mutagen, teratogen, and carcinogen. At comparatively low concentrations in birds and mammals, it adversely affects reproduction, growth and development, behavior, blood and serum chemistry, motor coordination, vision, hearing, histology, and metabolism. It has a high potential for bioaccumulation and biomagnification, and is slow to be metabolized by the body (Eisler, 1987).

Aquatic Organisms

According to Eisler (1987), toxic concentrations of mercury salts ranged from less than 0.1 ug/l to more than 200 ug/l for representative species of marine and freshwater organisms. Concentrations lower than 2.0 ug/l were usually associated with early developmental stages, long exposures, and flow through tests. Among metals tested, mercury was the most toxic to aquatic organisms. Signs of acute mercury poisoning in fish included flaring of gill covers, increased frequency of respiratory movements, loss of

equilibrium, sluggishness, and effects on the reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation, and oxygen exchange. Reproduction was inhibited among sensitive species of aquatic organisms at water concentrations of 0.03 to 1.6 ug Hg/l. Reduced growth of sensitive species of aquatic organisms has been recorded at water concentrations of 0.04 to 1 ug Hg/l. Signs of chronic mercury poisoning included emaciation, brain lesions, cataracts, diminished response to change in light intensity, inability to capture food, abnormal motor coordination, and various erratic behaviors. In general, the accumulation of mercury by aquatic biota is rapid, and depuration is slow.

Kew (1980) reported on chronic or sublethal toxicity for freshwater invertebrates. Minimum chronic effects levels for Dahnia magna were 0.9 ug/l and less than 0.01 ug/l for $HgCl_2$ and CH_3HgCl, respectively. The lowest concentration of mercury resulting in sublethal effects in marine finfish was 10 ug/l $HgCl_2$, causing abnormal development in the mummichog Fundulus Heteroclitus and decreased respiration in the winter flounder, Pseudopleuronecies americanus. The mean number of broods of brine shrimp decreased with concentrations of 1 ug/l and 10 ug/l $HgCl_2$ and of 1 ug/l CH_3HgCl, and adult reproductive lifespans were reduced at concentrations of 10 ug/l $HgCl_2$ and 5 ug/l CH_3HgCl. However, 10 ug/l $HgCl_2$ had no effect on the average number of offspring produced in each brood, while 1 ug/l CH_3HgCl significantly reduced the fecundity of the shrimp. The fiddler crab exhibited an increased metabolic rate when exposed to 1.8 ug/l $HgCl_2$. Sublethal effects in other invertebrates included decreased egg production, reduced shell growth, and inhibition of limb regeneration.

Terrestrial Organisms

According to Eisler (1987), mercury causes teratogenic, mutagenic, and carcinogenic effects in mammals; the fetus is the most sensitive life stage. Methylmercury irreversibly destroys the neurons of the central nervous system. Dietary concentrations of 3 mg/kg CH_3HgCl produced adverse reproductive effects in mallards and black ducks; oral doses of 13 mg/kg and 60 mg/kg were lethal to goshawks and ducklings, respectively.

Birds

Eisler (1987) depicted the signs of mercury poisoning in birds as muscular incoordination, falling, slowness, fluffed feathers, calmness, withdrawal, hyporeactivity, hypoactivity, and eyelid drooping. Sublethal effects of mercury, administered by a variety of routes on birds, included adverse effects on growth, development, reproduction, blood and tissue chemistry, metabolism, and behavior; histopathology and bioaccumulation were also noted.

Plants

Kew (1980) reviewed the effects of residues of 0.6 mg/kg and 10 mg/kg of mercury on maize seedlings. This concentration inhibited growth in the shoots and roots respectively. Methylmercury can act directly upon the genetic material of plants, producing chromosome fragmentation, somatic mutations and pollen sterility. According

to Siegel et al. (1984), mercury vapor, primarily elemental mercury, can accelerate senescence-related processes due to accelerating the production of ethylene.

CHAPTER 10

HEALTH EFFECTS ASSOCIATED WITH MERCURY

Introduction

Because mercury evaporates at room temperature and quickly builds up in the air as an odorless vapor, it needs to be handled carefully. This vapor is monatomic and lipid soluble. Therefore, 80% of mercury vapor inhaled will be absorbed through the alveoli upon inhalation (McCarthy, 1993 and Cragle et al., 1984).

Mercury Concentration Limits

Cragle et al. (1984) reported nonspecific symptoms such as shyness and loss of appetite in men exposed to less than 0.1 mg/m^3 time-weighted average (TWA) air concentrations of elemental mercury.

Many organizations report their limits of mercury in the workplace as the threshold limit value that is the Time Weighted Average (TWA) for an eight-hour day or 40-hour week. The Occupational Safety and Health Administration (OSHA) TWA for organic mercury is 0.1 mg/m^3, and for inorganic vapor is 0.05 mg/m^3. The National

Institute for Occupational Safety and Health and the American Conference of Governmental Industrial Hygienists TWA for inorganic mercury is 0.050 mg/m^3 (Burt, 1986; McCarth, 1993; Kew, 1980; Agency for Toxic Substances and Disease Registry, 1989 and Tierney et al., 1979) .

Mercury Exposure Indicators

Cragle et al. (1984) and Burt (1986) found urine mercury levels as a good indicator of personal exposure to inorganic mercury but not predictors of symptoms. The World Health Organization (WHO) uses a 1:2 ratio to relate air concentration of inorganic mercury in ug/m^3 and urine mercury levels in mg/l.

According to Susuki et al. (1991), slight effects on the central nervous system may occur in groups of workers with average urinary mercury levels of about 5 ug/g creatinine and possibly lower.

Acute Effects

Nriagu (1979a) pointed out that inorganic mercury is rapidly lost from the body. Therefore, exposure effects may disappear if the worker is removed from exposure for a period of time. Only in the case of recurrent exposures and toxicity, do manifestations of mercurialism become chronic.

Nriagu (1979a) and Cragle et al. (1984) indicated that acute toxicity due to inhalation of miligrams of mercury can cause, after a delay of a few hours, a metal fume fever with symptoms of nausea, abdominal cramps, diarrhea, muscle aches, fever and an

elevated white blood cell count and, within a few days, a metallic taste as well as further inflammation of the gums, loosening of the teeth, ulcers of the mouth and a blue line at the gum margins. In extreme instances, the exposure victim may die of acute chemical pneumonitis.

Roels et al. (1982), Nriagu (1979a) and Kew (1980) noted that the central nervous system and the kidney are the two major organs affected by mercury exposure. In low doses, mercury may induce renal and liver disease, and occasionally a tremor is noted.

The Agency for Toxic Substances and Disease Registry (1992) pointed out that in a survey performed by NIOSH, 70,000 workers were estimated to be potentially exposed to mercury in the workplace. In this number were included ink manufacturing workers.

Chronic Effects

Kew (1980) noted that the beginning of chronic mercury poisoning is often slow and insidious. The effects of mercury exposure appear in sequences. It begins with progressive numbness of the distal parts of the extremities and often of the lips and tongue, and is followed by an ataxic gait, clumsiness of the hands, dysarthria, dysphagia, deafness, and blurring of vision.

The EPA (1987a) pointed out that exposure to levels below 0.1 mg Hg/m3 causes non-specific symptoms such as introversion, insomnia, and anxiety. Kew (1980) reported that inhalation of 0.1 to 0.6 mg/m^3 of mercuric chloride may cause non-specific signs such as insomnia, loss of appetite and weight loss.

According to the EPA (1984, 1987a) and Kew (1980), in the workplace, chronic mercury vapor intoxication has resulted in mental disturbances (short term memory loss and changes in personality characteristics), objective tremors, and gingivitis, at average air

concentrations less than 0.1-0.2 mg Hg/m^3. Tremor is caused by the motor disfunctioning of the central nervous system.

Systemic Effects

Gingivitis

Gingivitis was defined by McCarthy (1993) as a disease caused by poor dental hygiene, and is seen initially as sore gums. According to McCarthy (1993) and Nriagu (1979a), atrophic changes appearing in the gums, known as gum disease, is a common sign of chronic high level mercury vapor exposure. The gums initially become swollen and boggy and later retract. In individuals who have pre-existing pyorrhea, evidence of infection can be aggravated. In severe cases, there can be loosening of the teeth with bony re-absorption of the jaw. With severe chronic exposure, one can even note necrosis of the lower jaw like the oral pathology noted in acute vapor toxicity.

Gastrointestinal Effects

The Agency for Toxic Substances and Disease Registry (1989) lists common symptoms from acute exposure to mercury vapor as nausea, vomiting, gingivitis, and mercurial stomatitis.

Cardiovascular Effects

The Agency for Toxic Substances and Disease Registry (1989) reported that acute inhalation of metallic mercury vapor can result in increased blood pressure.

Renal Effects

McCarthy (1993), the Agency for Toxic Substances and Disease Registry (1989) and Nriagu (1979a) remarked on the effects of mercury exposure on the renal system. The kidney has a remarkable capacity to concentrate mercury, due to its ability to eliminate mercury from the body, and is a target organ when inhalation exposure to metallic mercury occurs. The first sign of kidney damage is increased creatinine excretion, which means renal insufficiency, followed by damage of the proximal tubules.

Dermal/ocular Effects

The Agency for Toxic Substances and Disease Registry (1989) pointed out that acute exposure for 2 weeks to mercury concentrations less than 1 mg/m^3, and intermediate exposure for 2 months at unspecified concentrations, resulted in the beginning of erythematous and pruritic skin rashes. Ocular effects observed from this same acute exposure included red, burning eyes and conjunctivitis.

Nriagu (1979) and the Agency for Toxic Substances and Disease Registry (1989) stated that workers chronically exposed to low concentrations of mercury exhibited mercurialentis, which is a peculiar grayish-brown or yellow haze on the outer surface of their lenses, decreasing visual acuity.

Musculoskeletal Effects

Kew (1980) mentioned the existence of spasticity and rigidity due to exposure to mercury. Muscle stretch reflexes are usually maintained or become hyperactive, and extensor plantar responses are occasionally presented during the later stages. Insomnia, agitation, hypomania, and the loss of emotional control are frequently noted, and most individuals have abnormal involuntary movements, including choreoathetosis, myoclonus, and coarse resting and action tremors.

McCarthy (1993) reported that employees subjected to exposure to high concentrations of mercury oxide particles and vapor for 60-80 hours a week, 7 days a week, presented incapacitating muscular pains of the lower back and extremities, severe burning sensations of the feet and lower legs, muscle cramps, and muscle fasciculations.

Neurological Effects

According to Burt (1986) and Williamson et al. (1982), the central nervous system is the part of the body most severely affected by chronic high-level mercury exposure. This causes long-lasting and deep neurological damage. The initial outcomes of toxicity consist of fine tremor and erethism.

Tremor

McCarthy (1993) and Nriagu (1979) stated that tremors are the earliest and most notable mark of chronic exposure to moderate levels of inorganic mercury. It is seen at the beginning as a fine tremor in the hands; then it develops to a fine postural tremor. This is associated with trouble in accomplishing fine movements, in coordination, difficulty with gait (ataxia) and even hoarseness due to ataxia of the vocal cords. With greater or longer-

period exposures, the tremor multiplies in amplitude and becomes coarse, which is aggravated by voluntary movements. As the severity of the tremor increases, it can be interrupted by clonic-like jerks of one or more extremities. This progresses to involve the entire body as well.

Erethism

McCarthy (1993) and Nriagu (1979) defined erethism as a form of psychic disturbance. Workers affected experience nervousness, irritability and change of temperament. They become easily upset and embarrassed and lose self-confidence, and often have a feeling as if they are being watched. They usually have headaches, drowsiness, insomnia, fatigue or flushing.

Williamson et al. (1982) compared a group of 12 chronically mercury-exposed workers with a matched control group. The mercury-exposed group showed poorer psychomotor co-ordination and premature fatigue, although simple motor responses were not affected. General arousal levels also remained unaffected, but mercury-exposed workers were superior in sustaining attention. In spite of this, mercury-exposed groups showed clear deficits in short-term memory.

Respiratory effects

The Agency for Toxic Substances and Disease Registry (1989), Milne at al. (1973) and Nriagu (1979a) discuss respiratory effects as a result of mercury vapor intoxication. Due to inhalation of metallic mercury vapor through the lungs, the metal is absorbed, causing pulmonary irritation with chest tightness, a cough and shortness of breath followed by pulmonary edema, lobar pneumonia, desquamation of the bronchiolar epithelium, and death. The resulting obstruction (bronchiolar blockage by mucus and fluid)

results in alveolar dilation, emphysema, and pneumothorax. Death can occur due to these pulmonary effects. Survivors can acquire chronic shortness of breath as well as interstitial fibrosis of the lungs.

The Agency for Toxic Substances and Disease Registry (1989) separated human health effects from breathing metallic mercury into short-term and long-term exposure. At levels in air of 0.13 ppm for 3 hours, workers experienced chest pains, shortness of breath and cough. At levels in air of 5.4 ppm for 8 hrs, workers experienced persistent irritability, lack of ambition and lack of sexual desire. At levels in the air of 1.1 to 44 mg/m^3 for 4-8 hours workers exhibited chest pains, dyspnea, cough, hemoptysis, impairment of pulmonary function (reduced vital capacity), diffuse pulmonary infiltrates, and evidence of interstitial pneumonitis. At levels in air of 0.0032 ppm for 15 years, the workers experienced shakiness.

Developmental Effects

The Agency for Toxic Substances and Disease Registry (1989) and Clarkson (1993) stated that women exposed to methylmercury in fish may give birth to babies with severe brain damage. This happened in the Minamata Bay case, where 30 cases were reported. The mothers experienced no symptoms or only mild effects such as transient paresthesia. The prenatal brain damage was diffuse and widespread, due to interference with neuronal migration. The occurrence of microcephaly suggested that cell division had been suppressed. In women chronically exposed to metallic mercury vapor, increased frequencies of menstrual disturbances and spontaneous abortions were seen.

Mutation

According to the EPA (1984) and the Agency for Toxic Substances and Disease Registry (1989), chromosomal anomalies such as aneuploidy have been reported in lymphocytes from whole blood cultures of workers occupationally exposed to organic or inorganic mercury compounds, mainly by inhalation. Also, chromosome aberrations were reported in workers exposed to metallic mercury vapor.

CHAPTER 11

ENVIRONMENTAL EFFECTS ASSOCIATED WITH LEAD

Introduction

Lead is not essential nor beneficial to any living organisms (Eisler, 1988). Lead is incorporated in animals and humans by inhalation, ingestion, dermal absorption, and placental transfer to the fetus.

Uses

Lansdown and Yule (1986) estimated the world production of lead in 1986 as 5 million metric tons. Since 1981 the consumption of lead by industry has decreased in the USA and UK.

Winder (1984) discusses the use of lead II carbonate as a pigment. In the manufacturing of this material, sheets of lead undergo slow decomposition under the action of acetic acid vapor, moist air and carbon dioxide. Other lead compounds used as pigments are Lead II chromate (chrome yellow) and basic chromate (chrome red). Pigments used in paints have been replaced due to their toxicity. The substitute for lead

pigments in paints is titanium dioxide due to its opacity, covering power, cheapness, stability, and of course, safety.

Lansdown and Yule (1986) discuss use of lead as a stabilizer in the manufacturing of certain plastics. Polyvinyl chloride is stable over a broad temperature range due to the use of lead carbonate or lead silicate during processing. Lead dithiocarbonate is used also as an accelerator in the manufacture of rubber.

Packaging Inks

Lavelle and Fetsko (1977) listed pigments based on lead as white lead, basic lead sulfate, chrome yellows, phloxine toner, molybdate orange and chrome green. Among these pigments, white lead and chrome yellow cause concern among manufacturer. White lead causes poisoning in cases of ingestion of paint chips. Chrome yellow used in printing ink is the one which increases the content of lead in printed materials. Lead-based pigments are no longer used in printing inks and have been replaced by cobalt or manganese compounds. Lead-based pigments are part of different kinds of industrial coatings. Their low cost, good dispensability and opacity make them popular. Lead-based coatings are not considered hazardous because they are insoluble .

20 years ago lead content in paper used in the food industry ranged from 2 to 10,000 ppm depending on the quantity of printing and color of ink. Printed paper packages contained more lead than unprinted material, although some unprinted ones contained up to 58 ppm due to the use of recycled paper or virgin mixed with recycled paper (Heichel et al., 1976). The amount of lead in paper, both recycled and virgin, has decreased because of the decrease of heavy metal pigment used.

Lavelle and Fetsko (1977) referred to the British Printing Ink Manufacturer's clause b (ii) that limited lead content in immediate food wrappers for ice cream and ice lollies, and marking inks for pencils. The Society of British Printing Ink Manufacturers

recommended the elimination of lead and lead chromates from all printing inks in U.K. Hankin et al. (1974) mentioned the FDA regulation issued around 1950 which limits the use of lead by food packaging industries. According to such regulations, lead cannot exceed 0.06% (600 ppm) of the total weight of dried paint film on the package. The Hazardous Substances Act bans any paint or surface coating material intended for use in or around the house that contains more than 0.5 % (5000 ppm) lead in the coating. Hutchinson (1979) stated that the European Economic Community Directive mandates the labeling of printing inks with a harmful symbol where the soluble lead content is equal to or exceeds 1%.

Disposal of printing ink refuse can be simplified by the elimination of lead containing materials. The increase of lead content in recycled packaging material, potentially contaminating food products, can be avoided by reducing the content of lead-based ink used in the package, such as in corrugated cases where contamination of the content can be reduced by using flexographic inks formulated with lead-free pigments (Hutchinson, 1979) .

Organic pigments are replacing pigments such as Primrose chromate, Lemon chrome, Mid chrome and scarlet chrome shades. Difficulties arise in the matches for flexo inks for black colored grounds, films and foil, scarlet chrome and mid-chrome shades. Lead chromate give a broad variety of shades that in many cases is difficult to substitute. In addition, the new organic pigments used to replace lead-based pigments are relatively high in cost (Hutchinson, 1979).

Air

Lansdown and Yule (1986) estimated anthropogenic emissions of lead from mining as 8.2 tonnes/year , from primary lead production as 31 tonnes/year, from primary non-ferrous production as 45.5 tonnes/year, from secondary smelting as 0.8 tonnes/year , from iron and steel production as 50 tonnes/year, from industrial uses as 7.4 tonnes/year, from coal combustion as 14 tonnes/year, from petrol combustion as 273 tonnes/year and from waste incineration as 10.4 tonnes/year . The total anthropogenic emission of lead was estimated as 449 tonnes/year in 1980 . According to Needleman (1991), lead in the atmosphere derived from human activities, especially combustion of oil and its derivatives, has changed the natural cycle of lead.

The concentration of lead in urban areas is greater than the concentration in rural and sub rural areas. These concentrations average 1 ug/m^3 to 3 ug/m^3 in urban areas, 0.1 ug/m^3 in suburban areas, and less than 0.05 ug/m^3 in rural areas (Tierney at al., 1979).

Soil

According to Lansdown and Yule (1986) and Needleman (1991), due to its long half-life in soil, lead is in general immobile. Lead can persist in soil for many years after deposition has stopped.

Needleman (1991) pointed out that lead associates with dust (small particles) in soil. If these particles are resuspended in air, they can stay suspended for a long time. Dust with high lead content, therefore, is hazardous because it is not visible and can remain

trapped in carpet and upholstery. The concentration of lead in soil ranges from 10 to 50 ug/g in rural areas, and from 100 to 10,000 ug/g in urban areas.

Water

Needleman (1991) mentioned the presence of lead in small quantities in ground and natural surface water. Due to the insolubility of lead compounds, the concentration of lead in water supplies is low, with concentrations averaging less than 5 ug/l.

Aquatic Organisms

Eisler (1988) stated that lead is toxic to all aquatic biota. Waterborne lead is the most toxic. Lead concentrations of more than 10 ug/l in water produce spinal curvature; anemia; darkening of the dorsal tail region, producing a black-tail effect due to selective destruction of spinal neurons; ALAD inhibition in erythrocytes, spleen, liver, and renal tissues; reduced ability to swim against a current; destruction of the respiratory epithelium; basophilic stippling of erythrocytes; elevated lead concentrations in blood, bone, gill, liver, and kidney; muscular atrophy; paralysis; renal pathology; growth inhibition; retardation of sexual maturity; altered blood chemistry; testicular and ovarian histopathology; and death in fish. Although lead is concentrated by biota from water, there is no convincing evidence that it is transferred through food chains.

Birds

Eisler (1988) discussed mortality in waterfowl and other birds due to ingestion of spent lead shot. In sensitive birds, a reduction in survival rate was seen at doses of 75 to 150 mg Pb^{+2}/ kg or 28 mg alkyl lead/kg. Signs of toxicity in birds are loss of appetite, lethargy, weakness, emaciation, tremors, drooped wings, green liquid feces, and impairment of locomotion, balance, and depth perception.

Plants

Eisler (1988) referred to lead as a non-essential element for plants. On the contrary, high amounts of lead cause growth, photosynthesis, mitosis and water absorption reduction. Due to the long half-life of lead in soil, several hundred mg lead/kg in soil are needed to result in any adverse effects.

Lansdown and Yule (1986) studied the uptake of lead by plants. They stated that lead in edible plants is unaffected by increasing lead content in soil. Therefore, lead content in foodstuffs is limited.

CHAPTER 12

HEALTH EFFECTS ASSOCIATED WITH LEAD

Introduction

Hutchinson (1979) mentioned inhalation and ingestion as the only sources of lead poisoning in the printing industry, and small quantity exposure at regular periods of time as the most prevalent cause of poisoning. Inhalation of dust and fine powder is the major occupational risk. For these reasons, dust in workplaces needs to be kept to an absolute minimum. Needleman (1991) estimated that 40 - 50% of the lead inhaled is absorbed. Small particles (1 to 2 um) are trapped in the upper respiratory tract and passed to the digestive tract to be ingested. The remaining particles pass to the lower lung.

Eisler (1988) described progressive development of lead poisoning as mild or severe dysfunction of the alimentary tract as shown by loss of appetite, constipation, abdominal cramps, headaches, general weakness, fatigue, atrophy of forearm extensor muscles, or paralysis of these muscles and more striking atrophy. Lead encephalopathy occurs frequently in lead-poisoned infants and young children.

According to OSHA (1975), the threshold limit value for lead for an 8 hour-day in the workplace is 50 ug/m^3. OSHA suggests care in the handling of lead and its compounds. Workers who do not practice personal hygiene are at big risk. It is necessary

to wash any clothes used at work and shower before returning home. Alexander (1989) reported elevated blood lead levels among children of lead-exposed workers due to high levels of lead in the parents' clothing.

Eisler (1988) and Winder (1984) considered the hazardous increase of lead contamination in the environment. Exposure to environmental lead concentrations can put human health at risk. 7 ug of lead/l in human blood is enough to produce chronic symptoms in humans. Such concentration is close to the 2.3 ug/l that is the present average concentration of lead in human blood. Therefore, it is necessary to keep environmental lead levels to minimum.

Neurotoxicity

According to Winder (1984), exposure to lead for a long period of time may cause hyperactivity, mental retardation, intellectual and psychological impairment, and other behavioral changes. Urban air may contain enough lead to cause impairment of brain function in children. Needleman (1991) and Singhal and Thomas (1980) recognized that children ingest lead existing in the environment in forms such as dirt and dust. They referenced studies of children living in urban areas who had difficulties in completing intelligence, perceptual motor, and memory tests.

Singhal and Thomas (1980) pointed out behavioral changes as the most permanent and difficult effects to diagnose. The initial symptoms of behavioral changes are depression, insomnia, irritability, memory impairment, and clumsiness, which are difficult to recognize as low-level lead exposure symptoms. The disease develops to encephalopathy, beginning with intractable seizures, and leads to coma and death within a short period of time. In some cases, incidents of vomiting, drowsiness, altered consciousness (stupor), deep ataxia, muscle weakness or tremor, numbness, paralysis, or

persistent headache, or depression may be observed, after which the patient becomes comatose.

Singhal and Thomas (1980) and Winder (1984) described peripheral nerve damage as the most insidious abnormality in workers exposed to lead. Damage to peripheral nerves causes motor disorders such as atrophy of muscles in the forearm and upper arm. Needleman (1991) mentioned some central nervous system related symptoms as visuomotor abnormalities and uncontrolled fine movements at a lead exposure level of 0.4 mg/ cc in blood. Chronic lead exposure caused deficits in short-term memory, and visual acuity was affected.

Reproductive Effects

Singhal and Thomas (1980) reported abortions in women and sterility in men exposed to lead in printing operations. According to the World Health Organization (1977), lead poisoning and moderately increased lead absorption decreased male fertility due to lead's effects on gonad function. Lead not only affects the viability of the fetus, but development as well. Developmental consequences of prenatal exposure to low-levels of lead include reduced birth weight, reduced brain development, and premature birth.

Renal Effects

Singhal and Thomas (1980) considered acute and chronic renal damage as lead toxicity effects. Acute renal damage signs are situated in the proximal tubular epithelium, involving changes in membrane functions and in energy metabolism, structural aberrations of mitochondria, and the appearance of pathognomonic morphological features in nuclei and cytoplasm of proximal tubular epithelial cells.

Singhal and Thomas (1980), the U.S. Department of Health & Human Services (1990), and the World Health Organization (1977) mentioned severe acute neophropathy symptoms such as aminiaciduria, glycosuria, and hypophosphatemia , which result from depressed tubular reabsorption. Prolongation of the acute exposure leads to chronic lead neophropathy, which is usually associated with gout and hypertension. Hypertension is directly associated with atherosclerosis, coronary artery disease, heart disease, stroke, and end-stage renal disease. Renal disease, hypertension, and cerebrovascular disease are frequently reported as significant causes of excess mortality in lead workers. Gout may develop as a result of lead- induced hyperuricemia.

Cancer

Singhal and Thomas (1980) stated that lead exposure can cause the development of tumors (adenomas or adenocarcinomas) in the renal cortex. This development is induced by large doses of lead in the kidney in a short period of time.

Gastrointestinal Effects

The World Health Organization (1977) mentioned that in low-level lead exposure, acute pain in the abdomen can result. Such a symptom is consider a warning to avoid prolonged periods of exposure that can result in severe effects.

Cardiovascular Effects

The World Health Organization (1977) mentioned toxicity in the heart due to lead exposure. The toxicity manifestations were increased capillary permeability, and atherosclerosis.

Mutagenic Effects

The World Health Organization (1977) reported chromosomal aberrations in workers exposed to zinc, cadmium and lead. It was concluded that lead was responsible for the aberrations. Eisler (1988) pointed out that aberrations occurred in human blood lymphocytes .

Hematological Effects

The U.S. Department of Health & Human Services (1990b). stated that lead enters the human body and inhibits the production of hemoglobin in blood, causing two types of anemia. Hemolytic anemia results when the body is exposed to acute high levels of lead.

CHAPTER 13

THE DESIGN OF THE INVESTIGATION AND ANALYSIS OF DATA

The Study Population

This study was limited to companies which manufacture inks for the packaging industry. Individuals in charge of manufacturing, development or research operation in their company were asked to respond to the questionnaire.

The population for this investigation was selected from ink manufacturing companies classified by the National Association of Ink Manufacturers. Two hundred twenty-four companies were identified, each one receiving one questionnaire for a total of 224 possible respondents.

The Instrument

The format used for the instrument was as follows (see appendix B). The first page included the purpose of the study and general return instructions. The statement of the problem spelled out twenty-five questions to be answered. Three of the questions requested specific responses indicating the interest in the CONEG Model Toxics in Packaging legislation. Six questions requested information pertaining to limitations of heavy metal in inks and to written certificates of compliance required by the packaging

industry. Four of the questions required specific responses which indicated the increase of cost and/or price as a result of heavy metal content reduction. Five questions requested information pertaining to substitution of inks that contain heavy metals. The next two questions involved substitution for petroleum oil inks. The last questions in the questionnaire indicated the awareness of any tests and regulations related to use of heavy metals.

The Mailed Questionnaire Survey

The questionnaires were mailed to 224 individuals representing the different ink manufacturing companies. Three and one-half weeks later, 51 questionnaires had been returned. The returned instruments included 2 that had no forwarding address and 17 that indicated the respondent was no longer responsible for the activities described in this investigation. The net result at this point was 32 completed questionnaires, or 14%, a very good response rate for a survey of this type.

The percentage of replies to the survey was considered adequate to draw conclusions for this investigation. The survey provided a good number and distribution of responses. The number of personal comments returned with the survey instrument indicated that those individuals who did respond were doing so out of genuine interest in the problem.

Describing the Data

The data accumulated through the survey instrument will be summarized through statistical means, so that the results will indicate the most frequent option chosen.

The descriptive survey method was used to design and carry out the data collection process for this investigation. The data were arranged on an ordinal scale and thus will be described using descriptive statistics. Descriptive statistics is based upon central tendencies, a means of describing the typical or average values and variability, the spread or extent of the values (Hays, 1994).

Measures of central tendency are the mean and median. The arithmetic mean is calculated by adding the observations and dividing by the number of observations. The median is the point at or below which precisely 50% of the cases fall. The mean is the most serviceable measure for purely descriptive statistics and for distributions which lack a clearly dominant single peak (Hays, 1994 and Juran et al. 1974). Therefore, it will be used for this investigation.

The literature did not record any general rules to follow in setting numeric values for the responses presented in the instrument. We selected the following numbers to describe the data:

Never..... 1

Sometime.....2

Usually.....3

Frequently.....4

Always.....5

or

None of them.....1

Some of them.....2

Most of them.....3

All of them.....4

The mathematical formula for calculating the mean is (Edward, 1967) :

Mean = sum f X / sum f

Where:

X= Level of importance or midpoint of range percent

sum f X = the sum of the product of frequencies and X per range

sum f = the total number of frequencies

Profile of the Responses

The questionnaire asked the respondents to provide information concerning heavy metals used in ink formulation. Table I provides a summary of the responces. Of those companies responding, 94 percent (30 of 32) use copper in their inks, followed by zinc and lead with 53 percent and 44 percent respectively.

Respondents were asked about the Model Toxics in Packaging legislation as developed by the Coalition of Northeastern Governors (CONEG) Source Reduction Council. It was found that all the companies surveyed were familiar with this legislation. However, only three companies in New Jersey, two in Massachusetts, and one in Pennsylvania are in states which have enacted this legislation, limiting the total quantity of lead, cadmium, chromium (VI) and mercury to 100 ppm. 22 percent of the respondents did not know if their states had passed this regulation.

TABLE I

WHICH OF THESE HAS YOUR COMPANY USED IN INK FORMULATION?

Inks	Frequency	Percent
Copper	30	93.8%
Zinc	17	53.1%
Lead	14	43.8%
Cadmium	8	25.0%
Chromium (VI)	9	28.1%
Mercury	3	9.4%
Net Respondents	32	100.0%

Twenty-eight of thirty-two companies surveyed limit the amount of heavy metals in their inks. Table II shows reported limits on heavy metals and the source of limitation. As we can see, there were only two major sources of limitation that the industries took into account : CONEG and customers. We can see that 32 percent of the respondents limit their inks due to CONEG types of regulation. Most of the companies that limit heavy metals to below 100 ppm are regulated by CONEG types of regulations.

Table III and Figure 1 provide information about written certificates of compliance. Customers in the packaging industry usually ask for certificates of compliance. Table IV and Figure 2 illustrate the less frequent request for a certificate of compliance among non-packaging industries. Control of heavy metal content in packages is of greater concern among packaging manufacturers due to the existence of CONEG types of legislation in many states.

Respondents were asked about major non-packaging industries. Responses indicate misunderstanding of the question, with printers of packaging and labels, food packaging and corrugate cardboard industry listed as non-packaging industries. As shown in Table V, toy makers were listed most frequently as the major non-packaging industry that ask for compliance certification.

To comply with legislation, the ink industry uses mostly random sampling (53 percent). 28 percent of the respondents calculate the level of contamination of their products. 12.5 percent of the respondents don't test or use supplier certification (see Table VI). When the industry was asked if an ASTM testing method for heavy metal content for use by the ink industry is needed, 41 percent responded that it is not necessary and 22% that it is necessary.

Among the respondents, 59 percent stated the reduction of heavy metal content affected costs, and 38 percent their prices. Tables VII and VIII show the average increase of cost and price of products due to reduction of heavy metal content as 9% and 5% respectively, in cases where costs or price rose.

TABLE II

QUANTITY OF HEAVY METALS ALLOWED IN INKS AND SOURCES OF LIMITATION

Metal	Quantity in ppm	Source of limitation	Frequency
Copper	<100	CONEG	1
	<100,000	Customers	1
	5000	Customers	1
	>20 (a.i.s)	Waste control districts (a.i.s)	1
	No Limitation		6
Zinc	<100	CONEG	2
	<5		1
	No limitation		4
Lead	<50	Customers	2
	<100	CONEG	2
	<20	CONEG	1
	<1	CONEG	1
	<13		1
	<6		1
Cadmium	<2.5		
	<100	CONEG	2
	<10	CONEG	2
	<1		3
Chromium	<2.5		2
	<100	CONEG	2
	<2.5	CONEG	1
	<5		2
Mercury	<5		2
	<100	CONEG	2
	<20	CONEG	1
	<1		1
Total	<100	CONEG	12
Heavy	<100	Customer	2
Metals	<22		1

TABLE III

HOW OFTEN DO YOUR CUSTOMERS IN THE PACKAGING INDUSTRY ASK YOUR COMPANY FOR A WRITTEN CERTIFICATION OF COMPLIANCE WITH TOXICS LEGISLATION?

	Level	Frequency (f)
Never	1	0
Sometimes	2	12
Usually	3	0
Frequently	4	15
Always	5	3
Sum		30
Mean=	**3.3**	

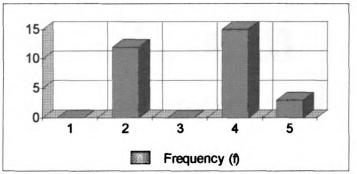

FIGURE 1

FREQUENCY OF USE OF WRITTEN CERTIFICATION OF COMPLIANCE WITH TOXICS LEGISLATION IN THE PACKAGING INDUSTRY

TABLE IV

HOW OFTEN DO YOUR CUSTOMERS IN OTHER INDUSTRIES ASK YOUR COMPANY FOR A WRITTEN CERTIFICATE OF COMPLIANCE WITH TOXICS LEGISLATION?

	Level	Frequency (f)
Never	1	6
Sometimes	2	15
Usually	3	0
Frequently	4	10
Always	5	0
Sum		25
Mean=	2.8	

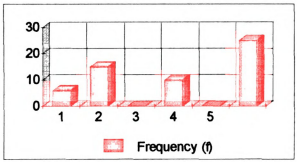

FIGURE 2

FREQUENCY OF USE OF WRITTEN CERTIFICATE OF COMPLIANCE WITH TOXICS LEGISLATION IN NON-PACKAGING INDUSTRIES

TABLE V

MAJOR INDUSTRIES WHICH ASK FOR CERTIFICATION

Industries	Frequency	Percent
Commercial Printers	2	6%
Toy Makers	7	22%
Printers of Packaging & Labels	4	13%
Food Packaging	5	16%
Corrugate Cardboard Industry	3	9%
Net Respondents	32	100%

TABLE VI

WHICH ANALYTICAL METHODS DO YOU GENERALLY USE TO DETERMINE METAL CONTENT IN YOUR INKS?

Responses	Frequency	Percent
Batch-to-batch testing	2	6.3%
Testing of random samples	17	53.1%
Calculation of contamination levels	9	28.1%
Commercial analytical laboratories	2	6.3%
ASTM-63	1	3.1%
Supplier Certification	6	18.8%
Don't test	4	12.5%
Net Respondents	32	100.0%

TABLE VII

BY ABOUT WHAT PERCENT HAVE YOUR COSTS INCREASED DUE TO REDUCTION OF HEAVY METAL CONTENT?

	Mid-point (mp)	Frequency (f)
1-5%	3	9
6-10%	8	4
11-15%	13	1
16-20%	18	6
Sum		20
Mean =	**9**	

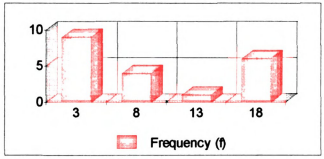

FIGURE 3

PERCENTAGE COST INCREASED DUE TO REDUCTION OF HEAVY METAL CONTENT

TABLE VIII

BY ABOUT WHAT PERCENT HAVE YOUR PRICES INCREASED DUE TO REDUCTION OF HEAVY METAL CONTENT?

	Mid-point (mp)	Frequency (f)
1-5%	3	3
6-10%	8	7
11-15%	13	1
16-20%	18	2
Sum		13
Mean=	4.85	

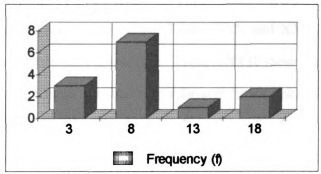

FIGURE 4

PERCENTAGE PRICE INCREASED DUE TO REDUCTION OF HEAVY METAL CONTENT

Tables IX and X provide information concerning reformulation of inks for the packaging and non-packaging industry. We found that 78% of manufacturers have reformulated at least some of their inks for the packaging industry, and 72% for the non-packaging industry, to reduce or eliminate their heavy metal content.

As shown in Tables XI and XII, Napthol and AZO reds are the main substitues for cadmium red. Tables XIII and XIV show diarylide yellow, benzimidazolone and hansa yellow are the major substitutes for cadmium yellow. Tables XV and XIV present dianisidine orange and DNA orange substitutes for cadmium orange. Tables XVII, XVIII, XIX and XX show diarylide yellow and hansa yellow as the main substitute for lead based yellow and chromate yellow. Tables XXI and XXII present diarylide orange as the main replacement for lead orange. Tables XXIII and XXIV show dianisidine orange and DNA exotic as the substitutes for lead molybdate. Tables XXV and XXVI present copper free phtalo blue as the main substitute for copper. Table XXVII show MetalStar Alugold and Toned Aluminum as the main substitutes for bronze based inks. Respondents did not expect future substitutes for bronze based inks. Appendix C includes a table which indicates the different possible substitutes for each heavy metal studied.

The majority of companies surveyed stated that they are almost certain that heavy metals will be replaced in 5, 10 or 20 years. There were seven companies that already have totally replaced heavy metals (Table XXVIII).

From Table XXIX, we can conclude that the quantity of ink produced in each company is kept as a corporation secret. When we asked about such quantity, the majority of companies did not respond.

Few companies have switched or plan to switch to soybean oil inks (see Tables XXX and XXXI). Table XXXII illustrates that the most influential regulations or tests related to use of heavy metals in the ink industry are the Resources Conservation and Recovery Act, Clean Water Act, and Toxic Characteristic Leaching Procedure, in addition to CONEG types of regulation.

TABLE IX

HAVE YOU REFORMULATED ANY OF YOUR INKS FOR THE PACKAGING INDUSTRY TO REDUCE OR ELIMINATE THEIR HEAVY METAL CONTENT?

	Level	Frequency (f)
None of them	1	7
Some of them	2	12
Most of them	3	5
All of them	4	8
Sum		32
Mean=	2.44	

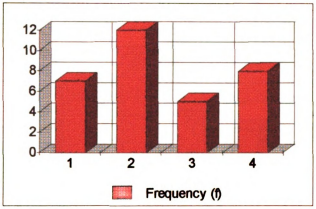

FIGURE 5

REFORMULATION INKS FOR THE PACKAGING TO REDUCE OR ELIMINATE HEAVY METAL CONTENT

TABLE X

HAVE YOU REFORMULATED ANY OF YOUR INKS FOR THE NON-PACKAGING INDUSTRY TO REDUCE OR ELIMINATE THEIR HEAVY METAL CONTENT?

	Level	Frequency
None of them	1	9
Some of them	2	12
Most of them	3	6
All of them	4	4
Sum		31
Mean=	2.16	

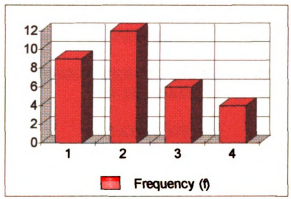

FIGURE 6

REFORMULATION OF INKS FOR THE NON-PACKAGING TO REDUCE OR ELIMINATE HEAVY METAL CONTENT

TABLE XI

CADMIUM RED REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
Napthol	18	45.0%
AZO Reds	12	30.0%
Quinacridone	7	17.5%
Disazo	3	7.5%
Total	40	100%

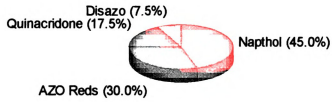

FIGURE 7

CADMIUM RED REFORMULATION IN THE PAST

TABLE XII

CADMIUM RED REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
AZO Reds	4	17.4%
Quinacridone	5	21.7%
Napthol	10	43.5%
Disazo	4	17.4%
Total	23	100.0%

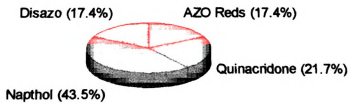

FIGURE 8
CADMIUM RED REFORMULATION IN THE FUTURE

TABLE XIII

CADMIUM YELLOW REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
Benzimidazolone	12	30.0%
Hansa Yellow	7	17.5%
Diarylide Yellow	18	45.0%
Titanium Dioxide	3	7.5%
Total	40	100.0%

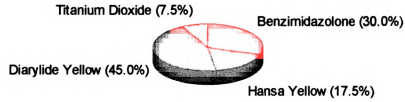

FIGURE 9

CADMIUM YELLOW REFORMULATION IN THE PAST

TABLE XIV

CADMIUM YELLOW REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
Benzimidazolone	2	11.1%
Hansa Yellow	7	38.9%
Diarylide Yellow	9	50.0%
Total	18	100.0%

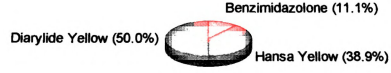

FIGURE 10

CADMIUM YELLOW REFORMULATION IN THE FUTURE

TABLE XV

CADMIUM ORANGE REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
Dianisidine Orange	12	63.2%
DNA Orange	7	36.8%
Total	19	100.0%

FIGURE 11

CADMIUM ORANGE REFORMULATION IN THE PAST IN THE PAST

TABLE XVI

CADMIUM ORANGE REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
Exotic Oranges	1	8.3%
Dianisidine Orange	7	58.3%
DNA Orange	3	25.0%
AAOT Yellow	1	8.3%
Total	12	100.0%

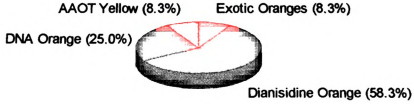

FIGURE 12

CADMIUM ORANGE REFORMULATION IN THE FUTURE IN THE FUTURE

TABLE XVII

LEAD BASED YELLOW REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
Hansa Yellow	3	25.0%
AAOT	2	16.7%
Diarylide Yellow	5	41.7%
Monoarylide yellow	1	8.3%
Titanium Dioxide	1	8.3%
Total	12	100.0%

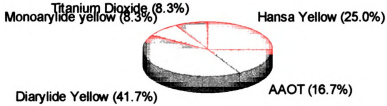

FIGURE 13

LEAD BASED YELLOW REFORMULATION IN THE PAST

TABLE XVIII

LEAD BASED YELLOW REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
Diarylide Yellow	3	60.0%
Monoarylide	1	20.0%
Titanium Dioxide	1	20.0%
Total	5	100.0%

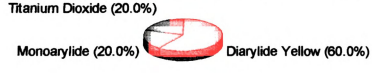

FIGURE 14
LEAD BASED YELLOW REFORMULATION IN THE FUTURE

TABLE XIX

CHROMATE YELLOW REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
Hansa Yellow	11	34.4%
Exotic Yellow	1	3.1%
Diarylide Yellow	19	59.4%
Benzimidazolone	1	3.1%
Total	32	100.0%

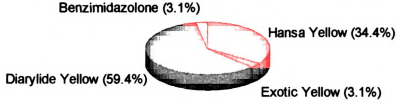

FIGURE 15

CHROMATE YELLOW REFORMULATION IN THE PAST

TABLE XX

CHROMATE YELLOW REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
Hansa Yellow	5	33.3%
Diarylide Yellow	8	53.3%
Benzimidazolone	1	6.7%
Titanium Dioxide	1	6.7%
Total	15	100.0%

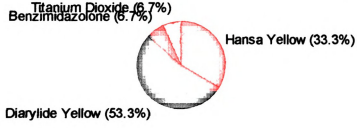

FIGURE 16
CHROMATE YELLOW REFORMULATION IN THE FUTURE

TABLE XXI

LEAD ORANGE REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
DNA Orange	2	14.3%
Diarylide Orange	4	28.6%
Dianisidine Oran.	2	14.3%
Napthol	2	14.3%
Azo Red	2	14.3%
Titanium Dioxide	2	14.3%
Total	14	100.0%

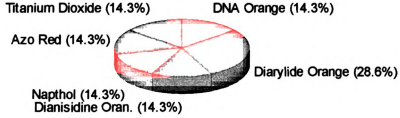

FIGURE 17

LEAD ORANGE REFORMULATION IN THE PAST

TABLE XXII

LEAD ORANGE REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
Diarylide Red	1	25.0%
Diarylide Orange	1	25.0%
AZO Red	1	25.0%
Titanium Dioxide	1	25.0%
Total	4	100.0%

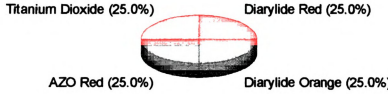

FIGURE 18
LEAD ORANGE REFORMULATION IN THE FUTURE

TABLE XXIII

LEAD MOLYBDATE REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
Dianisidine Oran.	12	54.5%
DNA Exotic	9	40.9%
Benzimidazolone	1	4.5%
Total	22	100.0%

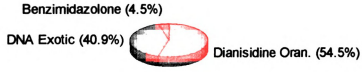

FIGURE 19

LEAD MOLYBDATE REFORMULATION IN THE PAST

TABLE XXIV

LEAD MOLYBDATE REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
Dianisidine Orange	5	100.0%

TABLE XXVI

COPPER REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
Cu free phtalo blue	4	57.1%
Peacock Blue	1	14.3%
Ultramarine Blue	1	14.3%
Cobalt Blue	1	14.3%
Total	7	100.0%

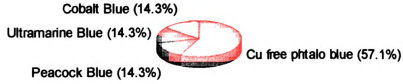

FIGURE 20

COPPER REFORMULATION IN THE PAST

TABLE XXVI

COPPER REFORMULATION IN THE FUTURE

Substitutes	Frequency	Percent of Responses
Cu free phtalo blue	2	40.0%
Peacock Blue	1	20.0%
Ultramarine Blue	1	20.0%
Cobalt Blue	1	20.0%
Total	5	100.0%

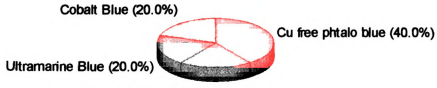

FIGURE 21

COPPER REFORMULATION IN THE FUTURE

TABLE XXVII

BRONZE REFORMULATION IN THE PAST

Substitutes	Frequency	Percent of Responses
MetalStar Alugold	3	37.5%
Croda ink	1	12.5%
Acrylac gold	1	12.5%
Toned Aluminum	3	37.5%
Total	8	100.0%

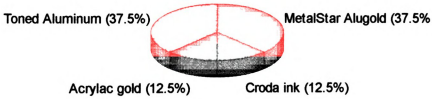

FIGURE 22

BRONZE REFORMULATION IN THE PAST

TABLE XXVIII

REPLACEMENT OF HEAVY METAL INKS IN THE FUTURE

Option/Year	1	5	10	20
Almost Certain	3	7	4	4
Probably will	1	2	1	0
Not sure	2	3	1	1
Probably won't	4	3	2	1

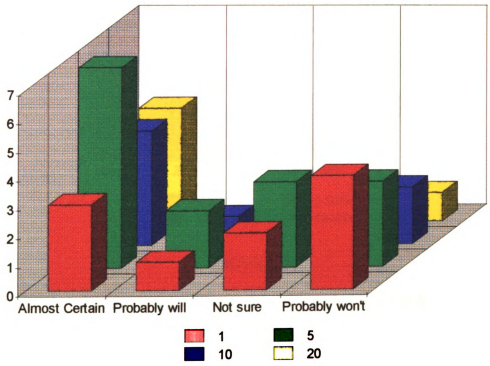

FIGURE 23

REPLACEMENT OF HEAVY METAL INKS IN THE FUTURE

TABLE XXIX

ESTIMATION OF SALES OF HEAVY METALS

Percent of sales	Copper	Zinc	Lead	Chromium
1-5%	4	3	2	1
6-10%	2	2	1	1
11-15%	3			
16-20%	2			

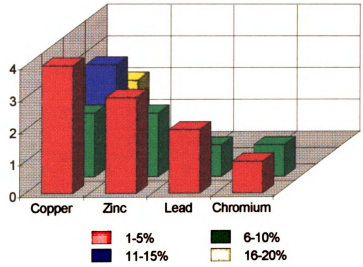

FIGURE 24

ESTIMATION OF SALES OF HEAVY METALS

TABLE XXX

REPLACEMENT OF PETROLEUM OIL INKS BY SOYBEAN OIL INKS IN THE PRESENT

	Level	Frequency
No	1	15
Some	2	7
Already	3	2
Sum		24
Mean=	1.46	

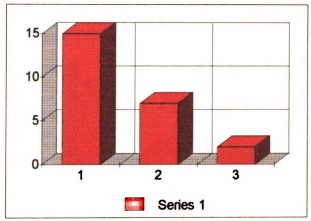

FIGURE 25

REPLACEMENT OF PETROLEUM OIL INKS BY SOYBEAN OIL INKS IN THE PRESENT

TABLE XXX

REPLACEMENT OF PETROLEUM OIL [illegible]
BY SOYBEAN OIL. INKS IN THE [illegible]

[illegible]	
No	
Some	
Almost	
Sun	

Mean

REPLACEMENT [illegible]
BY SOYBEAN [illegible]

TABLE XXXI

REPLACEMENT OF PETROLEUM OIL INKS BY SOYBEAN OIL INKS IN THE FUTURE

	Level	Frequency
No	1	16
Some	2	4
All	3	1
Sum		21
Mean=	1.29	

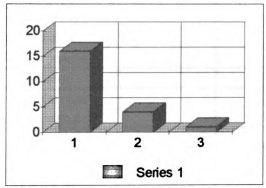

FIGURE 26

REPLACEMENT OF PETROLEUM OIL INKS BY SOYBEAN OIL INKS IN THE FUTURE

TABLE XXXII

AWARENESS OF TESTS AND REGULATIONS RELATED TO USE OF HEAVY METAL IN THE INK INDUSTRY

Regulations	Frequency	Percent
Resources Conservation and Recovery Act	17	53.1%
Clean Water Act	19	59.4%
Toxic Characteristic Leaching Procedure	17	53.1%
State Legislation	15	46.9%
American National Standards Institute	8	25.0%
American Society for Testing and Materials	9	28.1%
Lead Containing Paint	8	25.0%
Net Respondents	32	100.0%

CHAPTER 13

SUMMARY AND CONCLUSIONS

The Model Toxics in Packaging Legislation as developed by the Coalition of Northeastern Governors (CONEG) found and declared that:

1. " The management of solid waste can pose a wide range of hazards to public health and safety and to the environment."
2. " Packaging comprises a significant percentage of the overall solid waste stream."
3. Heavy metals included in packaging are likely to be present in emissions or ash when packaging is incinerated, or in leachate when packaging is landfilled.
4. " Lead, mercury, cadmium and hexavalent chromium, on the basis of available scientific and medical evidence, are of particular concern."
5. " It is desirable as a first step in reducing the toxicity of packaging waste to eliminate the addition of these heavy metals to packaging."

The following summary and conclusions have been made, based upon the five declarations in the Model Toxics in Packaging Legislation as develop by CONEG. Each declaration will be analyzed to corroborate the veracity of the statements.

Declaration 1. Solid Waste as a Hazard to Health and Environment

Heavy metals in solid waste represent a hazard to health and environment for two reasons. First, solid waste containing heavy metals disposed in landfills represents an important threat to groundwater resources if landfills are improperly operated. Unfortunately, many landfills were (are) used improperly; illegal dumps of hazardous waste or insufficient disposal facilities are not unusual. Second, rapid urban and suburban development in the United States has caused many once remote dumping grounds to be close to developed areas which can pose a hazard to public health and environment.

Declaration 2. Packaging as a Significant Percent of Solid Waste Stream

Packaging comprises 33% percent of total sources of landfilled municipal solid waste. Two-thirds of the total municipal waste is metals (8.7 %); glass (8.2%); plastic (6.6%); and paper and paperboard (41%).

Declaration 3. Environmental Hazards of Heavy Metal Disposal

Copper

Sewage sludge contains an elevated level of copper, the majority coming from industrial discharges. This sludge is frequently applied to land or landfilled. Most of the copper in soil is apparently tightly bound to soil components and may not be accessible for uptake by plants when sludge is applied to soil and it does not leach significantly from soil to groundwater when sludge is landfilled. However, generation of airborne copper from

waste sites is twice as high as from agricultural control areas. Sludge dumped in waste sites is increasing the level of copper in air that may reach excessive levels.

Chromium

Chromium-containing wastes have been disposed by discharging them to surface impoundments or lagoons. Leakage from these lagoons into groundwater has been relatively common. Almost all reported incidences of chromium related groundwater contamination are of industrial origin.

When sludge containing chromium (VI) was applied to land, it was found relatively low amounts of chromium (VI) were found in food plants. Cr (VI) does not appear to accumulate in mammalian systems. Therefore, bioaccumulation in the soil-plant-animal system does not appear to be a significant exposure source.

Cadmium

Cadmium based inks in packaging application are a concern due to the major packaging related industries that dump large amounts of sewage sludge and wastes containing cadmium. These include pulp and paper mills, rubber processing, and paint and ink plants.

Cadmium is used as a pigment and a stabilizer in the polymeric industry. When cadmium containing plastics are incinerated, the levels of cadmium in air are increased. Exposure to this level in air is predicted to increase human cancer risk in urban or industrial areas.

Cadmium from sludge applied to land is transmitted to the food chain in considerable quantities. Cadmium accumulates mainly in certain kinds of plants such as food crops, root crops, leafy vegetables and tobacco plants. Food containing elevated

levels of cadmium is expected to increase in the long-term the daily intake of cadmium from 20-40 ug/day to the World Health Organization's allowable range limit of 51-71 ug/day.

When waste containing cadmium is landfilled, considerable leaching of cadmium from soil to groundwater can occur only in cases of extreme contamination, low pH or both.

Mercury

Mercury is used as a catalyst in the synthesis of vinyl chloride and vinyl acetate, and is used in the production of urethane and urethane resins. Recover of mercury from mercurial catalysts is not economical; mercury is being effectively replaced by other materials.

Mercury migration from landfills to groundwater happens only in poorly operated landfill sites. However, it was found that ground water and surface-water from 25% of waste sites in the United States have an elevated mercury level.

Lead

Due to its long half-life in soil, lead is, in general, immobile. As a result, the concentration of lead in water supplies is low. Lead in edible plants is unaffected by increasing lead content in soil. Therefore, lead content in foodstuffs is limited.

Exposure to environmental lead concentration can put human health at risk. 7 ug of lead/l in human blood is enough to produce chronic symptoms in humans. Such concentration is close to the 2.3 ug/l that is the present average concentration of lead in human blood. Therefore, it is necessary to keep environmental lead levels to a minimum.

Declaration 4. Heavy Metal Exposure as a Health Hazard

Copper

If workers are subjected to excess concentrations of the metal in any of its forms, unwanted health effects can result. Industrial exposure to copper dust or fumes has been frequent, but health surveys of workers engaged in the processing of copper have not revealed signs of chronic illness.

Exposure to copper dust can produce metal fume fever, respiratory effects, anemia and hemolytic effects, hepatic effects, dermal/ocular effects, gastrointestinal effects, neurological effects, reproductive effects and cardiovascular effects.

Chromium

Cr (VI) has a high penetration power and can be absorbed through the skin, lungs, or intestinal tract. Due to Cr (VI) wide solubility, chromates of calcium, strontium, and zinc; chromium trioxide; and sodium dichromate are responsible for a wide variety of types of cancers, primarily lung cancer in workers who were exposed to high levels of zinc chromate in workroom air, such as in pigment production plants. Zinc chromate is a more potent human carcinogen by inhalation than are other Cr (VI) compounds.

Chromium is the second most common skin allergen, the most common sensitizer, and the most important cause of occupational dermatitis. Chromosomal aberrations and sister chromatid exchanges in lymphocytes of workers exposed to Cr (VI) occur.

Cadmium

Cadmium is a very widely known potent occupational metallic toxicant for an ample range of tissues and organ systems, whose toxicity depends on the concentration in the organ. Cadmium effects on different organ system include damage to the tissues of the reproductive system, induction of testicular interstitial cell cancer, structural and functional damage to the liver, kidney, lung and nervous system and osteoporosis and osteomalacia.

Cadmium once entering the human body stays in the body for a long period, having a half-life of 10 to 30 years. For such reason, concentration of this metal in humans has already increased resulting from accumulation of cadmium from daily intake. If cadmium emissions continue, cadmium in the environment will reach a dangerous level.

Mercury

Inorganic mercury is rapidly lost from the body. Therefore, only in the case of recurrent exposures and toxicity, do manifestations of mercurialism become chronic. Exposure effects may disappear if the worker is removed from exposure for a period of time.

The central nervous system and the kidney are the two major organs affected by mercury exposure. In low doses, mercury may induce renal and liver disease and occasionally a tremor is noted. In the workplace, chronic mercury vapor intoxication has resulted in mental disturbances, objective tremors, and gum diseases at average air concentrations less than 100-200 ug/m^3.

Lead

Inhalation of dust and fine powder is the major occupational risk of lead. Direct contact with such powder should be avoided and it is also necessary to wash any clothes used at work and shower before returning home to avoid exposure of children to high levels of lead.

Exposure to lead can cause neurotoxicity, reproductive effects, renal effects, cancer, gastrointestinal effects, cardiovascular effects, mutagenic effects, and hematological effects.

Declaration 5. Heavy Metal Reduction

From the profile of the responses we can conclude the following:

- 28 out of 32 ink industries limit heavy metal (cadmium, chromium (VI), mercury and lead) based ink, and 12 out of 32 limit to less than 100 ppm due to CONEG types of regulations.
- The major sources of limitation for heavy metals are the CONEG regulation and customers.
- Customers in the packaging industry ask more frequently for certificates of compliance with CONEG types of regulations than non- packaging industries.
- 59% of the respondents said cost were affected by the reduction of heavy metal content in their inks. These industries increased their costs by an average of 9 %.
- 38% of the respondents prices were affected by the reduction of heavy metal content in their inks. These industries increased their prices by an average of 5 %.

- Only seven companies (22%) were increased their costs due to reduction of heavy metal content, but, their prices were not affected. Twelve companies (38%) mentioned that their costs were increased, as a consequence, their prices were increased but not in the same proportion.
- One response to CONEG regulations was replacement of heavy metal based pigments used in the packaging industries. Many ink companies have almost eliminated the use of such pigments in their inks, or will eliminate them in 5 to 20 years.
- Substitutes such as hansa yellow, DNA orange, diarylide, benzimidazolone, titanium dioxide, dianisidine and AZO reds are used widely because they comply with current legislation.
- In the industry, the use of phthalocyanine ink, blue and green is in question. Non-copper based phthalo blue is used as a substitute for phthalo blue.

Recommendations for Future Work

I have three recommendations for future work. First, conduct a research to evaluate the content of heavy metals in post-consumer materials using analytical methods that measure soluble or leachable metals. The increase of heavy metal in recycled packaging material might increase the concentration of heavy metal in landfills and increase the risk of contamination of the contents.

A second recommendation will be to record heavy metal air content in different ink manufacturing industries and relate these to workers health conditions and ink powder manipulation procedures.

Finally, a specific analytical method for testing was not specified by the CONEG regulation, causing confusion between the ink industry and its suppliers. For this reason I recommend to evaluate the different types of tests used, and propose a standard heavy metal test to be used.

GLOSSARY

GLOSSARY

Abomasum:	The fourth or true digestive stomach of a ruminant
Adenocarcinoma:	Carcinoma derived from glandular tissue or in which the tumor cells form recognizable glandular structures.
Adenoma:	Benign epithelial tumor in which the cells are clearly derived from grandular ephitelium.
Adenosine triphospahteasa:	Nucleotide compound, occurring in all cells, where it represents energy storage in form of high-energy phosphate bonds.
Alveolus:	A general term used in anatomical nomenclature to designate a small saclike dilatation.
Aminoaciduria:	An excess of amino acids in the urine.
Angiosperms:	A plant of the class angiospermae. Plant that produce seeds enclosed in an ovary.
Anorexia:	Loss of appetite.
Anosmia:	Absence of the sense of smell.
Aorta:	The main trunk from which the systematic arterial system proceeds.
Atherosclerosis:	An extremely common form of arteriosclerosis in which deposits of yellowish plaques (atheromas) containing cholesterol, lipoid material, and lipophages are formed within the intima and inner media of large and medium-sized arteries.
Athetosis:	Atherosclerosis

* The source of the medical terms is Rosell, E. (1989). Human Medicine Dictionary. Orchard Publishers. New York.

Azotemia:	An excess of urea or other nitrogenous bodies in the blood.
Basophilic:	Staining readily with basic dyes.
Bronchospasm:	Spasmodic contraction of the smooth muscle of the bronchi, as occurs in asthma.
Cardiomegaly:	Cardiac hypertrophy
Coronary:	The term usually denotes the arteries that supply the heart muscle and, by extension, a pathologic involvement of them.
Corticosteroid :	Steroids produced by the adrenal cortex in response to the release of corticotropin by the pituitary gland. Influence carbohydrate, fat and protein metabolism and regulate electrolyte and water balance.
Cortisol:	The major natural glucocorticoid produced by the human adrenal cortex.
Chemosensory:	Relating to the perception of chemical substances, as in odor detection.
Chlorosis:	Disorder generally affecting adolescent females, believed to be associated with iron deficiency anemia.
Chorionepithelioma:	An epithelial malignancy of trophoblastic cells.
Choroid :	The thin, pigmented, vascular coat of the eye extending from the ora serrata to the optic nerve.
Chorea:	The ceaseless occurrence of a wide variety of rapid, highly complex, jerky movements that appear to be well coordinated but are performed involuntarily.
Choreoathetosis:	A condition marked by choreic and athetoid movements.
Dermatitis:	Inflammation of the skin.
Dermatosis:	Any skin disease, especially one not characterized by inflammation.
Dimorphism:	The property of having or existing in two forms, as fungi that can grow as molds or yeasts.
Dysphagia:	Difficulty in swallowing.

Dyspnea:	Difficult or labored breathing.
Edema:	The presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body.
Emphysema:	A pathological accumulation of air in tissues or organs; applied especially to such a condition of the lungs.
EPA:	U.S. Environmental Protection Agency .
Epithelium:	The covering of internal and external surfaces of the body.
Erythrocyte:	One of the elements found in peripheral blood; called also red blood cell.
Etiology:	All the factors that contribute to the occurrence of a disease or abnormal condition.
Fasciculation:	A small local contraction of muscles, visible through the skin, representing a spontaneous discharge of a number of fibers innervated by a single motor nerve filament.
Glucosuria:	An excess of glucose in the urine
Granuloma:	A tumor-like mass or nodule of granulation tissue, with actively growing fibroblast capillary buds.
Histopathology:	A branch of pathology concerned with the tissue changes characteristic of disease. The tissue changes that affect a part or accompany a disease.
Hematocrit:	The volume percentage of erythrocytes in whole blood
Hematuria:	Blood in the urine
Hemoglobinuria:	The presence of free hemoglobin in the urine
Hemoglobin:	The oxygen-carrying pigment of the erythrocytes, formed by the developing erythrocyte in bone marrow.
Hemolysis:	The liberation of hemoglobin.
Hemolytic:	Producing hemolysis
Hemoptysis:	The expectoration of blood or of blood-stained sputum.

Hypercholesterolemia: Excess of cholesterol in the blood.

Hyperemia: An excess of blood in a part; engorgement.

Hyperuricemia: Excess of uric acid in the blood.

Hypomania: Mania of a moderate type.

Hypophosphatemia: An abnormally decreased amount of phosphates in the blood.

Hypophyseal: Pertaining to a hypophysis, especially to the hypophysis cerebri, or pituitary gland.

Hypophysis: The pituitary gland. An epithelial body located at the base of the brain in the sella turcica.

Leukocytosis: A transient increase in the number of leukocytes in the blood.

Lymphocyte: A mononuclear leukocyte.

Macrophage: Any of the large, mononuclear highly phagocytic cells in the human blood.

Mucous: Pertaining to mucus.

Myoclonus: Shock like contractions of a portion of a muscle, an entire muscle, or a group of muscles.

Hypoxia: Reduction of oxygen supply to tissue below physiological levels despite adequate perfusion of the tissue by blood.

Nasal septum: The partition separating the two nasal cavities in the midplane.

Necrosis: Morphological changes indicative of cell death.

Nephritis: Inflammation of the kidney

Nephropathy: Disease of the kidneys

NIOSH: National Institute for Occupational Safety and Health

Oliguria: Secretion of a diminished amount of urine in relation to the fluid intake.

OSHA:	Occupational Safety and Health Administration
Osmotic:	The flow or diffusion that takes place through a membrane of a living cell.
Osmoregulation:	Regulation of osmotic pressure in the body of a living organism.
Osteomalacia:	A condition marked by softening of the bones.
Osteoporosis:	Abnormal rarefraction of bone.
Papule:	A small circumscribed, superficial, solid elevation of the skin.
Paresthesia:	Sensibility for weight or pressure; pressure sense.
Pathognomonic:	Specially distinctive or characteristic of a disease or pathologic condition.
Pillar cells:	Elongated supporting cells in a double row.
Plastid:	Any of the specialized organelles of plant cells that contain pigments.
Pneumoconiosis:	A condition characterized by permanent deposition of substantial amounts of particulate matter in the lungs.
Pneumonitis:	Inflammation of the lungs.
Pneumothorax:	An accumulation of air or gas in the pleural space.
Proteinuria:	An excess of serum proteins in the urine.
Pleura:	The serous membrane investing the lungs.
Polyp:	A morbid excrescence from mucous membrane.
RCRA:	Resource Conservation and Recovery Act.
Receptor:	Specific molecule on the surface of a cell that responds in a specific way
Teratogenic:	Tending to produce anomalies of formation.
Teratoma:	A true neoplasma made up of a number of different types of tissue.

APPENDIX A

Appendix A:
Model Toxics Legislation

as developed by
The Source Reduction Council of
The Coalition of Northeastern Governors (CONEG)
December 14, 1989

Summary*

The legislation calls for the reduction of lead, mercury, cadmium and hexavalent chromium in packaging or packaging materials used or sold within the state.

Manufacturers and distributors would have two years to clear inventory and make necessary adjustments to their operations in order to comply with the law.

Manufacturers and distributors of packaging or packaging materials would be required to reduce the sum of the concentration levels of incidentally introduced lead, cadmium, mercury and hexavalent chromium to 600 parts per million two years after the legislation is signed into law; 250 parts per million three years after it is signed into law; and 100 parts per million four years after it is signed into law. The legislation prohibits the intentional introduction of the four heavy metals during manufacturing or distribution.

The legislation provides an exemption for packaging made from post-consumer materials; packages and packaging components manufactured prior to the effective date of the legislation; packaging that is essential to the protection, safe handling or function of the package's contents—for example, medical products related to radiation therapy, x-rays, etc.; packages and packaging components for which there is no feasible alternative; reusable packaging for products that are subject to other federal or state health, safety, transportation or disposal requirements (i.e., hazardous waste); and packaging having a controlled distribution and reuse (i.e., beverage containers subject to mandatory deposit requirements).

Manufacturers and suppliers of packaging and packaging components are required to furnish a certificate of compliance to the purchasers of packaging. (This applies to companies that actually put their products in the package and does not apply to the retailer or the individual consumer.) The public and the state have access to these certificates.

The legislation also provides for a review process by the state to determine the effectiveness of the act. More specifically, that review will address the need to continue the recycling exemption and will determine if other toxic substances contained in packaging should be subject to reduction.

* Revised January 1995.

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Section 1. (Title)

Section 2. The legislature finds and declares that:

a. The management of solid waste can pose a wide range of hazards to public health and safety and to the environment;

b. Packaging comprises a significant percentage of the overall solid waste stream;

c. The presence of heavy metals in packaging is a part of the total concern in light of their likely presence in emissions or ash when packaging is incinerated, or in leachate when packaging is landfilled;

d. Lead, mercury, cadmium and hexavalent chromium, on the basis of available scientific and medical evidence, are of particular concern;

e. It is desirable, as a first step in reducing the toxicity of packaging waste, to eliminate the addition of these heavy metals to packaging; and

f. The intent of this act is to achieve this reduction in toxicity without impeding or discouraging the expanded use of post-consumer materials in the production of packaging and its components.

Section 3. Definitions*

"Package": means a container providing a means of marketing, protecting or handling a product and shall include a unit package, an intermediate package and a shipping container as defined in ASTM D996. "Package" shall also mean and include such unsealed receptacles as carrying cases, crates, cups, pails, rigid foil and other trays, wrappers and wrapping films, bags and tubs.

"Distributor": means any person, firm or corporation who takes title to goods purchased for resale.

"Packaging Component": means any individual assembled part of a package such as, but not limited to, any interior or exterior blocking, bracing, cushioning, weatherproofing, exterior strapping, coatings, closures, inks and labels.

Tin-plated steel that meets the American Society for Testing and Materials (ASTM) specification A-623 shall be considered as a single package component.** Electro-galvanized coated steel and hot dipped coated galvanized steel that meet the American Society for Testing and Materials (ASTM) specifications A-525 and A-879 shall be treated in the same manner as tin-plated steel.

"Manufacturing": means physical or chemical modification of (a) material(s) to produce packaging or packaging components.

"Distribution": means the practice of taking title to (a) package(s) or packaging component(s) for promotional purposes or resale. Persons involved solely in delivering (a) package(s) or packaging component(s) on behalf of third parties are not considered distributors.

"Manufacturer": means any person, firm, association, partnership or corporation producing (a) package(s) or packaging components(s) as defined in this act.

Revised January 1995.

** Tin-plated steel, electro-galvanized steel and hot dipped coated galvanized steel language was inadvertently omitted from December 14, 1989, adoption of the model legislation. It was later adopted and added to the model legislation by the member states of the Toxics in Packaging Clearinghouse (TPCH) on April 21, 1993.

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"Supplier": means any person, firm, association, partnership or corporation who sells, offers for sale, or offers for promotional purposes packages or packaging components which shall be used by any other person, firm, association, partnership or corporation to package (a) product(s).

"Intentional Introduction": means the act of deliberately utilizing a regulated metal in the formation of a package or packaging component where its continued presence is desired in the final package or packaging component to provide a specific characteristic, appearance or quality.

The use of a regulated metal as a processing agent or intermediate to impart certain chemical or physical changes during manufacturing, whereupon the incidental retention of a residue of said metal in the final package or packaging component is neither desired nor deliberate, is not considered intentional introduction for the purposes of this act where said final package or packaging component is in compliance with subsection c of section 4 of this act.

The use of recycled material as feedstock for the manufacture of new packaging materials, where some portion of the recycled materials may contain amounts of the regulated metals, is not considered intentional introduction for the purposes of this act where the new package or packaging component is in compliance with subsection c of section 4 of this act.

"Intentional Presence": means the presence of a regulated metal as an unintended or undesired ingredient of a package or packaging component.

Section 4. Prohibition/Schedule for Removal of Incidental Amounts

a. As soon as feasible but not later than two years after the adoption of this act, no package or packaging component shall be offered for sale or for promotional purposes by its manufacturer or distributor in the state of _____, which includes, in the package itself or in any packaging component, inks, dyes, pigments, adhesives, stabilizers or any other additives, any lead, cadmium, mercury or hexavalent chromium which has been intentionally introduced as an element during manufacturing or distribution as opposed to the incidental presence of any of these elements.

b. As soon as feasible but not later than two years after the adoption of this act, no product shall be offered for sale or for promotional purposes by its manufacturer or distributor in the state of _____ in a package which includes, in the package itself or in any of its packaging components, inks, dyes, pigments, adhesives, stabilizers or any other additives, any lead, cadmium, mercury or hexavalent chromium which has been intentionally introduced as an element during manufacturing or distribution as opposed to the incidental presence of any of these elements.

c. The sum of the concentration levels of lead, cadmium, mercury and hexavalent chromium present in any package or packaging component shall not exceed the following:

- 600 parts per million by weight (0.06 percent) effective two years after adoption of this statute;
- 250 parts per million by weight (0.025 percent) effective three years after adoption of this statute; and
- 100 parts per million by weight (0.01 percent) effective four years after adoption of this statute.

Section 5. Exemptions*

All packages and packaging components shall be subject to this act except the following:

a. those packages or package components with a code indicating date of manufacture that were manufactured prior to the effective date of this statute;

b. those packages or packaging components to which lead, cadmium, mercury or hexavalent chromium have been added in the manufacturing, forming, printing or distribution process in order to comply with health or safety requirements of federal law, provided that the manufacturer of a package or packaging component must petition the [state administrative agency] for any exemption from the provisions of this subsection for a particular package or packaging component based upon either criterion; and provided further that the [state administrative agency] may grant an exemption for up to two years if warranted by the circumstances; and provided further that such an exemption may, upon reapplication for exemption and meeting the criteria of this subsection, be renewed at two year intervals; or

c. packages and packaging components that would not exceed the maximum contaminant levels set forth in subsection c of section 4 of this act but for the addition of post-consumer materials; and provided that the exemption for this subparagraph shall expire January 1, 2000; or

d. those packages or packaging components to which lead, cadmium, mercury or hexavalent chromium have been added in the manufacturing, forming, printing or distribution process for which there is no feasible alternative, provided that the manufacturer of a package or packaging component must petition the [state administrative agency] for any exemption from the provisions of this subsection for a particular package or packaging component based upon the criterion; and provided further that the [state administrative agency] may grant an exemption for up to two years if warranted by the circumstances; and provided further that such an exemption may, upon reapplication for exemption and meeting the criterion of this subsection, be renewed at two-year intervals. For purposes of this subsection, a use for which there is no feasible alternative is one in which the regulated substance is essential to the protection, safe handling or function of the package's contents; or

e. packages and packaging components that are reused but exceed contaminant levels set forth in subsection c of section 4 of this act, provided that the product being conveyed by such package and/or the package/packaging component is (are) regulated under federal and/or state health or safety requirements; and provided that transportation of such packaged product is regulated under federal and/or state transportation requirements; and provided that disposal of such package is performed according to federal and/or state radioactive or hazardous waste disposal requirements, and provided that an exemption under this subparagraph shall expire on January 1, 2000; or

f. packages and packaging components having a controlled distribution and reuse that exceed the contaminant levels set forth in subsection c of section 4 of this act, provided that the manufacturer or distributor of such packages or packaging components must petition the [state administrative agency] for exemption and receive approval from the [state administrative agency], working with the CONEG Toxics in Packaging Clearinghouse, according to standards in subsection f.1 below set by such agency and based upon satisfactory demonstrations that the environmental benefit of the controlled distribution and reuse is significantly greater as compared to the same package manufactured in compliance with the contaminant levels set forth in subsection c of section 4; and provided that an exemption under this subparagraph shall expire on January 1, 2000.

* Revised January 1995.

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1. Standards

A plan, to be proposed by the manufacturer seeking the exemption or his designee, shall include each of the following elements:

i. a means of identifying in a permanent and visible manner those reusable entities containing regulated metals for which an exemption is sought;

ii. a method of regulatory and financial accountability so that a specified percentage of such reusable entities manufactured and distributed to other persons are not discarded by those persons after use, but are returned to the manufacturer or his/her designee;

iii. a system of inventory and record maintenance to account for reusable entities placed in, and removed from, service;

iv. a means of transforming returned entities that are no longer reusable into recycled materials for manufacturing or into manufacturing wastes that are subject to existing federal and/or state laws or regulations governing such manufacturing waste to ensure that these wastes do not enter the commercial or municipal waste stream; and

v. a system of annually reporting to the [appropriate state administrative agency] changes to the system and changes in designees.

Section 6. Certificate of Compliance

As soon as feasible but not later than two years after the adoption of this act, a certificate of compliance stating that a package or packaging component is in compliance with the requirements of this act shall be furnished by its manufacturer or supplier to its purchaser, provided, however, where compliance is achieved under the exemption(s) provided in subsection 5 b or c, the certificate shall state the specific basis upon which the exemption is claimed. The certificate of compliance shall be signed by an authorized official of the manufacturing or supplying company. The purchaser shall retain the certificate of compliance for as long as the package or packaging component is in use. A copy of the certificate of compliance shall be kept on file by the manufacturer or supplier of the package or packaging component. Certificates of compliance, or copies thereof, shall be furnished to the [state administrative agency] upon its request and to members of the public in accordance with section 9.

If the manufacturer or supplier of the package or packaging component reformulates or creates a new package or packaging component, the manufacturer or supplier shall provide an amended or new certificate of compliance for the reformulated or new package or packaging component.

Section 7. Enforcement

[Each state to add its own enforcement provisions]

Section 8. State Review*

[The state administrative agency] shall, in consultation with the Source Reduction Council of CONEG, review the effectiveness of this act no later than 42 months after its adoption and shall provide a report based upon that review to the governor and legislature. The report may contain recommendations to add other toxic substances contained in packaging to the list set forth in this act in order to further reduce the toxicity of packaging waste, and a description of the nature of the substitutes used in lieu of lead, mercury, cadmium and hexavalent chromium.

* Revised January 1995.

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[The state administrative agency] shall, in consultation wth the Source Reduction Task Force of CONEG, review the extension of the recycling exemption as it is provided for in subsection c of section 5 of this act. This review shall commence no later than January 1, 1997. A report based upon that review shall be provided to the governor and legislature by January 1, 1999.

Section 9. Public Access

Any request from a member of the public for any certificate of compliance from the manufacturer or supplier of a package or packaging component shall be:

a. Made in writing with a copy provided to the [state administrative agency];

b. Made specific as to package or packaging component information requested; and

c. Responded to by the manufacturer or supplier within 60 days.

Section 10. Effective Date

This act shall become effective immediately upon adoption.

Section 11. Severability and Construction*

The provisions of this act shall be severable, and if any court declares any phrase, clause, sentence or provision of this act to be invalid, or its applicability to any government, agency, person or circumstance is declared invalid, the remainder of the act and its relevant applicability shall not be affected. The provisions of this act shall be liberally construed to give effect to the purposes thereof.

Source: Thompson Publishing Group. Environmental Packaging. U.S. Guide to Green Labeling, Packaging and Recycling. Whashington, D.C.

* Added January 1995.

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APPENDIX B

Survey Questionnaire

Heavy Metals in the Ink Industry

Carla M. Vidal
School of Packaging
Michigan State University
East Lansing, MI 48823

1. _____
Name

2. _____
Company Name

3. _____
Street No.

_____, _____ (_____)
City State Zip Code

4. Telephone Number _____ - _____
area number

5. Date _____

Note:
If you have any questions please call me at (517) 353-5143.

Questionnaire

This questionnaire is intended for people working in the development, manufacturing or research division of companies that manufacture inks for the packaging industry. It is completely voluntary and confidential. No company or individual names will be identified in survey reports. If you don't want to answer a question , just skip over it.

Instructions:
Place a X in the block provided that best describes your operation.

1.- Does your company use or manufacture inks for the packaging industry?

Yes	No

Continue Stop - Your company is not in the scope of my study. Thank you for your time. Please return the questionnaire in the envelope provided.

2.- Are you knowledgeable about the manufacturing, development or research operation of your company?

Yes	No

Continue Stop - Please direct the questionnaire to the appropriate person in your company.

3.- Which of these has your company used in ink formulation? (Mark all which apply)

Copper	Zinc	Lead	Cadmium	Chromium (VI)	Mercury
A	B	C	D	E	F

4.- We are interested in how **the model toxics in packaging legislation as developed by the Coalition of Northeastern Governors (CONEG) Source Reduction Council** is affecting the ink industry. Are you familiar with this legislation?

1.Yes	2.No

Go to question 7.

5.- Fourteen states in the U.S. have already passed this law. Has your state passed this legislation? If yes, when?

1. Yes	2. No	3. Don't know

Year _____ Go to question 7.

6.- Does your company limit the total quantity of Lead, Cadmium, Chromium (VI) and Mercury, in your inks because of toxics in packaging legislation ?

Yes,< 600 ppm	Yes,< 250 ppm	Yes,< 100ppm	No	don't Know
1	2	3	4	

7.- Does your company limit the amount of heavy metal in your inks?

1. Yes	2. No	3. Don't know

Go to question 9.

8.- Please indicate the quantity of the following heavy metals you allow in your inks and the source of the limitation (internal, CONEG, state legislation, ANSI, ASTM, consumer standard, etc) ?

Metal	Quantity in ppm	Source of limitation	No Limitation
Copper			
Zinc			
Lead			
Cadmium			
Chromium (IV)			
Mercury			
Total heavy metals (Lead + Cadmium+ Chromium (IV)+ Mercury)			

9.- How often do your customers in the packaging industry ask your company for a written certificate of compliance with toxics legislation?

1. Never
2. Sometimes
3. Usually
4. Frequently
5. Always

10.- How often do your customers in other industries (non-packaging), ask your company for a written certificate of compliance with the toxics legislation?

1. Never - Go to question 11.

2. Sometimes

3. Usually

4. Frequently

5. Always

Please list the major industries which ask for such certification.

_____

_____

_____

_____

11.- Which types of analytical methods do you generally use to determine heavy metal content in your inks? (Mark all which apply)

1. Batch-to-batch testing ☐

2. Testing of random samples ☐

3. Calculation of contamination levels ☐

4. Don't test ☐

5. Don't know ☐

6. Other methods (list): _____

_____

_____

_____

12.- Do you feel that an ASTM standard is needed to provide a unique testing method for heavy metal content for use by the ink industry?

1. Yes
2. No
3. Don't Know

13.- Generally speaking, do you think that reduction of heavy metal content has increased your costs?

1. Yes | 2. No | 3. Don't know

Go to question 17.

14.- By about what percent have your costs increased ?

1. <1% | 2. 1-5% | 3. 6-10% | 4. 11-15% | 5. > 15%

6. Don't Know

15.- Has the price of your products increased in response to that cost increase?

1. Yes | 2. No | 3. Don't know

Go to question 17.

16.- By about what percent have your prices increased ?

1. <1% | 2. 1-5% | 3. 6-10% | 4. 11-15% | 5. > 15%

6. Don't Know

17.- Have you reformulated any of your inks for the packaging industry to reduce or eliminate their heavy metal content?

1. None of them
2. Some of them
3. Most of them
4. All of them

18.- Have you reformulated any of your inks for other industries (non-packaging) to reduce or eliminate their heavy metal content?

1. None of them
2. Some of them
3. Most of them
4. All of them

19.- Please provide below as much detail as you can about ink reformulations and the reasons for reformulation. We have selected some major pigments and alternatives to simplify your task. Please check all which apply.

A-Cadmium red

Substitutes: Please check all which your company is using
___ AZO reds
___ Quinacridone perylene
___ Napthol
___ Disazo
___ Other(please specify_____)

Reason for reformulation: Please check all which apply
___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

B-Cadmium yellow

Substitutes: Please check all which your company is using
___ Benzimidazolone
___ Hansa yellow
___ Diarylide yellow
___ Other(please specify_____)

Continue on the next page

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

C-Cadmium orange

Substitutes: Please check all which your company is using

___ Exotic Oranges
___ Dianisidine Orange
___ DNA Orange
___ Other(please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

D-Lead-based yellows

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

E-Chromate yellow

Substitutes: Please check all which your company is using

___ Hansa yellow
___ Diarylide yellow
___ Benzimidazolone
___ Exotic yellow
___ Other (please specify_____)

Continue on the next page

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

F-Zinc chromate

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

G-Lead- Orange

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

H-Lead Molybdate or Molybdate Orange

Substitutes: Please check all which your company is using

___ Dianisidine orange
___ DNA exotic
___ Benzimidazolone (VAT orange)
___ Other (please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

Continue on the next page

I-Zinc

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

J-Copper

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

K-Bronze (zinc+copper)

Substitutes: Please check all which your company is using

___ Acrylac gold
___ Croda ink
___ MetalStar Alugold
___ Other (please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

Continue on the next page

L-Mercury

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

M-Others (please specify below; attach an extra page if needed)

_____ _____ _____

Substitutes:

_____ _____ _____
_____ _____ _____
_____ _____ _____

Reason for reformulation:
(Please check all which apply)

___Legislation	___Legislation	___Legislation
___Environmental concern	___Environmental concern	___Environmental concern
___Health concern	___Health concern	___Health concern
___Customer request	___Customer request	___Customer request
___Other (please specify _____)	___Other (please specify _____)	___Other (please specify _____)

20.- Do you anticipate having to replace heavy metal inks in the next 1, 5, 10 or 20 years?

Option\Year	1	5	10	20
Almost certain				
Probably will				
Not sure				
Probably won't				

Will not - Go to question 22

21.- Please provide as much detail as you can about the ink reformulations you foresee and the reasons for reformulation. We have selected some major pigments and alternatives to simplify your task. Please check all which apply.

A-Cadmium red

Substitutes: Please check all which your company is likely to use

___ AZO reds
___ Quinacridone perylene
___ Napthol
___ Disazo
___ Other(please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

B-Cadmium yellow

Substitutes: Please check all which your company is likely to use

___ Benzimidazolone
___ Hansa yellow
___ Diarylide yellow
___ Other(please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

C-Cadmium orange

Substitutes: Please check all which your company is likely to use

___ Exotic Oranges
___ Dianisidine Orange
___ DNA Orange
___ Other(please specify_____)

Continue on the next page

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

D-Lead-based yellows

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

E-Chromate yellow

Substitutes: Please check all which your company is likely to use

___ Hansa yellow
___ Diarylide yellow
___ Benzimidazolone
___ Exotic yellow
___ Other (please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

F-Zinc chromate

Substitutes: Please specify below

_____
_____
_____

Continue on the next page

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

G-Lead- Orange

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

H-Lead Molybdate or Molybdate Orange

Substitutes: Please check all which your company is likely to use

___ Dianisidine orange
___ DNA exotic
___ Benzimidazolone (VAT orange)
___ Other (please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

I-Zinc

Substitutes: Please specify below

_____
_____
_____

Continue on the next page

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

J-Copper

Substitutes: Please specify below

_____
_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

K-Bronze (zinc+copper)

Substitutes: Please check all which your company is likely to use

___ Acrylac gold
___ Croda ink
___ MetalStar Alugold
___ Other (please specify_____)

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

L-Mercury

Substitutes: Please specify below

_____
_____

Reason for reformulation: Please check all which apply

___ Legislation
___ Environmental concern
___ Health concern
___ Customer request
___ Other (please specify_____)

Continue on the next page

M-Others (please specify below; attach an extra page if needed)

_____ _____ _____

Substitutes:

_____ _____ _____
_____ _____ _____
_____ _____ _____

Reason for reformulation:
(Please check all which apply)

___Legislation	___Legislation	___Legislation
___Environmental concern	___Environmental concern	___Environmental concern
___Health concern	___Health concern	___Health concern
___Customer request	___Customer request	___Customer request
___Other (please specify _____)	___Other (please specify _____)	___Other (please specify _____)

22.- Please estimate in tonnage, dollar amount and/or percent of sales the quantity of your total production which is copper, zinc, lead, cadmium, mercury or hexavalent chromium related pigments/inks.

	Copper	Zinc	Lead	Cadmium	Chromium	Mercury
	A	B	C	D	E	F
Tonnage						
Dollars						
% sales						

23.- Do you use soybean oil as the vehicle to replace petroleum oil inks?

1. Already Have converted
2. Have converted some
3. Not sure
4. No

24.- Do you plan to use soybean oil as the vehicle to replace petroleum oil inks?

1. Will convert all
2. Will convert some
3. Not sure
4. No

25.- Are you aware of any other regulations that limit the use of heavy metals in the ink industry? (mark all that are relevant)

1.Resources Conservation and Recovery Act (RCRA)
2. Clean water Act (EPA)
3. Toxic characteristic Leaching procedure (TCLP) (EPA)
4. State Legislation
5. American National Standards Institute (ANSI)
6.American Society for Testing and Materials (ASTM)
7. Lead Containing Paint

Other _____

_____

_____

NOTE: **When you have completed the questionnaire please return it in the enclosed envelope. Your early response by February 03, 1995 will be appreciated. I appreciate your professional assistance. Thank you for your valuable help.**

APPENDIX C

SUMMARY OF SUBSTITUTES FOR HEAVY METALS IN THE INK INDUSTRY

Metals/substitutes	Cd Red	Cd yellow	Cd orange	Pb yellow	Cr yellow	Pb orange	Pb molybdate	Copper	Bronze
AZO Reds	X					X			
Quinoacridone	X					X			
Napthol	X					X			
Disazo	X								
Chronopthal	X								
Benzimidazolone		X			X		X		
Hansa Yellow		X		X	X				
Diarylide		X		X	X	X			
Titanium Dioxide		X		X	X	X			
Dianisidine			X			X	X		
DNA Orange			X			X	X		
Exotic Orange			X						
AAOT Yellow			X	X					
Monoarylide Yellow				X					
Exotic Yellow					X				
Cu Free Phtalo Blue								X	
Peacock Blue								X	
Cobalt Blue								X	
MetalStar ALugold									X
Croda Ink									X
Acrylac Gold									X
Toned Aluminium									X

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LIST OF REFERENCES

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