Health Consultation

EVALUATION OF ENVIRONMENTAL DATA CONTRACT PLATING

STRATFORD, FAIRFIELD COUNTY, CONNECTICUT

EPA FACILITY ID: CTD001180462

NOVEMBER 15, 2006

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES Public Health Service Agency for Toxic Substances and Disease Registry Division of Health Assessment and Consultation Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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HEALTH CONSULTATION

Evaluation of Environmental Data

CONTRACT PLATING

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Prepared By:

Connecticut Department of Public Health Under a Cooperative Agreement with the U.S. Department of Health and Human Services Agency for Toxic Substances and Disease Registry The conclusions and recommendations in this health consultation are based on the data and information made available to the Connecticut Department of Public Health (CTDPH) and the Agency for Toxic Substances and Disease Registry (ATDSR). CTDPH and ATSDR will review additional information when received. The review of additional data could change the conclusions and recommendations listed in this document.

Background and Statement of Issue

The Stratford Health Department (SHD) and the Connecticut Department of Environmental Protection (CT DEP) requested that the Connecticut Department of Public Health (CT DPH) review existing environmental data for the Contract Plating Company Site to determine if this site poses a threat to public health and whether actions are needed to protect public health. The US Environmental Protection Agency (EPA) provided CT DPH with all available environmental data for the site. The data consist of several rounds of soils testing results (surface and depth soils), collected in 1986, 1988, 1994 and 2002.

The Contract Plating Company site is located at 540 Longbrook Avenue in Stratford. The site occupies approximately 10 acres in a mixed residential/industrial area. It is bordered on the east by railroad tracks. Directly across the railroad tracks is the former Raymark Industries facility (Operable Unit 1 of the Raymark Superfund Site). To the north, the site is bordered by the Raybestos Memorial Ballfield (Operable Unit 4 of the Raymark Superfund Site). To the west and south are residences. The closest residence is about 700 feet northwest of the site (ATSDR 1994). A site plan of the Contact Plating property is provided in Attachment A.

Beginning in 1936, Contract Plating provided metal finishing services to manufacturers, including electroplating, anodizing (finishing to improve corrosion resistance), black oxidizing, phosphating and lacquering processes. Contract Plating ceased its metal finishing operations in May 1995. Since that time, the site has not been used for industrial activities.

Buildings onsite consist of two process buildings, a boiler building and a chemical storage building. The larger of the two process buildings was used for electroplating and the smaller of the two was used for anodizing processes. Beginning in 1936 and continuing until 1976, Contract Plating's neutralized process plating waste sludge was pumped from the site wastewater treatment system to two on-site, unlined lagoons (Lagoons A and B). An old site diagram depicting the location of the former lagoons is included in Attachment A. From 1976 until 1985, process plating wastes were discharged into four sand-bed sludge drying impoundments. An underlying drain pipe collected leachate from these drying impoundments which discharged into Long Brook, which is located approximately 3,000 feet northwest of the site. De-watered sludge was periodically removed from the drying beds and stored on-site in an asphalt-lined storage pit adjacent to the drying beds for shipment and disposal offsite (EPA 1994). In September 1990, the drying beds were capped (closed) according to regulations under the Resource Conservation and Recovery Act (RCRA) (personal communication, EPA, 2006).

The property owner has recently hired at least one worker to assist her with various activities on site including consolidating brush and other debris on the property and improving the landscaping and appearance of the front of the property.

In 1994, ATSDR prepared a Record of Activity, which evaluated surface soil data collected by EPA from areas near the former lagoons and from areas within the lagoons (ATSDR 1994). ATSDR concluded that several metals (chromium, nickel and cadmium) are present at levels of public health concern. ATSDR also noted that access to the site is not fully restricted and there is evidence of trespassing. In the 1994 Record of Activity, ATSDR recommended that exposure to the contaminated soils should be prevented and if site conditions change from industrial, the site should be re-evaluated.

Site Visit

Representatives from CT DPH, ATSDR, EPA and CT DEP met with the property owner on February 28, 2006, for a site visit. Upon arrival at the site, CT DPH and ATSDR staff drove from the street into the parking lot in front of the large main building (southwest portion of the site). The property owner informed us that when no one is present onsite, they place a chain across the entrance to the parking lot to prevent vehicles from accessing the site. This chain has been used for approximately the past 6 months. Prior to that, there was nothing to restrict vehicles from driving into the front parking lot during off hours. The chain does restrict vehicles but would not prevent individuals from walking onto the site from the street. The large main process building is vacant; access to the building is possible from the parking lot (southern, or front side). There are many broken windows and large portions of the roof have collapsed. A walkthrough of the interior of this building showed numerous signs of vandalism and trespassing (graffiti on walls, beer cans, golf ball). Pipes near the ceiling of the building were wrapped in material that may be asbestos. Most equipment and machinery has been removed. No bulk chemicals were observed. Numerous physical/ safety hazards were observed: debris falling from the roof, broken windows, broken glass on the floor, piles of debris on the floor. Photographs in Attachment B show the exterior and interior of the main process building.

Access to the northern portion of the site is secured by a locked gated fence that can be opened to allow vehicular traffic. A walkthrough of the other process building (anodizing building) was also conducted. Process chemicals, equipment and machinery have been removed from this building and it is now being used for storage of tires and automotive tools. An open pit (approximately 5 feet deep) containing an old metering system was observed just outside the former anodizing building. This pit is a physical/safety hazard as it did not have an adequate cover to prevent someone from falling in it. Bulk chemicals and equipment have been removed from the former chemical storage building (located next to the former anodizing building). Most of the area around these buildings is paved, but there are also portions of bare ground and weedy grasses. In this area, a variety of sporting equipment and toys was observed (all-terrain vehicle, bicycle, radio-controlled toy truck, basketball hoop, soccer balls, baseball and boxing equipment. It is unknown whether this equipment is currently used at the site. Photographs in Attachment C document these observations.

The site visit also included a walkthrough of the land behind the buildings (the northern portion of the property). Former lagoons A and B and the former drying impoundments are located in

this area. The former drying impoundments are located to the north of the main process building and are currently covered with a RCRA-approved cap and asphalt pavement. Lagoons A and B are visible as depressions in the sandy soil further to the north. No sludge or other liquids are visible in the lagoons. We observed numerous tire tracks in the former lagoon area as well as brake parts (brake pads and gasket material). The property owner stated that she prohibits anyone she allows onsite from driving in the former lagoon area. However, the property owner discovered (and disclosed to us the day after the site visit) that a visitor allowed on the property by the owner had recently driven an all-terrain vehicle (ATV) in the former lagoon area. We also observed a serious physical hazard in the lagoon area, consisting of an open cement catch basin approximately three feet wide by four feet long by five feet deep. Soil in the lagoon area was sandy and very dry. Moderate winds on the day of the site visit produced noticeable dust in the air. Attachment D contains photographs of the former lagoon area. During the walkthrough we observed several areas near the lagoon areas where piles of brush, soil and construction-type debris had been placed. There were also tire tracks visible. The property owner stated that she had recently hired someone to help her improve the appearance of the property to discourage trespassing and to ready the property for possible commercial use. She emphasized that she did not allow any activities to occur in the former lagoon areas.

The property is enclosed by a chain link fence which extends along the property boundary next to the railroad tracks on the eastern side, and along the northern and western boundaries, to the locked gate. During the site visit, we observed several large breaches of the fence and signs of trespassing activity including beer cans, burned wood that may be from a campfire, and a deteriorated sleeping bag. Photographs documenting these observations are in Attachment E.

Demographics

Within one mile of the Contract Plating site, it is estimated that there is a total population of 14,061, with approximately 1,300 children aged 6 years and younger. A map and summary of demographic statistics is provided in Attachment F.

Discussion

This section presents the environmental data reviewed for this health consultation. EPA provided CT DPH with environmental site data to be reviewed. Environmental data are presented and discussed along with relevant health-based comparison values. Comparison values are screening levels, below which, there is little likelihood of adverse health effects from exposure. When contaminant concentrations are below comparison values, no further evaluation for human health is necessary and it can be concluded that adverse health impacts are not likely. When contaminant concentrations exceed comparison values, it indicates that further evaluation of exposures and health impacts is needed. The comparison values used in this health consultation are from the CT Remediation Standard Regulations (CT RSRs) residential direct exposure criteria for soil (CT DEP 1996). Comparison values are presented in the data tables in the Environmental Data section.

This section also evaluates the likely exposure pathways for the site. Exposure pathways are ways people could come into contact with contamination. For each exposure pathway, the public health implications from exposure to hazards at the site are discussed in this section.

Environmental Data

The data that EPA provided to CT DPH for review consist of soil (and in some cases, lagoon sludge) data from four sampling events at the Contract Plating site. Soil and lagoon sludge samples (surface and depth) were collected in 1986 and 1988 as part of the RCRA Facility assessment process (EPA 1994). In 1994, Roy F. Weston, under contract to EPA, conducted surface soil sampling in and around the former lagoon area as part of an EPA Removal Program Preliminary Assessment/Site Investigation (EPA 1994). In 2002, Tetra Tech NUS, Inc., under contract to EPA, collected soil samples from locations across the entire site as part of the Raymark Industries Superfund Site investigation. Soil sampling results from these four events comprise the environmental data that was reviewed for this health consultation. Soils were analyzed for metals, asbestos, polychlorinated biphenyls (PCBs), dioxins, volatile organics and semi-volatile organics. However, not every soil sample was analyzed for all of these constituents.

Samples were taken from a total of 40 locations across the site, during the four sampling events, combined. There are significantly more samples in the northern portion of the site (in and around the area where the former lagoons are located) than elsewhere on the site. For example, there are very limited soil sampling data from unpaved areas around the chemical storage and anodizing buildings and there are no surface soil samples taken anywhere except the northern portion of the site. A few depth samples were taken from the area in front of the main building (southeastern corner of the property) but contamination was not detected at elevated levels in these samples. Surface soil samples were taken from 0-0.3 feet below ground surface and 0-0.5 feet below ground surface. Depth samples were collected as deep as 14 feet below ground surface.

At one time, the Contract Plating site was considered part of the Raymark Superfund Site (Operable Unit 6 – Commercial Properties). EPA removed the Contract Plating parcel from the Raymark Superfund site several years ago when a review of the environmental sampling indicated that Raymark waste was not present on the site¹.

Soil results from the Contract Plating site show that a variety of metals, as well as PCBs are present at levels above Connecticut's cleanup standards for soil (CT Residential Direct Exposure Criteria, Remediation Standards Regulations; CT RDECs). These standards were developed to be protective of frequent, long-term contact with soil by children and adults. Table 1 on page 5 provides a summary of soil results for surface and subsurface soils.

As shown in Table 1, chromium, cadmium, nickel, and cyanide were detected at the highest concentrations, relative to comparison values. The highest concentrations of nearly all of the metals were found in soil/sludge samples taken from the 0-5 and 0-6 foot depth intervals in the former lagoon area. These samples were collected in 1986 and 2002 as part of the RCRA Facility assessment and were collected as composite samples from the ground surface to the bottom of the lagoon. Concentrations of metals ranged from just slightly above the comparison

¹ EPA defined Raymark waste as material containing two of three compounds indicative of Raymark materials (lead, polychlorinated biphenyls [PCBs], asbestos) at concentrations exceeding levels of concerns for those compounds (400 mg/kg, 1 mg/kg, 1%, respectively) (Stratford 1994).

value to as great as 280 times greater then the comparison value. Surface soil samples collected in 1994 were also collected from the lagoon area. Many of these samples had metals present at levels exceeding comparison values. Contaminants appear to be fairly evenly distributed in the lagoon area. Chromium was reported as total chromium and was not speciated. Therefore, Table 1 compares chromium concentrations at the site with both the trivalent and hexavalent chromium comparison values.

1900,	1994, 2002.	1						
Contaminant	Sampling	Concentration	Comparison Value	Comparison Value				
	Date(s)	Range (mg/kg)	Exceedances/Total	(mg/kg) ^{&}				
			Samples					
SURFACE SOILS								
(0 - 0.3 feet and 0 - 0.5 feet below ground surface)								
Total PCBs	1994	0.53 - 3.6	5/8	1.0				
Aroclor 1254	1994	ND – 1.5	1/8	1.0 (total PCBs)				
Aroclor 1260	1994	0.13 – 2.4	4/8	1.0 (total PCBs)				
Cadmium	1986, 1994	4.8 - 1,580	10/11	34				
Chromium	1986, 1994	100 - 17,200	3/11	Trivalent = 3900				
(unspeciated)		100 - 17,200	10/11	Hexavalent = 100				
Cyanide	1986, 1994	12 - 4,323	4/11	1,400				
Nickel	1986, 1994	58 - 25,000	5/11	1,400				
Selenium	1986, 1994	ND – 1640	3/11	340				
Zinc	1986, 1994	140 - 169,000	4/11	20,000				
SURFACE/SUBSURFACE SOILS								
(0-2 feet, 0-5 feet and 0-6 feet below ground surface composite samples)								
Lead	1986, 2002	ND - 8,400	3/17	400				
Antimony	1986, 2002	ND - 50	2/3	27				
Arsenic	1986, 2002	ND - 49	2/3	10				
Cadmium	1986, 2002	11.8 - 1700	2/3	34				
Chromium	1986, 2002	38 - 28,000	2/3	Trivalent = 3900				
(unspeciated)		38 - 28,000	2/3	Hexavalent = 100				
Cyanide	1986, 2002	6,620 - 27,700	2/2	1,400				
Nickel	1986	53.7 - 22,000	2/3	1,400				
Zinc	1986	191 - 280,000	2/3	20,000				
SUBSURFACE SOILS								
(greater than 0.5 feet below ground surface composite samples)								
Cadmium	1988, 2002	ND - 120	2/24	34				
Chromium	1988, 2002	2.9 - 1900	0/24	Trivalent = 3900				
(unspeciated)			2/24	Hexavalent = 100				
Nickel	1988, 2002	3.6 - 4200	1/24	1,400				

Table 1:Summary of Soil Sampling Results for Contaminants Exceeding Comparison Values,
Contract Plating, 540 Longbrook Avenue, Stratford, CT. Samples Collected in 1986,
1988, 1994, 2002.

[&] The source for comparison values is the Connecticut Remediation Standard Regulations residential direct exposure criteria for soil (CT RSRs) (CT DEP 1996). These soil standards are developed to be protective of children and adults who contact soil on a daily basis for many years.

Approximately 66 soil samples (28 different locations) were analyzed for asbestos but none of them exceeded $1\%^2$. The most recent asbestos sampling occurred in 2002. As noted in the Site

²Neither ATSDR nor Connecticut has a cleanup standard for asbestos in soil. However, 1% is an asbestos cleanup level for soil used in Stratford for the Raymark Superfund site. ATSDR reviewed the cleanup standard and concurred with it (EPA 1994).

Visit Section, numerous pieces of brake material (pads and gaskets), potentially containing asbestos, were observed in the sandy soil throughout the northern portion of the site.

Exposure Pathway Analysis

To evaluate potential exposures to contaminants at the Contract Plating site, CT DPH considered the available environmental data and how people might come into contact with contaminants. In order for exposure to occur, there must be a source of hazardous contaminants, a way for people to come into direct contact with the contaminants, and a way for the contaminants to enter the body. It is important to emphasize that if there is no exposure to a hazardous contaminant, there is no risk of adverse health effects.

As previously stated, contaminants have been found in surface and subsurface soil at the Contract Plating site. Environmental data for other media such as air or groundwater were not reviewed as part of this health consultation (no such data were available for review). However, it is known that there are no drinking water wells on the property or in the nearby area. In addition, there are physical/safety hazards on the property that were observed during the site visit.

There is evidence of trespasser access on the site that was observed during the site visit. The presence of a sleeping bag and remnants of a possible campfire suggests that a trespasser (perhaps a homeless person) may have been using the site for a prolonged period of time. At the time of the site visit, there had been recent ATV use in the lagoon area. Driving an ATV in the lagoon area could result in significant exposure to soil, particularly through inhalation. Another activity that could result in soil exposure is the work being done to consolidate brush and debris and improve the landscaping and appearance of the property. At the time of the site visit, the property owner stated that an individual she hired is doing the landscaping/clearing work in many areas of the site *except* the lagoon areas. In addition, there may be adults and children allowed on the site by the owner (friends/family members of the owner and workers employed by the owner) who could come into contact with soil around the existing buildings. The presence of toys and sporting equipment near the former anodizing and chemical storage buildings indicates that children or adults may use the area for play or sports activities. It is not likely that such individuals would be exposed on a regular basis to contamination in the lagoon areas because the owner stated that she does not permit people to walk or drive in the lagoon areas. Toys and sporting equipment were not observed in the lagoon area.

To summarize, CT DPH has identified four receptor groups who could be exposed to contamination at the Contract Plating site:

- teen/adult trespasser,
- a teen/adult ATV driver,
- onsite adult worker, and
- child/adult visitor.

These receptors could be exposed to contaminated surface soil at the site through activities such as walking, sitting, camping, gathering wood, running, playing, ATV use, and moving soil and other debris around the property. Exposure could occur by ingestion (eating soil particles adhered to fingers or food items), dermal contact (skin contact with soil) and inhalation (inhaling windblown soil particles). Exposure to contaminants in surface soil is considered to be a

complete exposure pathway and is evaluated in greater detail in the Public Health Implications section.

Contamination was also found in subsurface soil at the Contract Plating site. With the exception of the ATV driver, the activities mentioned above are unlikely to cause exposure to soils at depth because such activities do not involve digging or disruption of soils at depth. During the site visit, evidence of digging was not observed. CT DPH considers the potential for exposure to subsurface contaminants to be very low (except for the ATV driver). Exposure to contaminants in subsurface soil is considered to be a *complete exposure pathway* only for the ATV driver.

During the site visit, physical/safety hazards were observed inside the main building, outside the former anodizing building and in the former lagoon area. Trespassers coming on the site could easily come into contact with these physical/safety hazards. There is evidence that trespassers have entered the main building (graffiti on walls, beer cans, golf ball). It is not known whether this trespassing activity is recent or past. Debris falling from the roof of the main building could injure a person trespassing inside who is not wearing a hard hat. Broken glass and other piles of debris on the floors could injure a trespasser while walking or playing inside the building. As noted previously, deteriorating pipe insulation that could contain asbestos was observed near the ceiling. It is not known whether there are asbestos fibers in the air inside the building at levels of concern.

Other physical/safety hazards on the site consist of open pits without adequate cover or warning signage. Again, since there is evidence that trespassers are accessing the site, a trespasser could become injured by falling into one of these pits.

It is less likely that persons allowed to be on the site (friends/family members of the owner and workers employed by the owner) would become harmed by the physical/safety hazards onsite because such individuals presumably would not enter the main building without a hard hat, and would be informed about the outside physical hazards on the property.

If there is no potential for exposure to contaminants, then it can be concluded that there is no possibility of adverse health effects from the contaminants. However, if there is an actual (completed), or potential exposure pathway, contaminant concentrations are compared to health-protective comparison values. As stated previously, the comparison values used in this health consultation are the CT RSRs residential direct exposure criteria for soil. If contaminant concentrations exceed comparison values, exposures are evaluated further. Table 1 summarizes the data for contaminants that were detected at concentrations exceeding comparison values.

Public Health Implications for Adults and Children

This section presents the likely health impacts from exposure to soil at the Contract Plating site. Whether a person may become sick from exposure to hazardous contamination depends on a number of factors including:

- the concentration of the chemical someone is exposed to (how much),
- the duration and frequency of exposure (how long, how many times),
- the route of exposure (breathing, eating/drinking, skin contact), and
- the person's individual characteristics (age, diet, lifestyle, genetics).

To evaluate public health implications to adults and children from contamination in the soil at the Contract Plating site, CT DPH evaluated contaminants present at levels above comparison values to determine the likelihood that the exposures would be significant enough to cause health effects. As mentioned in the previous section, four receptor groups were identified. However, risks to one of the receptor groups (child/adult visitor) were *not* evaluated because there is no surface soil data from areas of the site where they would likely be exposed (immediate vicinity of the main building and former anodizing and chemical storage buildings). Ingestion and dermal exposures were evaluated for the trespasser, ATV driver, and onsite worker. Inhalation exposures were evaluated only for the ATV driver. The rationale for this is that ATV use has the potential to create large amounts of dust in the air.

CT DPH evaluated potential cancer and noncancer health effects from exposure to the contaminants present in soil at concentrations exceeding health-based comparison values. Short-term (acute) exposures were evaluated where appropriate. In cases where a contaminant has not been shown to be carcinogenic, only noncancer risks were evaluated. Exposure to contaminants present in soils at depth were evaluated only for the ATV driver because the use of such a vehicle could cause soils at depth to become disturbed. Exposure to lead was evaluated only for the ATV driver because lead was found at concentrations exceeding comparison values in subsurface soils, not surface soil. Lead was evaluated using ATSDR's lead screening procedure (ATSDR 1999). This procedure uses a blood lead slope factor which predicts the increase in blood lead per unit lead concentration in soil. CT DPH used the adult factor of 0.001 which is based on a study of U.S. males aged 18-65 years. Summaries of general toxicological information for each of the contaminants evaluated in this health consultation are included in Attachment G.

For the risk calculations, CT DPH assumed that people are exposed to average contaminant concentrations in soil, as estimated by the 95% Upper Confidence Limit (UCL). The 95% UCL accounts for variability in the data and ensures that the average is not underestimated. Because metal speciation was not done, it was assumed that all of a particular metal measured in soil was present in the form that is most toxic. This is a very conservative (health protective) assumption.

CT DPH calculated exposure doses and theoretical risks to a teen/adult trespasser and an adult onsite worker from exposure to cadmium, chromium, cyanide, nickel, selenium, zinc and PCBs in surface soils. For the ATV driver, risks were calculated based on exposure to the above mentioned contaminants, plus lead, arsenic and antimony (because these contaminants were present in subsurface soils). As stated previously, risks to a child/adult visitor were not evaluated because there is no surface soil data from areas of the site where they would likely be exposed (i.e. in the vicinity of the buildings onsite).

CT DPH assumed that the trespasser is exposed to soil 4 days per week, for 9 months of the year through ingestion and dermal contact with soil. It is assumed that for 3 months of the year, soil contact would not occur because the ground is likely to be frozen and possibly snow-covered. CT DPH used 4 days per week rather than the typical 2 days per week assumption for trespassers because of observations made during the site visit (evidence that the trespasser may have been a homeless person who might have more frequent exposure). For the ATV driver, it was assumed

that an ATV is operated in the lagoon area 1 day per week, for 9 months per year. For purposes of evaluating inhalation exposures, the duration of each event was assumed to be 4 hours. CT DPH assumed that the onsite worker is potentially exposed to soil 5 days per week for 9 months per year.

To evaluate exposures via dermal contact with the soil, CT DPH used oral toxicity factors without making any adjustments to represent absorbed dose (rather than administered dose). EPA recommends making such an adjustment for several inorganic compounds (including cadmium, chromium, and nickel, which are present at Contract Plating), because these compounds are poorly absorbed across the gastrointestinal (GI) tract.³ Not making an adjustment can lead to an underestimation in the risks from the dermal exposure pathway. CT DPH chose not to make the toxicity factor adjustment because the risk assessment already includes some very conservative assumptions (namely that all of a particular metal measured in soil was present in the form that is most toxic). This is discussed further in the Uncertainty Section.

Selected exposure assumptions and theoretical risks for each exposure scenario are provided in Table 2. Attachment H provides detailed risk calculations.

As shown in Table 2, theoretical excess lifetime cancer risks for the teen trespasser and onsite worker are, respectively, 5 excess cancers in one million exposed persons (5 x 10^{-6}) and 2 excess cancers in 100,000 exposed people (2 x 10^{-5}). These risks represent an insignificant cancer risk and are not meaningful with respect to the likelihood of individuals exposed at the Contract Plating site actually experiencing cancer effects. For the ATV driver, cumulative cancer risks are 2 x 10^{-4} (2 excess cancers in 10,000 exposed people), with the majority of the risk from inhalation exposure to chromium VI. While these risks represent a low increased risk with respect to the background cancer rate of approximately one in three (NCI 2001), they are well within the regulatory range for needing cleanup⁴.

With regard to chronic noncancer risks, the estimated chromium VI dose to the onsite worker is above the safe dose (i.e. Hazard Index [HI] greater than one). HIs for other contaminants and other exposure scenarios are all below one. When the HI exceeds one, we cannot rule out the possibility of adverse health impacts. When the HI is below one, adverse health effects are considered unlikely.

³ Adjusting a toxicity factor to represent an absorbed dose accounts for the absorption efficiency in the critical study, which forms the basis of the Reference Dose (RfD) or Cancer Slope Factor (CSF). RfDs and CSFs are typically based on administered doses. When oral absorption in the critical study is complete (i.e., 100%), the absorbed dose is equivalent to the administered dose, and no adjustment is necessary. However, when gastrointestinal absorption of a chemical in the critical study is poor, as is true for many inorganic compounds, the absorbed dose is much smaller than the administered dose. In this situation, if the toxicity factor is not adjusted, it can contribute to an underestimation of dermal risk for the chemicals that are poorly absorbed.

⁴ Within the Superfund Program, EPA may choose to cleanup environmental contamination when site risks are in the range of 10^{-4} to 10^{-6} . When risks exceed 10^{-4} , EPA must take action to reduce risks. CT DEP hazardous waste site cleanup standards for individual chemicals are set at 1 x 10^{-6} , with the goal to keep total site risks at or below 1 x 10^{-5} .

With regard to lead, the screening procedure for evaluating lead exposures to the ATV driver estimates that the incremental blood lead level is 1.3 ug/dL and not close to the level of concern for adults (>20 ug/L).

Scenario	Exposure	Exposure	Exposure	Soil Ingestion	Dermal	Excess	Chronic
	Route(s)	Duration	Frequency	Rate	Contact	Lifetime	Hazard
						Cancer Risk ^{&}	Index [#]
Teen Trespasser	Ingestion, Dermal	7 years (teen, aged 16-22 years)	4 days/week, 9 months/year = 144 days per year	50 mg/day, best estimate of average adult rate (EPA 1997)	head, hands, forearms, feet	5 x 10 ⁻⁶	0.8
Adult Onsite Worker	Ingestion, Dermal	10 years (adult, aged 25-35 years)	5 days/week, 9 months/year = 180 days per year	50 mg/day, best estimate of average adult rate (EPA 1997)	head, hands, forearms	2 x 10 ⁻⁵	2.4
ATV driver	Ingestion, Dermal, Inhalation	7 years (teen, aged 16-22 years)	1 day/week, 9 months/year = 36 days per year. For inhalation, 4 hours/day	100 mg/day, upper end estimate for activities where greater soil ingestion could occur (EPA 1997)	head, hands, forearms, feet	2 x 10 ⁻⁴	0.7

Table 2. Selected Soil Exposure Assumptions and Risks for Three Scenarios Evaluated at the Contract Plating Site, Stratford, CT

[&]Cumulative cancer risk for all carcinogens.

[#]Maximum noncancer Hazard Index.

With respect to acute exposures, CT DPH calculated an acute HI for arsenic well below one, indicating that acute health effects are unlikely. For other contaminants, CT DPH did not calculate HIs because safe doses (i.e., Reference Doses or Minimal Risk Levels) for acute exposures are not readily available. Instead, CT DPH compared acute site doses with acute effect levels from the scientific literature that were seen to produce adverse health effects in people or laboratory animals. Table 3 presents selected comparisons between acute site doses and levels shown to cause health effects. Also included in Table 3 is a comparison between the chronic site dose of chromium VI to the onsite worker. As stated above, this is the only site dose that exceeded the safe dose (HI greater than one). Even though the HI is greater than one, the comparison in Table 3 shows that the estimated site dose is almost 100 times less than the lowest dose observed to cause adverse health effects. Therefore, even though the HI for the onsite worker's exposure to chromium VI is greater than one, health effects are unlikely.

Because chromium VI (and to a lesser extent cadmium) exposures drive cancer risks, Table 3 also includes comparisons of long-term site exposures with cancer effect levels (CELs) for cadmium and chromium. Site doses are below the literature cancer effect levels. So, despite the fact that cancer risks to the ATV driver are within the regulatory range where cleanup is warranted, it is unlikely that increased cancer cases would be observed among those persons exposed.

Effect Levels.						
Estimated Site	Comment	Effect Level	Comment			
Dose		from Literature				
0.007 mg/kg/day	Chronic ADD (Onsite	0.57 mg/kg/day	Chronic LOAEL,			
			gastrointestinal and			
	dermal		hematological effects in humans			
			(ATSDR 2000)			
0.039 mg/kg/day	Acute dose (ATV driver)	0.04 mg/kg/day	Oral acute LOAEL, dermatitis in			
	ingestion and dermal		humans (ATSDR 2000)			
0.72 ug/m^3	Acute ADE, (ATV driver)	29,000 ug/m^3	Inhalation Acute LOAEL, LC ₅₀			
			in rats (ATSDR 2000)			
1.2%	Ave. conc. of chromium	0.175%	Acute Dermal LOAEL for			
	VI in soil ^{&}		$K_2Cr_2O_7$ (VI), dermatitis in			
			guinea pigs (ATSDR 2000)			
$1.19E-3 \text{ ug/m}^3$	Chronic ADE, (ATV	0.100 ug/m^3	CEL, lung tumors in humans for			
_	driver)	_	Cr VI (ATSDR 2000)			
0.036 mg/kg/day	Acute dose, (teen	0.097 mg/kg/day	Oral Acute LOAEL for nickel			
	trespasser) ingestion and		sulfate, allergic dermatitis in			
	dermal		sensitized humans, (ATSDR			
			2003)			
0.63 ug/m^3	Acute ADE, (ATV	635 ug/m^3	Inhalation Acute LOAEL for			
_	Driver)	-	nickel sulfate, respiratory effects			
			in rats (ATSDR 2003)			
1.9%	Ave. conc. of nickel in	0.03 %	Acute Dermal LOAEL for nickel			
	soil [#]		sulfate, contact dermatitis in			
			sensitive humans (ATSDR 2003)			
0.03 mg/kg/day	Acute dose (ATV driver)	1 mg/kg/day	Oral Acute LOAEL,			
	ingestion and dermal		developmental effects in			
			hamsters (ATSDR 1997)			
		1.52 mg/kg	Average fatal dose in humans			
			(ATSDR 1997)			
3.9 E-5	LADD (onsite worker),	3.5 mg/kg/day	CEL prostatic adenomas in rats,			
mg/kg/day	ingestion and dermal		cadmium chloride (ATSDR			
_			1999a)			
	Estimated Site Dose 0.007 mg/kg/day 0.039 mg/kg/day 0.72 ug/m ³ 1.2% 1.19E-3 ug/m ³ 0.036 mg/kg/day 0.63 ug/m ³ 1.9% 0.03 mg/kg/day 3.9 E-5	Estimated Site DoseComment0.007 mg/kg/dayChronic ADD (Onsite Worker), ingestion and dermal0.039 mg/kg/dayAcute dose (ATV driver) ingestion and dermal0.72 ug/m³Acute ADE, (ATV driver)1.2%Ave. conc. of chromium VI in soil*1.19E-3 ug/m³Chronic ADE, (ATV driver)0.036 mg/kg/dayAcute dose, (teen trespasser) ingestion and dermal0.63 ug/m³Acute ADE, (ATV Driver)1.9%Ave. conc. of nickel in soil*0.03 mg/kg/dayAcute dose (ATV driver)1.9%Ave. conc. of nickel in soil*0.03 mg/kg/dayAcute dose (ATV driver) ingestion and dermal0.03 mg/kg/dayAcute dose (ATV driver) ingestion and dermal3.9 E-5LADD (onsite worker),	Estimated Site DoseCommentEffect Level from Literature0.007 mg/kg/dayChronic ADD (Onsite Worker), ingestion and dermal0.57 mg/kg/day0.039 mg/kg/dayAcute dose (ATV driver) ingestion and dermal0.04 mg/kg/day0.72 ug/m³Acute dose (ATV driver) ingestion and dermal0.04 mg/kg/day1.2%Ave. conc. of chromium VI in soil ^{&} 0.175%1.19E-3 ug/m³Chronic ADE, (ATV driver)0.100 ug/m³0.036 mg/kg/dayAcute dose, (teen trespasser) ingestion and dermal0.097 mg/kg/day0.63 ug/m³Acute ADE, (ATV Driver)635 ug/m³1.9%Ave. conc. of nickel in soil [#] 0.03 %0.03 mg/kg/dayAcute dose (ATV driver) ingestion and dermal1 mg/kg/day1.9%Acute dose (ATV driver) Ingestion and dermal1 mg/kg/day1.9%Acute dose (ATV driver) ingestion and dermal1 mg/kg/day0.03 mg/kg/dayAcute dose (ATV driver) ingestion and dermal1 mg/kg/day0.99 Fe-5LADD (onsite worker),3.5 mg/kg/day			

 Table 3. Comparison of Selected Exposure Doses from the Contract Plating Site with Literature Effect Levels.

 LC_{50} – Lethal concentration to 50% of the animals

DC₅₀ – Concentration resulting in 50% decrease in average respiratory rate.

CEL – Cancer Effect Level

LOAEL – Lowest Observable Adverse Effect Level

ADD – Average Daily Dose

LADD – Lifetime Average Daily Dose

ADE – Average Daily Exposure (in air)

[#] Average soil concentration of 19,729 ppm * 1%/10,000 ppm = 1.9%

[&] Average soil concentration of 12,073 ppm * 1%/10,000 ppm = 1.2%

As stated above, Table 3 also presents a comparison of acute site doses with acute effect levels. Estimated acute exposures are close to or above literature effect levels for chromium VI and nickel. The effect seen in the literature studies is allergic dermatitis in sensitized humans and in animals. The compounds in the studies were nickel sulfate and potassium dichromate.

It is important to stress that chromium and nickel soil data from the site are not speciated. CT DPH assumed that all of the chromium measured in soil at the site is in the form of chromium

VI. This is an extremely conservative assumption because chromium VI is much more toxic than chromium III and is more likely to cause acute skin effects in sensitized individuals (ATSDR 2000). In addition, it would be very unusual for 100% of the chromium in the soil to be present as chromium VI. With regard to nickel, the soluble nickel salts (nickel chloride, nickel nitrate, nickel sulfate) are generally more toxic than the less soluble compounds, although the less soluble compounds are more likely to be carcinogenic at the site of deposition (ATSDR 2003). Since we do not have information about which nickel compounds are present at the site and how much of the chromium at the site is the more toxic chromium VI, it is not possible to rule out the possibility that acute exposures to soil in the former lagoon area could cause acute health impacts such as allergic dermatitis.

For the other contaminants for which acute exposures were evaluated (PCBs, cadmium, antimony, selenium and zinc), comparisons between site doses and literature values were not included in Table 3 because the site doses were well below effect levels.

Exposures and risks to asbestos in soil were not evaluated. Although the 2002 soil sampling did not find elevated asbestos levels, many brake parts (potentially containing asbestos) were observed in the soil during the site visit. Considering these observations, we cannot rule out the possibility that there are asbestos fibers in soil at high enough levels to cause a public health risk.

Physical/safety hazards exist, activities are occurring that could cause people to come into contact with the hazards, therefore, a threat to public safety exists.

Uncertainties

There are a number of important uncertainties that must be considered in evaluating whether a public health threat exists at the Contract Plating site.

- There are no surface soil data from any locations other than northern portion of site. Risks to a child/adult visitor could not be evaluated in this health consultation because of the absence of surface soil data from areas of the site where they would likely be exposed (immediate vicinity of the main building and former anodizing and chemical storage buildings).
- Asbestos soil data may not be representative of current conditions. The most recent asbestos sampling occurred in 2002. Although numerous pieces of brake material were observed in the soil in the northern portion of the site, the 2002 sampling did not detect asbestos at levels greater than 1%. It is possible that the brake materials have degraded during the four years since sampling occurred and if sampling occurred today, asbestos could be detected at elevated levels.
- The toxicity of nickel and chromium at the site depends on the form that is present in soil. The analyses of soil samples at Contract Plating did not speciate these metals. Therefore, it was necessary for CT DPH to make conservative assumptions about these metals when evaluating risks from exposure. Without speciated data for metals in soils, CT DPH cannot assess the magnitude of conservativeness in its exposure assumptions.
- For purposes of assessing exposures and risks, CT DPH assumed that the use of an ATV at the site occurs regularly (4 hours per day, one day per week, 9 months per year). It is possible that ATV use is much less frequent than this, considering the fact that the site owner stated that she does not permit anyone to drive vehicles in the former lagoon area.

We do know that some ATV use did occur recent to the site visit. If ATV use at the site is less frequent that what CT DPH assumed in the risk evaluation, exposures and potential health impacts would also be lower.

• When evaluating dermal exposures and risks, CT DPH did not adjust oral toxicity values to represent absorbed, rather than administered dose, even though the metals present at the site are poorly absorbed across the GI tract. As stated previously, EPA recommends adjusting a toxicity factor when GI absorption of a chemical is poor. Not making this adjustment could lead to an underestimation of the risks from the dermal exposure pathway. CT DPH chose not to make the toxicity factor adjustment because the risk assessment already includes some very conservative assumptions (namely that all of a particular metal measured in soil was present in the form that is most toxic). These conservative assumptions are likely to lead to an overestimate of the risks from the site.

Conclusions

Soil data from the Contract Plating site indicates that a variety of metals, as well as PCBs and cyanide are present in surface and subsurface soils in the former plating waste lagoon areas at concentrations exceeding health protective comparison values. Chromium, cadmium, nickel, and cyanide were found at very high concentrations in soil, relative to comparison values. In addition, physical/safety hazards were observed inside the former main process building as well as outside the former anodizing buildings and in the former lagoon area. Some of the most significant physical/safety hazards include open pits, debris falling from the roof, and broken glass.

The Contract Plating site is not adequately secured. There is evidence that trespassers have accessed the site, possibly recently. In addition, ATV use has occurred recently in the former lagoon area. Some landscaping-type work is also ongoing at the site. Sports equipment and toys were observed near one of the onsite buildings. Based on all of these observations, CT DPH concludes that there is the potential for exposure to soil contaminants and physical/safety hazards by four receptor groups (teen/adult trespasser, teen/adult ATV driver, onsite adult worker, and child/adult visitor). Because critical soil sampling data are absent, the level of public health hazard was not determined for child/adults visitors who may be accessing the areas near the onsite buildings.

Cancer risks to trespassers and onsite workers are low, relative to the very high background cancer risks that exist (NCI 2001). Cancer risks to the ATV driver receptor group are within the regulatory range requiring remediation. Onsite workers' long-term exposure to chromium VI is above the safe level. However, this exposure is still below health effect levels from the scientific literature. Acute exposures to the most toxic forms of nickel and chromium VI are *close to or above* literature effect levels for adverse health impacts (allergic dermatitis) in humans and animals. Even though we do not know how much of the most toxic forms of these metals are actually present at the site, we cannot rule out the possibility that there is a threat to public health from short-term exposures.

ATSDR has a categorization scheme whereby the level of public health hazard at a site is assigned to one of five conclusion categories. Conclusion categories are included as Attachment

I. CT DPH has concluded that under current conditions, the Contract Plating site presents a *public health hazard* because actions are needed to address physical/safety hazards at the site and to reduce the potential for harm from exposure to chemical hazards by trespassers, ATV drivers and onsite workers.

Recommendations

1. CT DEP and the Town of Stratford (the Town) should ensure that the property owner addresses the open pit and open catch basin safety hazards on the property. In addition, the CT DEP and the Town should work with the property owner on a plan to eliminate safety hazards in the main process building. Ideally, this plan would consist of demolishing the building because it is in great disrepair and presents serious safety hazards.

2. CT DEP and the Town should work with the property owner on ways to improve access restrictions on site. Actions that should be considered are repairing the breaches in the chain link fence which extends along the property boundary and improving the access restrictions from the road into the parking lot.

3. CT DEP and the Town should ensure that the property owner does not allow any employees or visitors to drive vehicles in the former lagoon area.

4. CT DEP and the Town should work to ensure that additional sampling on the property is conducted to fill data gaps identified in this health consultation. Of particular interest is whether there is asbestos present in soil at levels that could cause health concerns.

5. CT DEP and the Town should work to ensure that soil remediation on the property occurs as quickly as is feasible.

6. The Town should ensure that commercial uses of the property be prohibited until safety hazards and environmental data gaps are addressed.

7. The Town should ensure that warning signs (i.e., no trespassing, hazardous waste present) are posted along the property boundaries.

8. The Town should provide the property owner with information about ways to minimize exposure to soil and should encourage the owner to provide the information to individuals who work on the site or who visit the site regularly.

Public Health Action Plan

Actions Taken

1. At the request of the SHD and CT DEP, CT DPH conducted a site visit of the Contract Plating site in February 2006.

2. At the request of the SHD and CT DEP, CT DPH reviewed environmental data and prepared this health consultation.

Actions Planned

1. CT DPH will work with CT DEP, the property owner and the Town to provide technical assistance regarding sampling plans and evaluating data.

2. CT DPH will evaluate new sampling data as it becomes available and will update the conclusions and recommendations contained in this Health Consultation, as needed.

3. CT DPH will work with the Town, CT DEP and the property owner, as necessary to ensure that recommendations made in this Health Consultation are carried out in a reasonable timeframe.

4. CT DPH will work with the property owner to address their health or exposure-related questions or concerns.

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CERTIFICATION

The Health Consultation for the Public Health Evaluation of Environmental Data from Contract Plating, Stratford, Connecticut was prepared by the Connecticut Department of Public Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It was completed in accordance with approved methodology and procedures existing at the time the health consultation was initiated. Editorial review was completed by the ATSDR Cooperative Agreement Partner.

Greg Ulirsch Technical Project Officer Division of Health Assessment and Consultation (DHAC) Agency for Toxic Substances and Disease Registry (ATSDR)

The Division of Health Assessment and Consultation (DHAC), ATSDR, has reviewed this health consultation and concurs with its findings.

Team Leader-Coop Agreement Program CAT, DHAC, ATSDR

Preparer of Health Consultation

Margaret L. Harvey, MPH Epidemiologist Environmental and Occupation Health Assessment Program Connecticut Department of Public Health

ATSDR Regional Representative

William Sweet EPA/New England

ATSDR Technical Project Officer

Greg Ulirsch Division of Health Assessment and Consultation Agency for Toxic Substances and Disease Registry

Appendix A

Site Plan Contract Plating, Stratford, CT

Historic Site Diagram Contract Plating, Stratford, CT





Appendix B

Photographs of Interior and Exterior of Main Process Building, Contract Plating, Stratford, CT



Front of Main Process Building

Interior of Main Process Building

Interior of Main Process Building – Pipe Insulation



Interior of Main Process Building – Evidence of Trespassing

Interior of Main Process Building – Evidence of Trespassing

Exterior of Main Process Building – Broken Windows

Appendix C

Photographs of Process Buildings, Chemical Storage Buildings, Open Pit, Sporting Equipment Contract Plating, Stratford, CT



Toy car outside Former Chemical Storage Building

Inside Former Chemical Storage Building

Sporting equipment outside former Chemical Storage Building



Sporting Equipment Outside Former Chemical Storage Building

Open Pit near former Chemical Storage Building

All Terrain Vehicle parked outside former Chemical Storage Building Appendix D

Photographs of Former Lagoon Area

Contract Plating, Stratford CT



Former Lagoon Area

Tire Tracks in Former Lagoon Area

Brake Part in Former Lagoon Area.



Brake Part with Tire Tracks in Former Lagoon Area.

Open Catch Basin (Safety Hazard) in Former Lagoon Area.

Open Pipe (Safety Hazard) in Former Lagoon Area.

Appendix E

Photographs of Breaches in Fence, Evidence of Trespassing Contract Plating, Stratford, CT



Breach in the Fence along Northern Property Boundary

Breach in the Fence along Northeastern Property Boundary (Former Raymark Site is visible across railroad tracks).

Sleeping Bag (evidence of trespassing).


Burned wood in northern portion of the site near sleeping bag (evidence of trespassing).

Empty beer can (evidence of trespassing).

Breach in fence along northern property boundary. Appendix F

Demographic Statistics

Contract Plating, Stratford, CT

Contract Plating Stratford, CT

*A*TSDR

EPA Facility ID: CTD001180462



Base Map Source: Geographic Data Technology, May 2005. Site Boundary Data Source: ATSDR Geospatial Research, Analysis, and Services Program, Current as of Generate Date (bottom left-hand corner). Coordinate System (All Panels): NAD 1983 StatePlane Connecticut FIPS 0600 Feet



Demographic Statistics Within One Mile of Site*

Total Population	14,061
White Alone	13,150
Black Alone	380
Am. Indian & Alaska Native Alone	12
Asian Alone	178
Native Hawaiian &	
Other Pacific Islander Alone	1
Some Other Race Alone	170
Two or More Races	169
Hispanic or Latino**	715
Children Aged 6 and Younger	1,301
Adults Aged 65 and Older	2,214
Females Aged 15 to 44	2,935
Total Housing Units	5,829

Demographics Statistics Source: 2000 U.S. Census

* Calculated using an area-proportion spatial analysis technique ** People who identify their origin as Hispanic or Latino may

be of any race.



Adults 65 Years and Older By US Census Block Cero Population 1 - 9 Adults > 20 Adults > 20 Adults > 20 Adults - 20 Adults

k n By US Census Block By US Census Block Census Census Census Block C

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Appendix G

Toxicological Summaries

Agency for Toxic Substances and Disease Registry ToxFAQs

This fact sheet answers the most frequently asked health questions (FAQs) about antimony. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to antimony occurs in the workplace or from skin contact with soil at hazardous waste sites. Breathing high levels of antimony for a long time can irritate the eyes and lungs, and can cause problems with the lungs, heart, and stomach. This chemical has been found in at least 403 of 1,416 National Priorities List sites identified by the Environmental Protection Agency.

What is antimony?

(Pronounced ăn/tə-mō'nē)

Antimony is a silvery-white metal that is found in the earth's crust. Antimony ores are mined and then mixed with other metals to form antimony alloys or combined with oxygen to form antimony oxide.

Little antimony is currently mined in the United States. It is brought into this country from other countries for processing. However, there are companies in the United States that produce antimony as a by-product of smelting lead and other metals.

Antimony isn't used alone because it breaks easily, but when mixed into alloys, it is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony oxide is added to textiles and plastics to prevent them from catching fire. It is also used in paints, ceramics, and fireworks, and as enamels for plastics, metal, and glass.

What happens to antimony when it enters the environment?

- Antimony is released to the environment from natural sources and from industry.
- □ In the air, antimony is attached to very small particles that may stay in the air for many days.

- □ Most antimony ends up in soil, where it attaches strongly to particles that contain iron, manganese, or aluminum.
- □ Antimony is found at low levels in some rivers, lakes, and streams.

How might I be exposed to antimony?

- □ Because antimony is found naturally in the environment, the general population is exposed to low levels of it every day, primarily in food, drinking water, and air.
- □ It may be found in air near industries that process or release it, such as smelters, coal-fired plants, and refuse incinerators.
- □ In polluted areas containing high levels of antimony, it may be found in the air, water, and soil.
- □ Workers in industries that process it or use antimony ore may be exposed to higher levels.

How can antimony affect my health?

Exposure to antimony at high levels can result in a variety of adverse health effects.

Breathing high levels for a long time can irritate your eyes and lungs and can cause heart and lung problems, stomach pain, diarrhea, vomiting, and stomach ulcers.

In short-term studies, animals that breathed very high levels of antimony died. Animals that breathed high levels

September 1995

ANTIMONY

CAS # 7440-36-0



ANTIMONY CAS # 7440-36-0

ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html

had lung, heart, liver, and kidney damage. In long-term studies, animals that breathed very low levels of antimony had eye irritation, hair loss, lung damage, and heart problems. Problems with fertility were also noted. In animal studies, problems with fertility have been seen when rats breathed very high levels of antimony for a few months.

Ingesting large doses of antimony can cause vomiting. We don't know what other effects may be caused by ingesting it. Long-term animal studies have reported liver damage and blood changes when animals ingested antimony. Antimony can irritate the skin if it is left on it.

Antimony can have beneficial effects when used for medical reasons. It has been used as a medicine to treat people infected with parasites.

How likely is antimony to cause cancer?

The Department of Health and Human Services, the International Agency for Research on Cancer, and the Environmental Protection Agency (EPA) have not classified antimony as to its human carcinogenicity.

Lung cancer has been observed in some studies of rats that breathed high levels of antimony. No human studies are available. We don't know whether antimony will cause cancer in people.

Is there a medical test to show whether I've been exposed to antimony?

Tests are available to measure antimony levels in the body. Antimony can be measured in the urine, feces, and blood for several days after exposure. However, these tests cannot tell you how much antimony you have been exposed to or whether you will experience any health effects. Some tests are not usually performed in most doctors' offices and may require special equipment to conduct them.

Has the federal government made recommendations to protect human health?

The EPA allows 0.006 parts of antimony per million parts of drinking water (0.006 ppm). The EPA requires that discharges or spills into the environment of 5,000 pounds or more of antimony be reported.

The Occupational Safety and Health Administration (OSHA) has set an occupational exposure limit of 0.5milligrams of antimony per cubic meter of air (0.5 mg/m³) for an 8-hour workday, 40-hour workweek.

The American Conference of Governmental Industrial Hygienists (ACGIH) and the National Institute for Occupational Safety and Health (NIOSH) currently recommend the same guidelines for the workplace as OSHA.

Glossary

Carcinogenicity: Ability to cause cancer. CAS: Chemical Abstracts Service. Ingestion: Taking food or drink into your body. Long-term: Lasting one year or more. Milligram (mg): One thousandth of a gram. Parasite: An organism living in or on another organism. ppm: Parts per million. Short-term: Lasting 14 days or less.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological profile for antimony. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



Division of Toxicology and Environmental Medicine ToxFAQsTM

This fact sheet answers the most frequently asked health questions (FAQs) about arsenic. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to higher than average levels of arsenic occur mostly in the workplace, near hazardous waste sites, or in areas with high natural levels. At high levels, inorganic arsenic can cause death. Exposure to lower levels for a long time can cause a discoloration of the skin and the appearance of small corns or warts. Arsenic has been found in at least 784 of the 1,662 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is arsenic?

Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds.

Inorganic arsenic compounds are mainly used to preserve wood. Copper chromated arsenic (CCA) is used to make "pressure-treated" lumber. CCA is no longer used in the U.S. for residential uses; it is still used in industrial applications. Organic arsenic compounds are used as pesticides, primarily on cotton plants.

What happens to arsenic when it enters the environment?

□ Arsenic occurs naturally in soil and minerals and it therefore may enter the air, water, and land from wind-blown dust and may get into water from runoff and leaching.

□ Arsenic cannot be destroyed in the environment. It can only change its form.

□ Rain and snow remove arsenic dust particles from the air. □ Many common arsenic compounds can dissolve in water. Most of the arsenic in water will ultimately end up in soil or sediment.

□ Fish and shellfish can accumulate arsenic; most of this arsenic is in an organic form called arsenobetaine that is much less harmful.

How might I be exposed to arsenic?

□ Ingesting small amounts present in your food and water or breathing air containing arsenic.

□ Breathing sawdust or burning smoke from wood treated with arsenic.

Living in areas with unusually high natural levels of arsenic in rock.

U Working in a job that involves arsenic production or use, such as copper or lead smelting, wood treating, or pesticide application.

How can arsenic affect my health?

Breathing high levels of inorganic arsenic can give you a sore throat or irritated lungs.

Ingesting very high levels of arsenic can result in death. Exposure to lower levels can cause nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of "pins and needles" in hands and feet.

Ingesting or breathing low levels of inorganic arsenic for a long time can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso.

Skin contact with inorganic arsenic may cause redness and swelling.

ARSENIC

CAS # 7440-38-2



September 2005

ARSENIC CAS # 7440-38-2

ToxFAQs[™] Internet address is http://www.atsdr.cdc.gov/toxfaq.html

Organic arsenic compounds are less toxic than inorganic arsenic compounds. Exposure to high levels of some organic arsenic compounds may cause similar effects as inorganic arsenic.

How likely is arsenic to cause cancer?

Several studies have shown that ingestion of inorganic arsenic can increase the risk of skin cancer and cancer in the lungs, bladder, liver, kidney and prostate. Inhalation of inorganic arsenic can cause increase risk of lung cancer. The Department of Health and Human Services (DHHS) has determined that inorganic arsenic is a known carcinogen. The International Agency for Research on Cancer (IARC), and the EPA have determined that inorganic arsenic is carcinogenic to humans.

How can arsenic affect children?

There is also some evidence that suggests that long-term exposure to arsenic in children may result in lower IQ scores. There is some information suggesting that children may be less efficient at converting inorganic arsenic to the less harmful organic forms. For this reason, children may be more susceptible to health effects from inorganic arsenic than adults.

There is some evidence that inhaled or ingested arsenic can injure pregnant women or their unborn babies, although the studies are not definitive. Studies in animals show that large doses of arsenic that cause illness in pregnant females can also cause low birth weight, fetal malformations, and even fetal death. Arsenic can cross the placenta and has been found in fetal tissues. Arsenic is found at low levels in breast milk.

How can families reduce the risks of exposure to arsenic?

□ If you use arsenic-treated wood in home projects, you should wear dust masks, gloves, and protective clothing to decrease exposure to sawdust.

□ If you live in an area with high levels of arsenic in water or soil, you should use cleaner sources of water and limit contact with soil.

Is there a medical test to determine whether I've been exposed to arsenic?

There are tests available to measure arsenic in your blood, urine, hair, and fingernails. The urine test is the most reliable test for arsenic exposure within the last few days. Tests on hair and fingernails can measure exposure to high levels of arsenic over the past 6-12 months. These tests can determine if you have been exposed to above-average levels of arsenic. They cannot predict how the arsenic levels in your body will affect your health.

Has the federal government made recommendations to protect human health?

The EPA has set limits on the amount of arsenic that industrial sources can release to the environment and has restricted or cancelled many of the uses of arsenic in pesticides. EPA has set a limit of 0.01 parts per million (ppm) for arsenic in drinking water.

The Occupational Safety and Health Administration (OSHA) has set a permissible exposure limit (PEL) of 10 micrograms of arsenic per cubic meter of workplace air ($10 \mu g/m^3$) for 8 hour shifts and 40 hour work weeks.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological Profile for Arsenic (Draft for Public Comment). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



Agency for Toxic Substances and Disease Registry ToxFAQs

This fact sheet answers the most frequently asked health questions (FAQs) about cadmium. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to cadmium happens mostly in the workplace where cadmium products are made. The general population is exposed from breathing cigarette smoke or eating cadmium contaminated foods. Cadmium damages the lungs, can cause kidney disease, and may irritate the digestive tract. This substance has been found in at least 776 of the 1,467 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is cadmium?

(Pronounced kăd/mē-əm)

Cadmium is a natural element in the earth's crust. It is usually found as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide).

All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Most cadmium used in the United States is extracted during the production of other metals like zinc, lead, and copper. Cadmium does not corrode easily and has many uses, including batteries, pigments, metal coatings, and plastics.

What happens to cadmium when it enters the environment?

- Cadmium enters air from mining, industry, and burning coal and household wastes.
- Cadmium particles in air can travel long distances before falling to the ground or water.
- □ It enters water and soil from waste disposal and spills or leaks at hazardous waste sites.
- □ It binds strongly to soil particles.
- □ Some cadmium dissolves in water.

- □ It doesn't break down in the environment, but can change forms.
- □ Fish, plants, and animals take up cadmium from the environment.
- □ Cadmium stays in the body a very long time and can build up from many years of exposure to low levels.

How might I be exposed to cadmium?

- □ Breathing contaminated workplace air (battery manufacturing, metal soldering or welding).
- □ Eating foods containing it; low levels in all foods (highest in shellfish, liver, and kidney meats).
- □ Breathing cadmium in cigarette smoke (doubles the average daily intake).
- Drinking contaminated water.
- □ Breathing contaminated air near the burning of fossil fuels or municipal waste.

How can cadmium affect my health?

Breathing high levels of cadmium severely damages the lungs and can cause death. Eating food or drinking water with very high levels severely irritates the stomach, leading to vomiting and diarrhea. Long-term exposure to lower levels of cadmium in air, food, or water leads to a buildup of cadmium in the kidneys and possible kidney disease.

June 1999

CADMIUM

CAS # 7440-43-9



ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html

Other long-term effects are lung damage and fragile bones. Animals given cadmium in food or water had high blood pressure, iron-poor blood, liver disease, and nerve or brain damage.

We don't know if humans get any of these diseases from eating or drinking cadmium. Skin contact with cadmium is not known to cause health effects in humans or animals.

How likely is cadmium to cause cancer?

The Department of Health and Human Services (DHHS) has determined that cadmium and cadmium compounds may reasonably be anticipated to be carcinogens.

How can cadmium affect children?

The health effects in children are expected to be similar to those in adults (kidney, lung and intestinal damage).

We don't know if cadmium causes birth defects in people. Cadmium does not readily go from a pregnant woman's body into the developing child, but some portion can cross the placenta. It can also be found in breast milk. The babies of animals exposed to high levels of cadmium during pregnancy had changes in behavior and learning ability. Cadmium may also affect birth weight and the skeleton in developing animals.

Animal studies also indicate that more cadmium is absorbed into the body if the diet is low in calcium, protein, or iron, or is high in fat. A few studies show that younger animals absorb more cadmium and are more likely to lose bone and bone strength than adults.

How can families reduce the risk of exposure to cadmium?

In the home, store substances that contain cadmium safely, and keep nickel-cadmium batteries out of reach of young children. If you work with cadmium, use all safety precautions to avoid carrying cadmium-containing dust home from work on your clothing, skin, hair, or tools.

A balanced diet can reduce the amount of cadmium taken into the body from food and drink.

Is there a medical test to show whether I've been exposed to cadmium?

Tests are available in some medical laboratories that measure cadmium in blood, urine, hair, or nails. Blood levels show recent exposure to cadmium, and urine levels show both recent and earlier exposure. The reliability of tests for cadmium levels in hair or nails is unknown.

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 5 parts of cadmium per billion parts of drinking water (5 ppb). EPA doesn't allow cadmium in pesticides.

The Food and Drug Administration (FDA) limits the amount of cadmium in food colors to 15 parts per million (15 ppm).

The Occupational Safety and Health Administration (OSHA) limits workplace air to 100 micrograms cadmium per cubic meter (100 μ g/m³) as cadmium fumes and 200 μ g cadmium/m³ as cadmium dust.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for cadmium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.





CHROMIUM CAS # 7440-47-3

February 2001

Division of Toxicology ToxFAQsTM

This fact sheet answers the most frequently asked health questions (FAQs) about chromium. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to chromium occurs from ingesting contaminated food or drinking water or breathing contaminated workplace air. Chromium(VI) at high levels can damage the nose and can cause cancer. Chromium has been found at 1,036 of the 1,591 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is chromium?

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms. The most common forms are chromium(0), chromium(III), and chromium(VI). No taste or odor is associated with chromium compounds.

Chromium(III) occurs naturally in the environment and is an essential nutrient. Chromium(VI) and chromium(0) are generally produced by industrial processes.

The metal chromium, which is the chromium(0) form, is used for making steel. Chromium(VI) and chromium(III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving.

What happens to chromium when it enters the environment?

□ Chromium enters the air, water, and soil mostly in the chromium(III) and chromium(VI) forms.

In air, chromium compounds are present mostly as fine dust particles which eventually settle over land and water.
Chromium can strongly attach to soil and only a small

amount can dissolve in water and move deeper in the soil to underground water.

□ Fish do not accumulate much chromium in their bodies from water.

How might I be exposed to chromium?

□ Eating food containing chromium(III).

□ Breathing contaminated workplace air or skin contact during use in the workplace.

□ Drinking contaminated well water.

□ Living near uncontrolled hazardous waste sites containing chromium or industries that use chromium.

How can chromium affect my health?

Chromium(III) is an essential nutrient that helps the body use sugar, protein, and fat.

Breathing high levels of chromium(VI) can cause irritation to the nose, such as runny nose, nosebleeds, and ulcers and holes in the nasal septum.

Ingesting large amounts of chromium(VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, Public Health Service Agency for Toxic Substances and Disease Registry

CHROMIUM CAS # 7440-47-3

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Skin contact with certain chromium(VI) compounds can cause skin ulcers. Some people are extremely sensitive to chromium(VI) or chromium(III). Allergic reactions consisting of severe redness and swelling of the skin have been noted.

How likely is chromium to cause cancer?

Several studies have shown that chromium(VI) compounds can increase the risk of lung cancer. Animal studies have also shown an increased risk of cancer.

The World Health Organization (WHO) has determined that chromium(VI) is a human carcinogen.

The Department of Health and Human Services (DHHS) has determined that certain chromium(VI) compounds are known to cause cancer in humans.

The EPA has determined that chromium(VI) in air is a human carcinogen.

How can chromium affect children?

We do not know if exposure to chromium will result in birth defects or other developmental effects in people. Birth defects have been observed in animals exposed to chromium(VI).

It is likely that health effects seen in children exposed to high amounts of chromium will be similar to the effects seen in adults.

How can families reduce the risk of exposure to chromium?

□ Children should avoid playing in soils near uncontrolled hazardous waste sites where chromium may have been discarded.

□ Although chromium(III) is an essential nutrient, you should avoid excessive use of dietary supplements containing chromium.

Is there a medical test to show whether I've been exposed to chromium?

Since chromium(III) is an essential element and naturally occurs in food, there will always be some level of chromium in your body. There are tests to measure the level of chromium in hair, urine, and blood. These tests are most useful for people exposed to high levels. These tests cannot determine the exact levels of chromium that you may have been exposed to or predict how the levels in your tissues will affect your health.

Has the federal government made recommendations to protect human health?

EPA has set a limit of 100 μ g chromium(III) and chromium(VI) per liter of drinking water (100 μ g/L).

The Occupational Safety and Health Administration (OSHA) has set limits of 500 μ g water soluble chromium(III) compounds per cubic meter of workplace air (500 μ g/m³), 1,000 μ g/m³ for metallic chromium(0) and insoluble chromium compounds, and 52 μ g/m³ for chromium(VI) compounds for 8-hour work shifts and 40-hour work weeks.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological Profile for Chromium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQsTM Internet address is http://www.atsdr.cdc.gov/toxfaq.html . ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



Cyanide

CAS# 74-90-8, 143-33-9, 151-50-8, 592-01-8, 544-92-3, 506-61-6, 460-19-5, 506-77-4

Division of Toxicology ToxFAQsTM

This fact sheet answers the most frequently asked health questions (FAQs) about cyanide. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to high levels of cyanide harms the brain and heart, and may cause coma and death. Exposure to lower levels may result in breathing difficulties, heart pains, vomiting, blood changes, headaches, and enlargement of the thyroid gland. Cyanide has been found in at least 471 of the 1,647 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is cyanide?

Cyanide is usually found joined with other chemicals to form compounds. Examples of simple cyanide compounds are hydrogen cyanide, sodium cyanide and potassium cyanide. Certain bacteria, fungi, and algae can produce cyanide, and cyanide is found in a number of foods and plants. In certain plant foods, including almonds, millet sprouts, lima beans, soy, spinach, bamboo shoots, and cassava roots (which are a major source of food in tropical countries), cyanides occur naturally as part of sugars or other naturally-occurring compounds. However, the edible parts of plants that are eaten in the United States, including tapioca which is made from cassava roots, contain relatively low amounts of cyanide.

Hydrogen cyanide is a colorless gas with a faint, bitter, almond-like odor. Sodium cyanide and potassium cyanide are both white solids with a bitter, almond-like odor in damp air. Cyanide and hydrogen cyanide are used in electroplating, metallurgy, organic chemicals production, photographic developing, manufacture of plastics, fumigation of ships, and some mining processes.

What happens to cyanide when it enters the environment?

□ Cyanide enters air, water, and soil from both natural processes and industrial activities.

In air, cyanide is mainly found as gaseous hydrogen cyanide; a small amount is present as fine dust particles.
The half-life (the time needed for half of the material to be removed) of hydrogen cyanide in the atmosphere is about 1–3 years.

□ Most cyanide in surface water will form hydrogen cyanide and evaporate.

□ Cyanide in water does not build up in the bodies of fish. □ Cyanides are fairly mobile in soil. Once in soil, cyanide can be removed through several processes. Some cyanide compounds in soil can form hydrogen cyanide and evaporate whereas some cyanide compounds will be transformed into other chemical forms by microorganisms in soil. At the high concentrations, cyanide becomes toxic to soil microorganisms. Because these microorganisms can no longer change cyanide to other chemical forms, cyanide is able to passes through soil into underground water.

How might I be exposed to cyanide?

□ Breathing air, drinking water, touching soil, or eating foods that contain cyanide.

□ Smoking cigarettes and breathing smoke-filled air during fires are major sources of cyanide exposure.

□ Breathing air near a hazardous waste site containing cyanide.

□ Eating foods naturally containing cyanide compounds, such as tapioca (made from cassava roots), lima beans, and almonds. However, the portions of these plants that are eaten in the United States contain relatively low amounts of cyanide.

How can cyanide affect my health?

You are not likely to be exposed to large enough amounts of cyanide in the environment to cause adverse health effects. The severity of the harmful effects following cyanide exposure depends in part on the form of cyanide, such as

September 2004



CAS# 74-90-8, 143-33-9, 151-50-8, 592-01-8, 544-92-3, 506-61-6, 460-19-5, 506-77-4

ToxFAQsTM Internet address is http://www.atsdr.cdc.gov/toxfaq.html

hydrogen cyanide gas or cyanide salts. Exposure to high levels of cyanide for a short time harms the brain and heart and can even cause coma and death. Workers who inhaled low levels of hydrogen cyanide over a period of years had breathing difficulties, chest pain, vomiting, blood changes, headaches, and enlargement of the thyroid gland. Some of the first indications of cyanide poisoning are rapid, deep breathing and shortness of breath, followed by convulsions (seizures) and loss of consciousness. These symptoms can occur rapidly, depending on the amount eaten. The health effects of large amounts of cyanide are similar, whether you eat, drink, or breathe it; cyanide uptake into the body through the skin is slower than these other means of exposure. Skin contact with hydrogen cyanide or cyanide salts can irritate and produce sores.

How likely is cyanide to cause cancer?

There are no reports that cyanide can cause cancer in people or animals. EPA has determined that cyanide is not classifiable as to its human carcinogenicity.

How can cyanide affect children?

Effects reported in exposed children are like those seen in exposed adults. Children who ate large quantities of apricot pits, which naturally contain cyanide as part of complex sugars, had rapid breathing, low blood pressure, headaches, and coma, and some died. Cyanide has not been reported to directly cause birth defects in people. However, among people in the tropics who eat cassava root, children have been born with thyroid disease because of the mothers' exposure to cyanide and thiocyanate during pregnancy. Birth defects on the reproductive system occurred in rats and mice that drank water containing sodium cyanide.

How can families reduce the risk of exposure to cyanide?

Families can reduce their exposure to cyanide by not breathing in tobacco smoke, which is the most common source of cyanide exposure for the general population. In the event of a building fire, families should evacuate the building immediately, because smoke from burning plastics contains cyanide (and carbon monoxide). Breathing this smoke can lead to unconsciousness or death. Cyanide in smoke can arise from the combustion of certain plastics (e.g., polyacrylamines, polyacrylics, polyurethane, etc.). Compounds that release cyanide are naturally present in plants. The amounts are usually low in the edible portion but are higher in cassava. Pits and seeds of common fruits, such as apricots, apples, and peaches, may have substantial amounts of cyanide-releasing chemicals, so people should avoid eating these pits and seeds to prevent accidental cyanide poisoning.

Is there a medical test to show whether I've been exposed to cyanide?

There are medical tests to measure blood and urine levels of cyanide; however, small amounts of cyanide are always detectable in blood and urine. Tissue levels of cyanide can be measured if cyanide poisoning is suspected, but cyanide is rapidly cleared from the body, so the tests must be done soon after the exposure. An almond-like odor in the breath may alert a physician that a person was exposed to cyanide.

Has the federal government made

recommendations to protect human health?

EPA regulates the levels of cyanide that are allowable in drinking water. The highest level of cyanide allowed in drinking water is 0.2 parts cyanide per 1 million parts of water (0.2 ppm).

The Occupational Safety and Health Administration (OSHA) has set a limit for hydrogen cyanide and most cyanide salts of 10 parts cyanide per 1 million parts of air (10 ppm) in the workplace.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Toxicological Profile for Cyanide (Draft for Public Comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



Division of Toxicology and Environmental Medicine ToxFAQsTM

This fact sheet answers the most frequently asked health questions (FAQs) about lead. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to lead can happen from breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system. Lead has been found in at least 1,280 of the 1,662 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is lead?

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing.

Lead has many different uses. It is used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from gasoline, paints and ceramic products, caulking, and pipe solder has been dramatically reduced in recent years.

What happens to lead when it enters the environment?

□ Lead itself does not break down, but lead compounds are changed by sunlight, air, and water.

□ When lead is released to the air, it may travel long distances before settling to the ground.

□ Once lead falls onto soil, it usually sticks to soil particles.

□ Movement of lead from soil into groundwater will depend on the type of lead compound and the characteristics of the soil.

How might I be exposed to lead?

□ Eating food or drinking water that contains lead. Water pipes in some older homes may contain lead solder. Lead can leach out into the water.

□ Spending time in areas where lead-based paints have been used and are deteriorating. Deteriorating lead paint can contribute to lead dust.

Working in a job where lead is used or engaging in certain hobbies in which lead is used, such stained glass.
Using health-care products or folk remedies that contain lead.

How can lead affect my health?

The effects of lead are the same whether it enters the body through breathing or swallowing. Lead can affect almost every organ and system in your body. The main target for lead toxicity is the nervous system, both in adults and children. Long-term exposure of adults can result in decreased performance in some tests that measure functions of the nervous system. It may also cause weakness in fingers, wrists, or ankles. Lead exposure also causes small increases in blood pressure, particularly in middle-aged and older people and can cause anemia. Exposure to high lead levels can severely damage the brain and kidneys in adults or children and ultimately cause death. In pregnant women, high levels of exposure to lead may cause miscarriage. Highlevel exposure in men can damage the organs responsible for sperm production.

How likely is lead to cause cancer?

We have no conclusive proof that lead causes cancer in humans. Kidney tumors have developed in rats and mice that had been given large doses of some kind of lead compounds. The Department of Health and Human Services (DHHS) has determined that lead and lead compounds are reasonably anticipated to be human carcinogens and the EPA has determined that lead is a probable human carcinogen. The International Agency for Research on

September 2005

CAS # 7439-92-1

LEAD



LEAD CAS # 7439-92-1

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Cancer (IARC) has determined that inorganic lead is probably carcinogenic to humans and that there is insufficient information to determine whether organic lead compounds will cause cancer in humans.

How can lead affect children?

Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint, or swallowing house dust or soil that contains lead.

Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth.

Exposure to lead is more dangerous for young and unborn children. Unborn children can be exposed to lead through their mothers. Harmful effects include premature births, smaller babies, decreased mental ability in the infant, learning difficulties, and reduced growth in young children. These effects are more common if the mother or baby was exposed to high levels of lead. Some of these effects may persist beyond childhood.

How can families reduce the risks of exposure to lead?

Avoid exposure to sources of lead.

□ Do not allow children to chew or mouth painted surfaces that may have been painted with lead-based paint.

□ If you have a water lead problem, run or flush water that has been standing overnight before drinking or cooking with it.

□ Some types of paints and pigments that are used as make-up or hair coloring contain lead. Keep these kinds of products away from children

□ If your home contains lead-based paint or you live in an area contaminated with lead, wash children's hands and faces often to remove lead dusts and soil, and regularly clean the house of dust and tracked in soil.

Is there a medical test to determine whether I've been exposed to lead?

A blood test is available to measure the amount of lead in your blood and to estimate the amount of your recent exposure to lead. Blood tests are commonly used to screen children for lead poisoning. Lead in teeth or bones can be measured by X-ray techniques, but these methods are not widely available. Exposure to lead also can be evaluated by measuring erythrocyte protoporphyrin (EP) in blood samples. EP is a part of red blood cells known to increase when the amount of lead in the blood is high. However, the EP level is not sensitive enough to identify children with elevated blood lead levels below about 25 micrograms per deciliter (μ g/dL). These tests usually require special analytical equipment that is not available in a doctor's office. However, your doctor can draw blood samples and send them to appropriate laboratories for analysis.

Has the federal government made

recommendations to protect human health?

The Centers for Disease Control and Prevention (CDC) recommends that states test children at ages 1 and 2 years. Children should be tested at ages 3-6 years if they have never been tested for lead, if they receive services from public assistance programs for the poor such as Medicaid or the Supplemental Food Program for Women, Infants, and Children, if they live in a building or frequently visit a house built before 1950; if they visit a home (house or apartment) built before 1978 that has been recently remodeled; and/or if they have a brother, sister, or playmate who has had lead poisoning. CDC considers a lead level of 10 μ g/dL to be a level of concern for children.

EPA limits lead in drinking water to 15 µg per liter. **References**

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological Profile for lead (Draft for Public Comment). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, Public Health Service Agency for Toxic Substances and Disease Registry

Division of Toxicology ToxFAQsTM

This fact sheet answers the most frequently asked health questions (FAQs) about nickel. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Nickel is a naturally occurring element. Pure nickel is a hard, silvery-white metal used to make stainless steel and other metal alloys. Skin effects are the most common effects in people who are sensitive to nickel. Workers who breathed very large amounts of nickel compounds developed chronic bronchitis and lung and nasal sinus cancers. Nickel has been found in at least 882 of the 1,662 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is nickel?

Nickel is a very abundant natural element. Pure nickel is a hard, silvery-white metal. Nickel can be combined with other metals, such as iron, copper, chromium, and zinc, to form alloys. These alloys are used to make coins, jewelry, and items such as valves and heat exchangers. Most nickel is used to make stainless steel.

Nickel can combine with other elements such as chlorine, sulfur, and oxygen to form nickel compounds. Many nickel compounds dissolve fairly easy in water and have a green color. Nickel compounds are used for nickel plating, to color ceramics, to make some batteries, and as substances known as catalysts that increase the rate of chemical reactions. Nickel is found in all soil and is emitted from volcanoes. Nickel is also found in meteorites and on the ocean floor. Nickel and its compounds have no characteristic odor or taste.

What happens to nickel when it enters the environment?

Nickel is released into the atmosphere by industries that make or use nickel, nickel alloys, or nickel compounds. It is also released into the atmosphere by oil-burning power plants, coal-burning power plants, and trash incinerators.
In the air, it attaches to small particles of dust that settle to the ground or are taken out of the air in rain or snow; this usually takes many days.

□ Nickel released in industrial waste water ends up in soil or sediment where it strongly attaches to particles containing iron or manganese.

□ Nickel does not appear to accumulate in fish or in other animals used as food.

How might I be exposed to nickel?

□ By eating food containing nickel, which is the major source of exposure for most people.

□ By skin contact with soil, bath or shower water, or metals containing nickel, as well as by handling coins or touching jewelry containing nickel.

D By drinking water that contains small amounts of nickel.

□ By breathing air or smoking tobacco containing nickel.

□ Higher exposure may occur if you work in industries that process or use nickel.

How can nickel affect my health?

The most common harmful health effect of nickel in humans is an allergic reaction. Approximately 10-20% of the population is sensitive to nickel. People can become sensitive to nickel when jewelry or other things containing it are in direct contact with the skin for a long time. Once a person is sensitized to nickel, further contact with the metal may produce a reaction. The most common reaction is a skin rash at the site of contact. The skin rash may also

August 2005

NICKEL

CAS # 7440-02-0



NICKEL CAS # 7440-02-0

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occur at a site away from the site of contact. Less frequently, some people who are sensitive to nickel have asthma attacks following exposure to nickel. Some sensitized people react when they consume food or water containing nickel or breathe dust containing it.

People working in nickel refineries or nickel-processing plants have experienced chronic bronchitis and reduced lung function. These persons breathed amounts of nickel much higher than levels found normally in the environment. Workers who drank water containing high amounts of nickel had stomach ache and suffered adverse effects to their blood and kidneys.

Damage to the lung and nasal cavity has been observed in rats and mice breathing nickel compounds. Eating or drinking large amounts of nickel has caused lung disease in dogs and rats and has affected the stomach, blood, liver, kidneys, and immune system in rats and mice, as well as their reproduction and development.

How likely is nickel to cause cancer?

Cancers of the lung and nasal sinus have resulted when workers breathed dust containing high levels of nickel compounds while working in nickel refineries or nickel processing plants. The Department of Health and Human Services (DHHS) has determined that nickel metal may reasonably be anticipated to be a carcinogen and that nickel compounds are known human carcinogens. The International Agency for Research on Cancer (IARC) has determined that some nickel compounds are carcinogenic to humans and that metallic nickel may possibly be carcinogenic to humans. The EPA has determined that nickel refinery dust and nickel subsulfide are human carcinogens.

How can nickel affect children?

It is likely that the health effects seen in children exposed to nickel will be similar to those seen in adults. We do not know whether children differ from adults in their susceptibility to nickel. Human studies that examined whether nickel can harm the fetus are inconclusive. Animal studies have found increases in newborn deaths and decreased newborn weight after ingesting very high amounts of nickel. Nickel can be transferred from the mother to an infant in breast milk and can cross the placenta.

How can families reduce the risks of exposure to nickel?

□ Avoiding jewelry containing nickel will eliminate risks of exposure to this source of the metal.

 \Box Exposures of the general population from other sources, such as foods and drinking water, are almost always too low to be of concern.

Is there a medical test to determine whether I've been exposed to nickel?

There are tests available to measure nickel in your blood, feces, and urine. More nickel was measured in the urine of workers who were exposed to nickel compounds that dissolve easily in water than in the urine of workers exposed to nickel compounds that are hard to dissolve. This means that it is easier to tell if you have been exposed to soluble nickel compounds than less-soluble compounds. The nickel measurements do not accurately predict potential health effects from exposure to nickel.

Has the federal government made recommendations to protect human health?

The EPA recommends that drinking water should contain no more than 0.1 milligrams of nickel per liter of water (0.1 mg/L). To protect workers, the Occupational Safety and Health Administration (OSHA) has set a limit of 1 mg of nickel per cubic meter of air (1 mg/m³) for metallic nickel and nickel compounds in workplace air during an 8-hour workday, 40-hour workweek.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological Profile for Nickel (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.





POLYCHLORINATED BIPHENYLS

Division of Toxicology ToxFAQsTM

February 2001

This fact sheet answers the most frequently asked health questions (FAQs) about polychlorinated biphenyls. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Polychlorinated biphenyls (PCBs) are a mixture of individual chemicals which are no longer produced in the United States, but are still found in the environment. Health effects that have been associated with exposure to PCBs include acne-like skin conditions in adults and neurobehavioral and immunological changes in children. PCBs are known to cause cancer in animals. PCBs have been found in at least 500 of the 1,598 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What are polychlorinated biphenyls?

Polychlorinated biphenyls are mixtures of up to 209 individual chlorinated compounds (known as congeners). There are no known natural sources of PCBs. PCBs are either oily liquids or solids that are colorless to light yellow. Some PCBs can exist as a vapor in air. PCBs have no known smell or taste. Many commercial PCB mixtures are known in the U.S. by the trade name Aroclor.

PCBs have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment because they don't burn easily and are good insulators. The manufacture of PCBs was stopped in the U.S. in 1977 because of evidence they build up in the environment and can cause harmful health effects. Products made before 1977 that may contain PCBs include old fluorescent lighting fixtures and electrical devices containing PCB capacitors, and old microscope and hydraulic oils.

What happens to PCBs when they enter the environment?

□ PCBs entered the air, water, and soil during their manufacture, use, and disposal; from accidental spills and leaks during their transport; and from leaks or fires in products containing PCBs.

□ PCBs can still be released to the environment from hazardous waste sites; illegal or improper disposal of industrial wastes and consumer products; leaks from old electrical transformers containing PCBs; and burning of some wastes in incinerators.

□ PCBs do not readily break down in the environment and thus may remain there for very long periods of time. PCBs can travel long distances in the air and be deposited in areas far away from where they were released. In water, a small amount of PCBs may remain dissolved, but most stick to organic particles and bottom sediments. PCBs also bind strongly to soil.

□ PCBs are taken up by small organisms and fish in water. They are also taken up by other animals that eat these aquatic animals as food. PCBs accumulate in fish and marine mammals, reaching levels that may be many thousands of times higher than in water.

How might I be exposed to PCBs?

□ Using old fluorescent lighting fixtures and electrical devices and appliances, such as television sets and refrigerators, that were made 30 or more years ago. These items may leak small amounts of PCBs into the air when they get hot during operation, and could be a source of skin exposure.

□ Eating contaminated food. The main dietary sources of PCBs are fish (especially sportfish caught in contaminated lakes or rivers), meat, and dairy products.

□ Breathing air near hazardous waste sites and drinking contaminated well water.

□ In the workplace during repair and maintenance of PCB transformers; accidents, fires or spills involving transformers, fluorescent lights, and other old electrical devices; and disposal of PCB materials.

How can PCBs affect my health?

The most commonly observed health effects in people exposed to large amounts of PCBs are skin conditions such as acne and rashes. Studies in exposed workers have shown changes in blood and urine that may indicate liver damage. PCB exposures in the general population are not likely to result in skin and liver effects. Most of the studies of health effects of PCBs in the general population examined children of mothers who were exposed to PCBs.

Animals that ate food containing large amounts of PCBs for short periods of time had mild liver damage and some died. Animals that ate smaller amounts of PCBs in food over several weeks or months developed various kinds of health effects, including anemia; acne-like skin conditions; and liver, stomach, and thyroid gland injuries. Other effects

Page 2 POLYCHLORINATED BIPHENYLS

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of PCBs in animals include changes in the immune system, behavioral alterations, and impaired reproduction. PCBs are not known to cause birth defects.

How likely are PCBs to cause cancer?

Few studies of workers indicate that PCBs were associated with certain kinds of cancer in humans, such as cancer of the liver and biliary tract. Rats that ate food containing high levels of PCBs for two years developed liver cancer. The Department of Health and Human Services (DHHS) has concluded that PCBs may reasonably be anticipated to be carcinogens. The EPA and the International Agency for Research on Cancer (IARC) have determined that PCBs are probably carcinogenic to humans.

How can PCBs affect children?

Women who were exposed to relatively high levels of PCBs in the workplace or ate large amounts of fish contaminated with PCBs had babies that weighed slightly less than babies from women who did not have these exposures. Babies born to women who ate PCBcontaminated fish also showed abnormal responses in tests of infant behavior. Some of these behaviors, such as problems with motor skills and a decrease in short-term memory, lasted for several years. Other studies suggest that the immune system was affected in children born to and nursed by mothers exposed to increased levels of PCBs. There are no reports of structural birth defects caused by exposure to PCBs or of health effects of PCBs in older children. The most likely way infants will be exposed to PCBs is from breast milk. Transplacental transfers of PCBs were also reported In most cases, the benefits of breastfeeding outweigh any risks from exposure to PCBs in mother's milk.

How can families reduce the risk of exposure to PCBs?

You and your children may be exposed to PCBs by eating fish or wildlife caught from contaminated locations. Certain states, Native American tribes, and U.S. territories have issued advisories to warn people about PCB-contaminated fish and fish-eating wildlife. You can reduce your family's exposure to PCBs by obeying these advisories.
Children should be told not play with old appliances,

electrical equipment, or transformers, since they may contain PCBs.

Children should be discouraged from playing in the dirt near hazardous waste sites and in areas where there was a transformer fire. Children should also be discouraged from eating dirt and putting dirty hands, toys or other objects in their mouths, and should wash hands frequently.
If you are exposed to PCBs in the workplace it is possible to carry them home on your clothes, body, or tools. If this is the case, you should shower and change clothing before leaving work, and your work clothes should be kept separate from other clothes and laundered separately.

Is there a medical test to show whether I've been exposed to PCBs?

Tests exist to measure levels of PCBs in your blood, body fat, and breast milk, but these are not routinely conducted. Most people normally have low levels of PCBs in their body because nearly everyone has been environmentally exposed to PCBs. The tests can show if your PCB levels are elevated, which would indicate past exposure to above-normal levels of PCBs, but cannot determine when or how long you were exposed or whether you will develop health effects.

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 0.0005 milligrams of PCBs per liter of drinking water (0.0005 mg/L). Discharges, spills or accidental releases of 1 pound or more of PCBs into the environment must be reported to the EPA. The Food and Drug Administration (FDA) requires that infant foods, eggs, milk and other dairy products, fish and shellfish, poultry and red meat contain no more than 0.2-3 parts of PCBs per million parts (0.2-3 ppm) of food. Many states have established fish and wildlife consumption advisories for PCBs.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for polychlorinated biphenyls (PCBs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQsTM Internet address is http://www.atsdr.cdc.gov/toxfaq.html . ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, Public Health Service Agency for Toxic Substances and Disease Registry

Division of Toxicology ToxFAQsTM

This fact sheet answers the most frequently asked health questions (FAQs) about selenium. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other <u>chemicals are present</u>.

HIGHLIGHTS: People may be exposed to low levels of selenium daily through food and water. Selenium is a trace mineral needed in small amounts for good health, but exposure to much higher levels can result in neurological effects and brittle hair and deformed nails. Occupational inhalation exposure to selenium vapors may cause dizziness, fatigue, irritation of mucous membranes, and respiratory effects. This substance has been found in at least 508 of the 1,636 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is selenium?

Selenium is a naturally occurring mineral element that is distributed widely in nature in most rocks and soils. In its pure form, it exists as metallic gray to black hexagonal crystals, but in nature it is usually combined with sulfide or with silver, copper, lead, and nickel minerals. Most processed selenium is used in the electronics industry, but it is also used: as a nutritional supplement; in the glass industry; as a component of pigments in plastics, paints, enamels, inks, and rubber; in the preparation of pharmaceuticals; as a nutritional feed additive for poultry and livestock; in pesticide formulations; in rubber production; as an ingredient in antidandruff shampoos; and as a constituent of fungicides. Radioactive selenium is used in diagnostic medicine.

What happens to selenium when it enters the environment?

Selenium occurs naturally in the environment and can be released by both natural and manufacturing processes.
Selenium dust can enter the air from burning coal and oil. This selenium dust will eventually settle over the land and water.

□ It also enters water from rocks and soil, and from agricultural and industrial waste. Some selenium compounds will dissolve in water, and some will settle to the bottom as particles.

□ Insoluble forms of selenium will remain in soil, but soluble forms are very mobile and may enter surface water from soils.

□ Selenium may accumulate up the food chain.

How might I be exposed to selenium?

 \Box The general population is exposed to very low levels of selenium in air, food, and water. The majority of the daily intake comes from food.

□ People working in or living near industries where selenium is produced, processed, or converted into commercial products may be exposed to higher levels of selenium in the air.

□ People living in the vicinity of hazardous waste sites or coal burning plants may also be exposed to higher levels of selenium.

How can selenium affect my health?

Selenium has both beneficial and harmful effects. Low doses of selenium are needed to maintain good health. However, exposure to high levels can cause adverse health effects. Short-term oral exposure to high concentrations of selenium may cause nausea, vomiting, and diarrhea. Chronic oral exposure to high concentrations of selenium compounds can produce a disease called selenosis. The major signs of selenosis are hair loss, nail brittleness, and neurological abnormalities (such as numbness and other odd sensations

September 2003

SELENIUM

CAS # 7782-49-2

ATSDR AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

SELENIUM CAS # 7782-49-2

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in the extremities).

Brief exposures to high levels of elemental selenium or selenium dioxide in air can result in respiratory tract irritation, bronchitis, difficulty breathing, and stomach pains. Longerterm exposure to either of these air-borne forms can cause respiratory irritation, bronchial spasms, and coughing. Levels of these forms of selenium that would be necessary to produce such effects are normally not seen outside of the workplace.

Animal studies have shown that very high amounts of selenium can affect sperm production and the female reproductive cycle. We do not know if similar effects would occur in humans.

How likely is selenium to cause cancer?

Studies of laboratory animals and people show that most selenium compounds probably do not cause cancer. In fact, studies in humans suggest that lower-than-normal selenium levels in the diet might increase the risk of cancer. The International Agency for Research on Cancer (IARC) has determined that selenium and selenium compounds are not classifiable as to their carcinogenicity to humans. The EPA has determined that one specific form of selenium, selenium sulfide, is a probable human carcinogen. Selenium sulfide is not present in foods and is a very different chemical from the organic and inorganic selenium compounds found in foods and in the environment.

How can selenium affect children?

It is likely that the health effects seen in children exposed to selenium will be similar to the effects seen in adults. However, one study found that children may be less susceptible to the health effects of selenium than adults. Selenium compounds have not been shown to cause birth defects in humans or in other mammals.

How can families reduce the risk of exposure to selenium?

□ Certain dietary supplements and shampoos contain selenium; these should be used according to the

manufacturer's directions.

□ Children living near waste sites that contain selenium or coal burning plants should be encouraged to wash their hands before eating and to avoid putting their unwashed hands in their mouths.

Is there a medical test to show whether I've been exposed to selenium?

Low levels of selenium are normally found in body tissues and urine. Blood and urine tests for selenium are most useful for people who have recently been exposed to high levels. Toenail clippings can be used to determine longerterm exposure. These tests are not usually available at your doctor's office, but your doctor can send the samples to a laboratory that can perform the tests. None of these tests, however, can predict whether you will experience any health effects.

Has the federal government made recommendations to protect human health?

The EPA restricts the amount of selenium allowed in public water supplies to 50 parts total selenium per billion parts of water (50 ppb).

The Occupational Safety and Health Administration (OSHA) sets a limit of 0.2 mg selenium/m³ of workroom air for an 8-hour work shift.

ATSDR and the EPA have determined that 5 micrograms of selenium per kilogram of body weight taken daily would not be expected to cause any adverse health effects over a lifetime of such intake.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological Profile for Selenium (Update) Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

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Division of Toxicology ToxFAQsTM

This fact sheet answers the most frequently asked health questions (FAQs) about zinc. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Zinc is a naturally occurring element. Exposure to high levels of zinc occurs mostly from eating food, drinking water, or breathing workplace air that is contaminated. Low levels of zinc are essential for maintaining good health. Exposure to large amounts of zinc can be harmful. It can cause stomach cramps, anemia, and changes in cholesterol levels. Zinc has been found in at least 985 of the 1,662 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is zinc?

Zinc is one of the most common elements in the earth's crust. It is found in air, soil, and water, and is present in all foods. Pure zinc is a bluish-white shiny metal.

Zinc has many commercial uses as coatings to prevent rust, in dry cell batteries, and mixed with other metals to make alloys like brass, and bronze. A zinc and copper alloy is used to make pennies in the United States.

Zinc combines with other elements to form zinc compounds. Common zinc compounds found at hazardous waste sites include zinc chloride, zinc oxide, zinc sulfate, and zinc sulfide. Zinc compounds are widely used in industry to make paint, rubber, dyes, wood preservatives, and ointments.

What happens to zinc when it enters the environment?

□ Some is released into the environment by natural processes, but most comes from human activities like mining, steel production, coal burning, and burning of waste.

 \Box It attaches to soil, sediments, and dust particles in the air.

□ Rain and snow remove zinc dust particles from the air.

Depending on the type of soil, some zinc compounds can move into the groundwater and into lakes, streams, and rivers.

 $\hfill\square$ Most of the zinc in soil stays bound to soil particles and

does not dissolve in water.

 \Box It builds up in fish and other organisms, but it does not build up in plants.

How might I be exposed to zinc?

Ingesting small amounts present in your food and water.
Drinking contaminated water or a beverage that has been stored in metal containers or flows through pipes that have been coated with zinc to resist rust.

Eating too many dietary supplements that contain zinc.
Working on any of the following jobs: construction, painting, automobile mechanics, mining, smelting, and welding; manufacture of brass, bronze, or other zinc-containing alloys; manufacture of galvanized metals; and manufacture of machine parts, rubber, paint, linoleum, oilcloths, batteries, some kind of glass, ceramics, and dyes.

How can zinc affect my health?

Zinc is an essential element in our diet. Too little zinc can cause problems, but too much zinc is also harmful.

Harmful effects generally begin at levels 10-15 times higher than the amount needed for good health. Large doses taken by mouth even for a short time can cause stomach cramps, nausea, and vomiting. Taken longer, it can cause anemia and decrease the levels of your good cholesterol. We do not know if high levels of zinc affect reproduction in humans. Rats that were fed large amounts of zinc became infertile.

August 2005

CAS # 7440-66-6

ZINC



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Inhaling large amounts of zinc (as dusts or fumes) can cause a specific short-term disease called metal fume fever. We do not know the long-term effects of breathing high levels of zinc.

Putting low levels of zinc acetate and zinc chloride on the skin of rabbits, guinea pigs, and mice caused skin irritation. Skin irritation will probably occur in people.

How likely is zinc to cause cancer?

The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not classified zinc for carcinogenicity. Based on incomplete information from human and animal studies, the EPA has determined that zinc is not classifiable as to its human carcinogenicity.

How can zinc affect children?

Zinc is essential for proper growth and development of young children. It is likely that children exposed to very high levels of zinc will have similar effects as adults. We do not know whether children are more susceptible to the effects of excessive intake of zinc than the adults.

We do not know if excess zinc can cause developmental effects in humans. Animal studies have found decreased weight in the offspring of animals that ingested very high amounts of zinc.

How can families reduce the risks of exposure to zinc?

□ Children living near waste sites that contain zinc may be exposed to higher levels of zinc through breathing contaminated air, drinking contaminated drinking water, touching or eating contaminated soil.

□ Discourage your children from eating soil or putting their hands in their mouths and teach them to wash their hands frequently and before eating.

□ If you use medicines or vitamin supplements containing

zinc, make sure you use them appropriately and keep them out of the reach of children.

Is there a medical test to determine whether I've been exposed to zinc?

There are tests available to measure zinc in your blood, urine, hair, saliva, and feces. These tests are not usually done in the doctor's office because they require special equipment. High levels of zinc in the feces can mean high recent zinc exposure. High levels of zinc in the blood can mean high zinc consumption and/or high exposure. Tests to measure zinc in hair may provide information on long-term zinc exposure; however, the relationship between levels in your hair and the amount of zinc you were exposed to is not clear.

Has the federal government made recommendations to protect human health?

The EPA recommends that drinking water should contain no more than 5 milligrams per liter of water (5 mg/L) because of taste. The EPA requires that any release of 1,000 pounds (or in some cases 5,000 pounds) into the environment be reported to the agency.

To protect workers, the Occupational Safety and Health Administration (OSHA) has set an average limit of 1 mg/m^3 for zinc chloride fumes and 5 mg/m^3 for zinc oxide (dusts and fumes) in workplace air during an 8-hour workday, 40-hour workweek.

Similarly, the National Institute for Occupational Safety and Health (NIOSH) has set the same standards for up to a 10-hour workday over a 40-hour workweek.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological Profile for Zinc (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

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Appendix H

Risk Calculation Spreadsheets

AP = averaging period; acute: 1 day, chronic: 2555 days, cancer: 25550 days

PM10 = concentration of PM10 in air. 60 ug/m3 is the default value for construction sites (MA DEP 2002). %PM10 in lungs = percentage of inhaled PM10 that reaches the lungs, conservatively assumed 100% RAF = relative absorption factor, conservatively assumed 100%

ADE = average daily exposure, ug/m3 CSF = oral cancer slope factor, risk per mg/kg-day Unit Risk = inhalation unit risk, risk per ug/m3 RfD = Reference Dose, ug/kg-day HI - Hazard Index, site dose/reference dose SA - Skin surface area exposed to soil, (EPA 2004) Onsite worker = 3300 cm2 (head, hands, forearms). Trespasser = 4508 cm2 (head, hands, forearms, feet) ATV Driver = 4508 cm2 (head, hands, forearms, feet) AF = Skin-soil adherence factor (EPA 2004), 0.2 mg/cm2 ABSd = dermal absorption fraction from soil; EPA 2001(arsenic, cadmium, PCBs) and CA 1994 (default for other metals)

Sources of CSF, RfD and unit risk values used in risk calculations

Contaminant	oral RfD (mg/kg-day)	Source	oral CSF (mg/kg-d) ⁻¹	Source	Unit Risk (ug/m3) ⁻¹	Source	Acute (mg/kg-day)	Source
Nickel	0.02	EPA IRIS		EPA IRIS	4.80E-04	EPA IRIS		
			nickel subsulfide					
Chromium 6	0.003	EPA IRIS			0.15	CA OEHHA		
Chromium 3	1.5	EPA IRIS						
Cadmium	0.001 (soil)	EPA IRIS	0.38	CA OEHHA	4.20E-03	CA OEHHA		
Selenium	0.005	EPA IRIS						
Zinc	0.3	EPA IRIS						
PCBs*	2.00E-05	EPA IRIS	2	EPA IRIS	1.00E-04	EPA IRIS		
Cyanide	0.02	EPA IRIS						
Arsenic	3.00E-04	1 EPA IRIS	1.5	5 EPA IRIS	0.0043	B EPA IRIS	0.005 N	IRL, ATSDR
Antimony	4.00E-04	1 EPA IRIS						

*RfD is for Aroclor 1254

RISKS FROM LEAD EXPOSURE

Soil lead concentration * lead slope factor = estimated blood lead level

1386 mg/kg * 0.001 ug/dL oer mg/kg = 1.3 ug/dL

Appendix I

ATSDR Public Health Hazard Conclusion Categories

ATSDR PUBLIC HEALTH HAZARD CATEGORIES

Category / definition	Criteria	ASTDR Actions
<i>A. Urgent Public Health Hazard</i> This category is used for sites where short- term exposures (< 1 year) to hazardous substances or conditions could result in adverse health effects that require rapid intervention.		ATSDR will expeditiously issue a health advisory that includes strong recommendations to immediately stop or reduce exposure to mitigate the health risks posed by the site.
This determination represents a professional judgment based on critical data which ATSDR has judged sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.		
<i>B. Public Health Hazard</i> This category is used for sites that pose a public health hazard due to the existence of long-term exposures (> 1 year) to hazardous substance or conditions that could result in adverse health effects. This determination represents a professional judgment based on critical data which ATSDR has judged sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.	Evaluation of available relevant information* suggests that, under site- specific conditions of exposure, long-term exposures to site-specific contaminants (including radionuclides) have had, are having, or are likely to have in the future, an adverse impact on human health that requires one or more public health interventions. Such site-specific exposures may include the presence of serious physical or safety hazards.	ATSDR will make recommendations to stop or reduce exposure in a timely manner to mitigate the health risks posed by the site.
<i>C. Indeterminate Public Health Hazard</i> This category is used for sites in which "critical" data are insufficient with regard to extent of exposure and/or toxicologic properties at estimated exposure levels. This determination represents a professional judgment that critical data are missing and ATSDR has judged the data are insufficient to support a decision. This does not necessarily imply all data are required to support a decision.	The health assessor must determine, using professional judgment, the "criticality" of such data and the likelihood that the data can be obtained and will be obtained in a timely manner. Where some data are available, even limited data, the health assessor is encouraged to the extent possible to select other hazard categories and to support their decision with clear narrative that explains the limits of the data and the rationale for the decision.	ATSDR will make recommendations in the public health assessment to identify the data or information needed to adequately assess the public health risks posed by the site.

Category / definition	Criteria	ASTDR Actions
D. No Apparent Public Health Hazard This category is used for sites where human exposure to contaminated media may be occurring, may have occurred in the past, and/or may occur in the future, but the exposure is not expected to cause any adverse health effects. This determination represents a professional	Evaluation of available relevant information* indicates that, under site- specific conditions of exposure, exposures to site-specific contaminants in the past, present, or future are not likely to result in any adverse impact on human health.	
judgment based on critical data which ATSDR considers sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.		
E: No Public Health Hazard		
This category is used for sites that, because of the absence of exposure, do NOT pose a public health hazard.	Sufficient evidence indicates that no human exposures to contaminated media have occurred, no exposures are currently occurring, and exposures are not likely to occur in the future.	
Sufficient evidence indicates that no human exposures to contaminated media have occurred, none are now occurring, and none are likely to occur in the future		

* Such as environmental and demographic data; health outcome data; exposure data; community health concerns information; toxicologic, medical, and epidemiologic data; monitoring and management plans