TOXICOLOGICAL PROFILE FOR ANTIMONY AND COMPOUNDS

Agency for Toxic Substances and Disease Registry
U.S. Public Health Service

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FOREWORD

The Superfund Amendments and Reauthorization Act (SARA) of 1986 (Public Law 99-499) extended and amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund). This public law directed the Agency for Toxic Substances and Disease Registry (ATSDR) to prepare toxicological profiles for hazardous substances which are most commonly found at facilities on the CERCLA National Priorities List and which pose the most significant potential threat to human health, as determined by ATSDR and the Environmental Protection Agency (EPA). The lists of the 250 most significant hazardous substances were published in the Federal Resister on April 17, 1987; on October 20, 1988; on October 26, 1989; and on October 17, 1990. A revised list of 275 substances was published on October 17, 1991.

Section 104(i)(3) of CERCLA, as amended, directs the Administrator of ATSDR to prepare a toxicological profile for each substance on the lists. Each profile must include the following content:

(A) An examination, summary, and interpretation of available toxicological information and epidemiological evaluations on the hazardous substance in order to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects.

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure which present a significant risk to human health of acute, subacute, and chronic health effects.

(C) Where appropriate, an identification of toxicological testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

This toxicological profile is prepared in accordance with guidelines developed by ATSDR and EPA. The original guidelines were published in the Federal Register on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile is intended to characterize succinctly the toxicological and adverse health effects information for the hazardous substance being described. Each profile identifies and reviews the key literature (that has been peer-reviewed) that describes a hazardous substance's toxicological properties. Other pertinent literature is also presented but described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

Foreword

Each toxicological profile begins with a public health statement, which describes in nontechnical language a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health will be identified by ATSDR, the National Toxicology Program (NTP) of the Public Health Service, and EPA. The focus of the profiles is on health and toxicological information; therefore, we have included this information in the beginning of the document.

The principal audiences for the toxicological profiles are health professionals at the federal, state, and local levels, interested private sector organizations and groups, and members of the public.

This profile reflects our assessment of all relevant toxicological testing and information that has been peer reviewed. It has been reviewed by scientists from ATSDR, the Centers for Disease Control, the NTP, and other federal agencies. It has also been reviewed by a panel of nongovernment peer reviewers. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.

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1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about antimony and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,177 sites on its National Priorities List (NPL). Antimony and its compounds have been found at 52 of these sites. However, we do not know how many of the 1,177 NPL sites have been evaluated for antimony. As EPA evaluates more sites, the number of sites at which antimony and its compounds are found may change. The information is important for you because antimony may cause harmful health effects and because these sites are potential or actual sources of human exposure to antimony.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous substance such as antimony, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS ANTIMONY?

Antimony is a silvery white metal of medium hardness that breaks easily. Small amounts of antimony are found in the earth's crust. Antimony ores are mined and then either changed into antimony metal or combined with oxygen to form antimony oxide.

Antimony oxide is a white powder that does not evaporate. Only a small amount of it will dissolve in water. Most antimony oxide produced is added to textiles and plastics to prevent their catching on fire.

Antimony metal is too easily broken to be used much by itself. To make it stronger, a little antimony is usually mixed with other metals such as lead and zinc to form mixtures of metals called alloys. These alloys are used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, type metal, ammunition, and pewter.

Antimony enters the environment during the mining and processing of its ores and in the production of antimony metal, alloys, antimony oxide, and

combinations of antimony with other substances. Little or no antimony is mined in the United States, Antimony ore and impure metals are brought into this country from other countries for processing. Small amounts of antimony are also released into the environment by incinerators and coal-burning power plants. The antimony that comes out of the smoke stacks of these plants is attached to very small particles that settle to the ground or are washed out of the air by rain. It usually takes many days for antimony to be removed from the air. Antimony attached to very small particles may stay in the air for more than a month. Antimony cannot be destroyed in the environment. It can only change its form or become attached to or separated from particles. Most antimony will end up in the soil or sediment, where it attaches strongly to particles that contain iron, manganese, or aluminum. For more information, see Chapters 3, 4, and 5.

1.2 HOW HIGHT I BE EXPOSED TO ANTIMONY?

Antimony is found at very low levels in the environment, so low that we often cannot measure it. You may be exposed to antimony by breathing air, drinking water, and eating foods that contain it. You also may be exposed by skin contact with soil, water, and other substances that contain antimony. The analytical methods used by scientists testing for the presence of antimony in the environment do not determine the specific form of antimony present. Therefore, we do not always know what form of antimony persons may be exposed to. Similarly, we do not know what forms of antimony are found in hazardous waste sites. Much of the antimony found in sediment, soil, and rock is so strongly attached to dust and dirt or buried in minerals that it cannot easily affect your health. Some antimony in the environment is less tightly attached to particles and may be taken up by plants and animals.

The concentration of antimony in air ranges from a very small part of a nanogram (1 nanogram equals a billionth of a gram) in a cubic meter (m^3) of air (ng/m^3) to about 170 ng/m^3. However, near companies that change antimony ores into metal or make antimony oxide, concentrations may be more than 1,000 ng/m^3. You may breathe high levels of antimony in dust if you live or work near antimony mines or processing companies.

The concentration of antimony that is dissolved in rivers and lakes is very low, usually less than 5 parts of antimony in 1 billion parts of water (ppb) . We cannot measure such small amounts without special equipment. Antimony does not appear to accumulate in fish and other aquatic animals. The concentration of antimony dissolved in one polluted river where wastes from antimony mining and processing had been dumped was as high as 8 ppb. Most of the antimony in the river, however, was not dissolved, but was attached to particles of dirt. Although antimony is used in solder for water pipes, it does not seem to get into the drinking water.

Soil usually contains very low concentrations of antimony, less than 1 part of antimony in a million parts of soil (ppm). However, concentrations

close to 9 ppm have been found. The highest soil concentrations found at hazardous waste sites on the NPL and at antimony-processing sites range from 109 to 2,550 ppm. High concentrations of antimony may be found in soil because dust sent out during processing settles out from the air. Also, waste from antimony-processing and other antimony-using industries is usually dumped onto the soil. We do not know the form of antimony at these sites. However, we know that much of the antimony in antimony-processing wastes is strongly attached to soil. You may be exposed to this antimony by skin contact. Children may also be exposed to this antimony by eating the dirt.

Food usually contains small amounts of antimony. You eat and drink about 5 micrograms (5 millionths of a gram) of antimony every day. The average concentration of antimony in meats, vegetables, and seafood is 0.2-1.1 ppb. The antimony oxide that is added to many materials for fire protection is very tightly attached to these materials and does not expose people to antimony.

You may also be exposed to antimony in the workplace. If you work in industries that process antimony ore and metal or make chemicals that contain antimony, such as antimony oxide, you may be exposed to antimony by breathing dust or by skin contact.

For more information on how you may be exposed to antimony, see Chapter 5.

1.3 HOW CAN ANTIMONY ENTER AND LEAVE MY BODY?

Antimony can enter your body when you drink water or eat food, soil, or other substances that contain antimony. Antimony can also enter your body if you breathe air or dust containing antimony. We do not know if antimony can enter your body when it is placed on your skin.

A small amount of the antimony you eat or drink enters the blood after a few hours. The amount and the form of antimony in the food or water will affect how much antimony enters your blood. After you eat or drink very large doses of antimony, you may vomit. This will prevent most of the antimony from entering through the stomach and intestines into your blood. Antimony in your lungs will enter your blood after several days or weeks. The amount of antimony that will enter your blood from your lungs is not known.

After antimony enters your blood, it goes to many parts of your body. Most of the antimony goes to the liver, lungs, intestines, and spleen. Antimony will leave your body in feces and urine over several weeks. Further information on how antimony enters and leaves your body is presented in Chapter 2.

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1.4 HOW CAN ANTIMONY AFFECT MY HEALTH?

Exposure to 9 milligrams per cubic meter of air (mg/m^3) of antimony for a long time can irritate your eyes, skin, and lungs. Breathing 2 mg/m^3 of antimony for a long time can cause problems with the lungs (pneumoconiosis) heart problems (altered electrocardiograms), stomach pain, diarrhea, vomiting and stomach ulcers. People who drank over 19 ppm of antimony once, vomited. We do not know what other health effects would occur to people who swallow antimony. We do not know if antimony can cause cancer or birth defects, or affect reproduction in humans. Antimony can have beneficial effects when used for medical reasons, It has been used as a medicine to treat people infected with parasites. Persons who have had too much of this medicine or are sensitive to it when it was injected into their blood or muscle have experienced adverse health effects. These health effects include diarrhea, joint and/or muscle pain, vomiting, problems with the blood (anemia) and heart problems (altered electrocardiograms).

Rats and guinea pigs that breathed very high levels of antimony for a short time died. Rats breathing high levels of antimony for several days had lung, heart, liver, and kidney damage. Breathing very low levels of antimony for a long time has resulted in eye irritation, hair loss, and lung damage in rats. Dogs and rats that breathed low levels of antimony for a long period had heart problems (changes in EKGs). Problems with fertility have been observed in rats that breathed very high levels of antimony for a couple of months. Lung cancer has been observed in some studies of rats breathing high concentrations of antimony. Antimony has not been classified for cancer effects by the Department of Health and Human Services, the International Agency for Research on Cancer or the Environmental Protection Agency.

Dogs that drank very high levels of antimony for several weeks lost weight and had diarrhea. Rats that drank very low levels of antimony for most of their lives died sooner than rats not drinking antimony. Rats eating high levels of antimony for a long time had liver damage and fewer red blood cells.

Rabbits that had very small amounts of antimony placed on their skin for less than 1 day had skin irritation. Small amounts of antimony placed in rabbit eyes resulted in eye irritation. Large amounts of antimony placed on rabbit's skin resulted in death.

More information on how antimony can affect your health is presented in Chapter 2.

1.5 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO ANTIMONY?

There are reliable and accurate ways of measuring antimony levels in the body. Antimony can be measured in the urine, feces, and blood for several days after exposure. High levels of antimony in these fluids will show that

you have been exposed to high levels of antimony. However, these measurements can not tell you how much antimony you have been exposed to or whether you will experience any health effects. For more information, see Chapters 2 and 6.

1.6 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

EPA has set a limit of 145 ppb in lakes and streams to protect human health from the harmful effects of antimony taken in through water and contaminated fish and shellfish. EPA has also set limits on the amount of antimony that industry can release.

The Occupational Safety and Health Administration (OSHA) has set a limit of 0.5 mg/m^3 of antimony in workroom air to protect workers during an 8-hour work shift (40-hour workweek). The National Institute of Occupational Safety and Health (NIOSH) also recommends that the concentration in workroom air be limited to 0.5 mg/m^3 for antimony, averaged over an 8-hour work shift. Further information on regulations and guidelines pertaining to antimony is provided in Chapter 7.

1.7 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns not covered here, please contact your state health or environmental department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. Such clinics specialize in recognizing, evaluating, and treating illnesses that result from exposure to hazardous substances.

2. HEALTH EFFECTS

2.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective of the toxicology of antimony and a depiction of significant exposure levels associated with various adverse health effects. It contains descriptions and evaluations of studies and presents levels of significant exposure for antimony based on toxicological studies and epidemiological investigations.

Studies in which humans or animals are exposed to various antimony compounds are discussed in this chapter. The antimony compounds include organic forms (potassium antimony tartrate, sodium antimony tartrate, antimony acetate), inorganic trivalent antimony (antimony trioxide, antimony trichloride, antimony trisulfide, stibine), pentavalent inorganic antimony (antimony pentoxide, antimony pentasulfide), antimony-containing drugs (stibocaptate, stibophen), and metallic antimony. No limitations were placed on the selection of compounds for inclusion in this toxicological profile.

2.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure--inhalation, oral, and dermal--and then by health effect--death, systemic, immunological, neurological, developmental, reproductive, genotoxic, and carcinogenic effects. These data are discussed in terms of three exposure periods--acute (less than 15 days), intermediate (15-364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELS) reflect the actual doses (levels of exposure) used in the studies. LOAELs have been classified into "less serious" or "serious" effects. These distinctions are intended to help the users of the document identify the levels of exposure at which adverse health effects start to appear. They should also help to determine whether or not the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the tables and figures may differ depending on the user's perspective. For example, physicians concerned with the interpretation of clinical findings in exposed persons may be interested in levels of exposure associated with "serious" effects. Public health officials and project managers concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAEL) or exposure

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levels below which no adverse effects (NOAEL) have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels, MRLs) may be of interest to health professionals and citizens alike.

Estimates of exposure levels posing minimal risk to humans (MRLs) have been made, where data were believed reliable, for the most sensitive noncancer effect for each exposure duration. MRLs include adjustments to reflect human variability from laboratory animal data to humans.

Although methods have been established to derive these levels (Barnes et al. 1988; EPA 1989a), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired followLng repeated acute insults, such as hypersensitivity reactions, asthma, or chronic bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs will be revised.

2.2.1 Inhalation Exposure

Health effects have been observed in humans and animals following inhalation exposure to several antimony compounds. Health effects following exposure to airborne stibine, antimony trisulfide, antimony trioxide, antimony pentoxide, antimony trichloride, antimony pentasulfide, and metallic antimony are discussed below. Of these, stibine (antimony hydride) is a naturally occurring gas; for ease of comparison, its concentrations will be expressed in units of mg/m^3 (1 ppm stibine = 5.1 mg/m^3).

2.2.1.1 Death

No studies were located regarding death in humans after inhalation exposure to antimony.

Guinea pigs exposed to approximately 37.9 mg antimony/m^3 as antimony trioxide dust for 52-125 days (Dernehl et al. 1945) or guinea pigs and rats exposed to 1,395 mg antimony/m^3 as stibine gas for 30 minutes (Price et al. 1979) died. In the Dernehl et al. (1945) study, four guinea pigs died, one animal following each of 52, 90, 98, and 125 days of exposure. Pulmonary edema was a contributing factor to the death of rats and guinea pigs exposed to stibine (Price et al. 1979). None of the rats or guinea pigs exposed to 799 mg antimony/m3 for 30 minutes died (Price et al. 1979). Lower concentrations of antimony trisulfide or antimony trioxide did not affect the survival of rats exposed for 1 year (Groth et al. 1986; Wong et al. 1979).

The highest NOAEL values and all reliable LOAEL values for death in each species and duration are presented in Table 2-l and plotted in Figure 2-l.

TABLE 2-1. Levels of Significant Exposure to Antimony - Inhalation

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (mg/m^3)	LOAEL (effect) Less serious (mg/m^3)	LOAEL (effect) Serious (mg/m^3)	Reference	Form
ACUTE EXPOSURE								
Death								
1	Rat	30 min		799		1,395 (increased mortality)	Price et al. 1979	Stibine
2	Gn pig	30 min		799		1,395 (increased mortality)	Price et al. 1979	Stibine
Systemic								
3	Rat	30 min	Resp			1,395 (pulmonary edema)	Price et al. 1979	Stibine
			Renal		799 (tubular dilation)			
4	Rabbit	5 d 7 hr/d 5 d/wk	Resp		19.94 (inflammation)		Brieger et al. 1954	Trisulfide
			Cardio			19.94 (myocardial damage altered EKG)		
			Hepatic		19.94 (parenchymatous degeneration)			
			Renal		19.94 (parenchymatous degeneration)			
5	Gn pig	30 min	Resp			1,395 (pulmonary edema)	Price et al. 1979	Stibine
			Renal		799 (tubular dilation)			
INTERMEDIATE EXPOSURE								
Systemic								
6	Rat	13 wk 6 hr/d 5 d/wk	Resp		0.92 (proliferation of macrophages)		Bio/dynamics 1985	Trioxide
			Hemato	19.61				
7	Rat	6 wk 7 hr/d 5 d/wk	Resp		2.20 (congestion)		Brieger et al. 1954	Trisulfide
			Cardio			2.20 (myocardial damage altered EKG)		
8	Rabbit	6 wk 7 hr/d 5 d/wk	Cardio			4.02 (myocardial damage altered EKG)	Brieger et al. 1954	Trisulfide

TABLE 2-1 (Continued)

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (mg/m^3)	LOAEL (effect) Less serious (mg/m^3)	LOAEL (effect) Serious (mg/m^3)	Reference	Form
9	Dog	7 wk 7 hr/d 5 d/wk	Cardio	3.81			Brieger et al. 1954	Trisulfide
10	Dog	10 wk 7 hr/d 5 d/wk	Cardio Hemato	 3.98		3.98 (myocardial damage altered EKG)	Brieger et al. 1954	Trisulfide
Developmental								
11	Rat	63-78 d 4 hr/d				209 (decreased number of offspring)	Belyaeva 1967	Trioxide
Reproductive								
12	Rat	63-78 d 4 hr/d				209 (difficulty conceiving)	Belyaeva 1967	Trioxide
CHRONIC EXPOSURE								
Death								
13	Rat	52 wk 7 hr/d 5 d/wk		17.48			Groth et al. 1986; Wong et al. 1979	Trisulfide
14	Rat	52 wk 7 hr/d 5 d/wk		36			Groth et al. 1986; Wong et al. 1979	Trioxide
Systemic								
15	Human	9-31 yr	Resp Resp		8.87 (pneumoconiosis) 8.87 (upper airway inflammation)		Potkonjak and Pavlovich 1983	Trioxide and pentoxide
16	Human	8 mo-2 yr 8 hr/d 5 d/wk	Cardio Gastro			2.15 (altered EKG, elevated blood pressure) 2.15 (ulcer)	Brieger et al. 1954	Trisulfide

TABLE 2-1 (Continued)

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (mg/m^3)	LOAEL (effect) Less serious (mg/m^3)	LOAEL (effect) Serious (mg/m^3)	Reference	Form
17	Rat	12 mo 6 hr/d 5 d/wk	Resp Gastro Musc/skel	 4.2 4.2		1.6 (focal fibrosis)	Watt 1980	Trioxide
18	Rat	1 yr 6 hr/d 5 d/wk	Resp Hemato Other	 4.01 	0.07 (chronic inflammation and proliferation of macrophages) 0.07 (hyperplasia in peribronchiolar lymph nodes)	4.01 (fibrosis)	Bio/dynamics 1990	Trioxide
19	Rat	14.5 mo 25 hr/wk	Resp			83.6 (lipoid pneumonia)	Gross et al. 1952	Trioxide
Systemic								
20	Rat	52 wk 7 hr/d 5 d/wk	Resp Cardio Hepatic Renal	 36 36 36		36 (interstitial fibrosis)	Groth et al. 1986; Wong et al. 1979	Trioxide
21	Rat	52 wk 7 hr/d 5 d/wk	Resp Cardio Hepatic Renal	 17.48 17.48 17.48		17.48 (interstitial fibrosis)	Groth et al. 1986; Wong et al. 1979	Trisulfide
22	Rat	1 yr 6 hr/d 5 d/wk	Resp Hemato	 4.2		1.6 (focal fibrosis)	Watt 1983	Trioxide
23	Pig	1 yr 6 hr/d 5 d/wk	Resp Cardio Hemato	4.2 4.2 4.2			Watt 1983	Trioxide

TABLE 2-1 (Continued)

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (mg/m^3)	LOAEL (effect) Less serious (mg/m^3)	LOAEL (effect) Serious (mg/m^3)	Reference	Form
Cancer								
24	Rat	1 yr 6 hr/d 5 d/wk				4.2 (CEL-lung neoplasms)	Watt 1983	Trioxide
25	Rat	52 wk 7 hr/d 5 d/wk				36 (CEL-lung tumor)	Groth et al. 1986; Wong et al. 1979	Trioxide
26	Rat	52 wk 7 hr/d 5 d/wk				17.48 (CEL-lung tumors)	Groth et al. 1986; Wong et al. 1979	Trisulfide

[a]The number corresponds to entries in Figure 2-1.

Cardio = cardiovascular; CEL = cancer effect level; d = day; EKG = electrocardiogram; Gastro = gastrointestinal; Gn pig = guinea pig; Hemato = hematological; hr = hour; LOAEL = lowest-observed-adverse-effect level; min = minute; mo = month; Musc/skel = muscular/skeletal; NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week; yr = year

FIGURE 2-1. Levels of Significant Exposure to Antimony - Inhalation

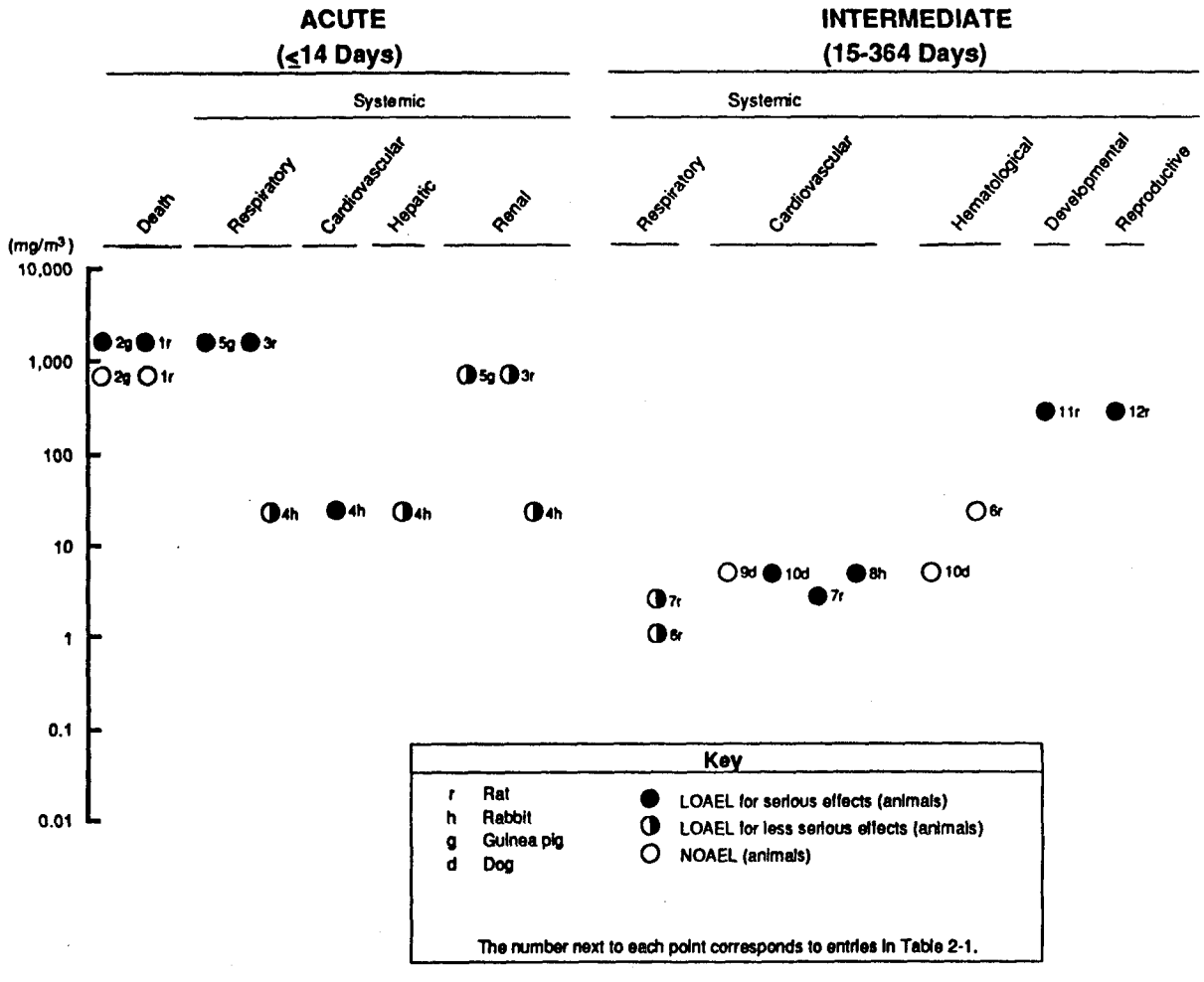
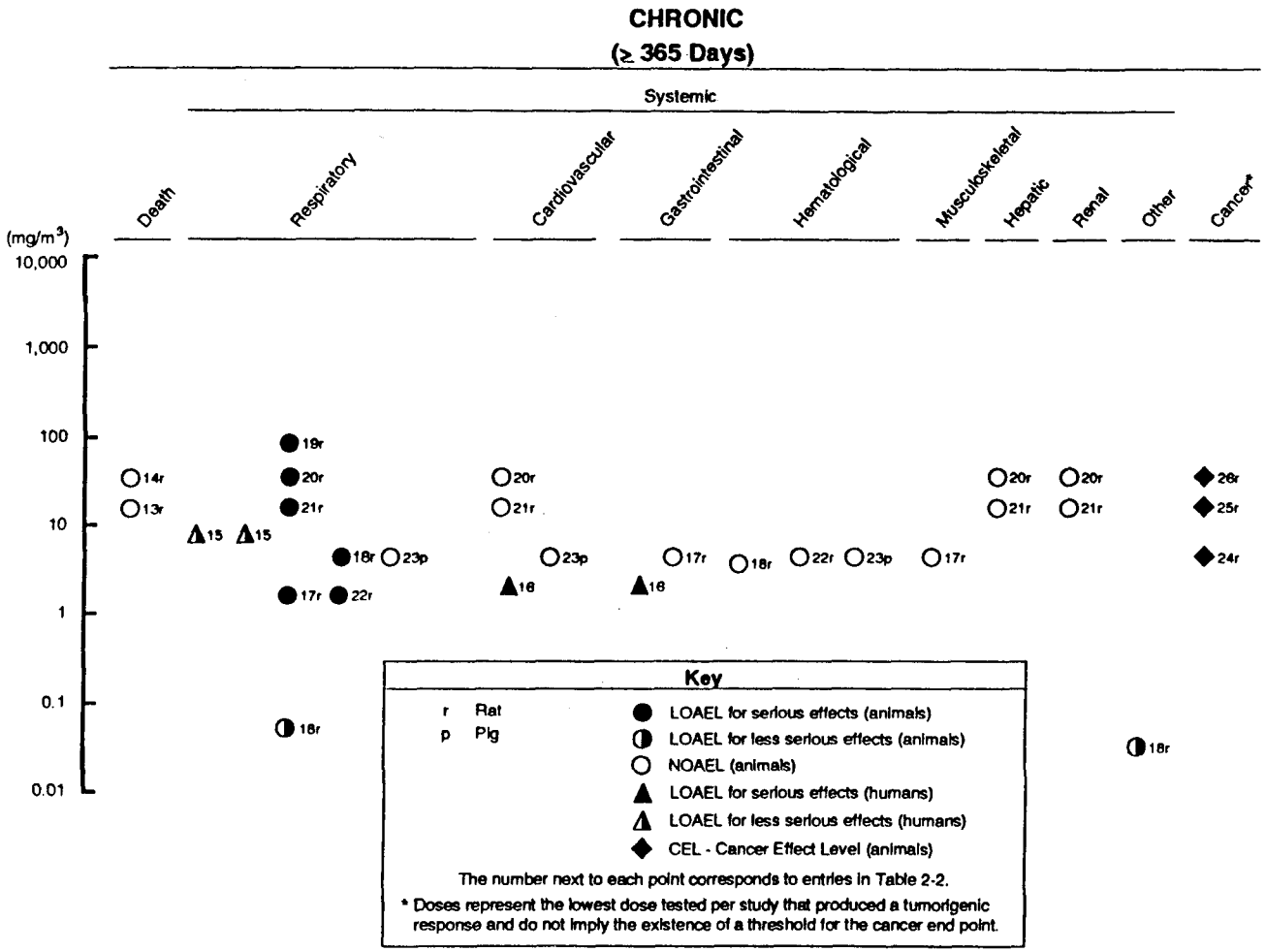


FIGURE 2-1 (Continued)



2.2.1.2 Systemic Effects

The highest NOAEL values and all reliable LOAEL values for each systemic effect in each species and duration are presented in Table 2-l and plotted in Figure 2-1.

Respiratory Effects. Occupational exposure to antimony trioxide and/or pentoxide dust (8.87 mg antimony/m^3 or greater) resulted in antimony pneumoconiosis (inflammation of the lungs due to the irritation caused by the inhalation of dust) (Cooper et al. 1968; Potkonjak and Pavlovich 1983; Renes 1953). Alterations in pulmonary function (airway obstruction, bronchospasm, and hyperinflation) have been reported in workers exposed to airborne antimony (Cooper et al. 1968; Potkonjak and Pavlovich 1983). Other respiratory effects reported in workers include chronic bronchitis, chronic emphysema, inactive tuberculosis, pleural adhesions, and irritation (Potkonjak and Pavlovich 1983). The respiratory irritation reported in the workers diagnosed as having pneumoconiosis was characterized by chronic coughing, wheezing, and upper airway inflammation. Respiratory irritation was not noted in workers exposed to antimony trisulfide for 8 months to 2 years (Brieger et al. 1954). In the reports of health effects associated with occupational exposure to antimony, the workers inhaled a variety of compounds including antimony pentoxide, arsenic oxide, iron oxide, hydrogen sulfide, and sodium hydroxide (Cooper et al. 1968; Potkonjak and Pavlovich 1983; Renes 1953).

A variety of respiratory effects have been reported in animals exposed to antimony. A majority of these effects are associated with the physiological response to dust accumulation in the lung (pneumoconiosis). The effects progress from pneumoconiosis and a proliferation of alveolar macrophages to fibrosis.

Lung inflammation was noted in rabbits exposed to antimony trisulfide for 5 days (Brieger et al. 1954).

Acute exposure to stibine gas also results in lung effects. Pulmonary edema was observed in rats and guinea pigs exposed to a lethal concentration of stibine for 30 minutes (Price et al. 1979).

A dose-related increase in the number of alveolar and/or intraalveolar macrophages was observed in rats exposed to antimony trioxide for 13 weeks or more (Bio/dynamics 1985, 1990). In rats exposed to 0.07 mg antimony/m^3 for 1 year or to 0.92 mg antimony/m^3 for 13 weeks, the proliferation of macrophages was still present for 12 months or 28 weeks, respectively, after exposure termination (Bio/dynamics 1985, 1990). Chronic interstitial inflammation was also observed in rats exposed to 0.07 mg antimony/m^3 for 1 year with a 1 year recovery.

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The proliferation of macrophages is a normal physiological response to the deposition of insoluble particulates in the lung. However, excessive phagocytic activity prompted by extensive or repeated deposition of particulates in the lung probably contributes to the development of fibrosis. Because of the integral role the macrophages have in the progression to fibrosis, nonreversible proliferation of macrophages is considered a less serious adverse health effect.

More severe respiratory effects have also been reported in animals exposed to antimony. Interstitial fibrosis and lipoid pneumonia have been observed in rats exposed to antimony trisulfide or antimony trioxide for 1 year (Bio/dynamics 1990; Gross et al. 1952; Groth et al. 1986; Watt 1980, 1983; Wong et al. 1979). These effects have been reported at exposure levels between 1.6 and 83.6 mg antimony/m^3. No respiratory effects were reported in pigs exposed to 4.2 mg antimony/m^3 as antimony trioxide for 1 year (Watt 1983).

Cardiovascular Effects. Increased blood pressure (greater than 150/90) and altered EKG readings were observed in workers exposed to 2.15 mg antimony/m^3 as antimony trisulfide for 8 months to 2 years (Brieger et al. 1954). Of the 75 workers examined, 37 showed changes in the EKG, mostly of the T-waves; these workers had also been exposed to phenol formaldehyde resin (Brieger et al. 1954). In another group of antimony workers, one out of seven had altered EKG readings (Renes 1953). These limited data on cardiovascular effects in humans are supported by the finding of cardiac effects following parenteral administration of antimony to humans (see discussion of systemic effects in Section 2.4).

Inhalation exposure to antimony trisulfide dust (the same dust the factory workers were exposed to) resulted in degenerative changes in the myocardium and related EKG abnormalities (elevation of the RS-T segments and flattening of T-waves) in a variety of animal species (Brieger et al. 1954). Five days of exposure to 19.94 mg antimony/m^3 as antimony trisulfide resulted in EKG alterations in rabbits. The effective exposure levels resulting in cardiovascular effects were at least four times lower (2-4 mg antimony/m^3) in rats, rabbits, and dogs exposed to airborne antimony for 6-10 weeks, as compared to rabbits acutely exposed (Brieger et al. 1954). Dogs exposed to 3.81 mg antimony/m^3 as antimony trisulfide for 7 weeks (Brieger et al. 1954) or pigs exposed to 4.2 mg antimony/m^3 as antimony trioxide for 1 year (Watt 1983) did not exhibit changes in EKG readings. The degenerative changes of the myocardium observed in rats, rabbits, and dogs exposed to antimony trisulfide consisted of hyperemia and swelling of myocardial fibers (Brieger et al. 1954). Myocardial damage was not observed in rats exposed to 17.48 mg antimony/m^3 as antimony trioxide for 1 year (Groth et al. 1986; Watt 1980; Wong et al. 1979).

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Gastrointestinal Effects. A variety of gastrointestinal disorders have occurred in factory workers engaged in activities including repeated prolonged exposure to airborne antimony trichloride (Taylor 1966), antimony trisulfide (Brieger et al. 1954) or antimony oxide (Renes 1953). These disorders include abdominal pain, diarrhea, vomiting, and ulcers. A causal relationship to antimony exposure has not been definitely established because workers were exposed to a variety of other agents in addition to antimony that might cause or contribute to gastrointestinal effects (e.g., hydrogen chloride, sodium hydroxide). Furthermore, in all likelihood, both inhalation and oral exposure to antimony occur at the workplace. Assuming that gastrointestinal effects are related to antimony-exposure, site monitoring data indicate that effective exposure levels may range from approximately 2 to 70 mg antimony/m^3.

Symptoms of gastrointestinal disturbances were not reported in animals, and no histopathological alterations were observed in rats exposed to antimony trioxide (4.2 mg antimony/m^3) for 1 year (Watt 1980).

Hematological Effects. No studies were located regarding hematological effects in humans after inhalation exposure to antimony.

Toxicologically significant hematological effects have not been observed in rats and pigs following long-term exposure to antimony aerosols ranging from 4 to 20 mg antimony/m^3 as antimony trioxide (Bio/dynamics 1985, 1990; Watt 1983). The only effects observed were small (but statistically significant) changes in the hemoglobin concentration in the erythrocytes and erythrocyte volume in rats exposed to 4.01 mg antimony/m^3 as antimony trioxide (Bio/dynamics 1990).

Musculoskeletal Effects. No studies were located regarding musculoskeletal effects in humans after inhalation exposure to antimony. No histopathological alterations were noted in the musculoskeletal system in rats exposed to 4.2 mg antimony/m^3 as antimony trioxide for 1 year (Watt 1980).

Hepatic Effects. No studies were located regarding hepatic effects in humans after inhalation exposure to antimony.

Parenchymatous and fatty degeneration was observed in rabbits exposed to 19.94 mg antimony/m^3 as antimony trisulfide for 5 days (Brieger et al. 1954) and in guinea pigs exposed to 37.9 mg antimony/m^3 as antimony trioxide for 30 weeks (Dernehl et al. 1945). The duration of exposure is unclear in the Dernehl et al. (1945) study. No hepatic effects were observed in rats exposed to antimony trioxide for 13 weeks (Bio/dynamics 1985) or after 1 year of exposure to antimony trioxide or antimony trisulfide concentrations of 36 mg antimony/m^3 or lower (Bio/dynamics 1990; Groth et al. 1986; Watt 1980; Wong et al. 1979).

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Renal Effects. No studies were located regarding renal effects in humans after inhalation exposure to antimony.

Tubular dilation was observed in rats and guinea pigs exposed to stibine gas for 30 minutes at a concentration of 799 mg antimony/m^3 (Price et al. 1979). Parenchymatous degeneration was observed in rabbits exposed to 19.94 mg antimony/m^3 as antimony trisulfide for 5 days (Brieger et al. 1954). No renal effects were noted in rats exposed to 19.6 mg antimony/m^3 as antimony trioxide for 13 weeks (Bio/dynamics 1985) or 17.5 mg antimony/m^3 as antimony trisulfide or up to 36 mg antimony/m^3 as antimony trioxide for 1 year (Bio/dynamics 1990; Groth et al. 1986; Wong et al. 1979).

Dermal/Ocular Effects. Dermal and ocular effects have been reported in humans. and animals. These effects (ocular conjunctivitis and dermatosis) result from airborne antimony coming into contact with the skin and/or eyes (Potkonjak and Pavlovich 1983; Renes 1953; Stevenson 1965).

The dermatitis associated with exposure to airborne antimony is characterized as epidermal cellular necrosis with associated acute inflammatory cellular reactions (Stevenson 1965). The dermatitis is seen more often during the summer months and in workers exposed to high temperatures (Potkonjak and Pavlovich 1983; Stevenson 1965). Stevenson (1965) concluded that the dermatitis resulted from the action of antimony trioxide on the dermis after dissolving in sweat and penetrating the sweat glands. Transferring the worker to a cooler environment often resulted in the rash clearing up within 3-14 days. Antimony trioxide is not a skin sensitizer in humans following topical application (see Section 2.2.3.3).

Eye irritation has been observed in rats and guinea pigs exposed to stibine gas (Price et al. 1979) and antimony trioxide (Bio/dynamics 1985). Cataracts and chromodacryorrhea have been observed in rats exposed to antimony trioxide for 1 year with a 1 year recovery period (Bio/dynamics 1990). The authors suggest that the chromodacryorrhea may have been secondary to dental abnormality, infectious disease, or xerosis.

Because these dermal and ocular effects may not be the result of inhalation exposure, but rather dermal contact with airborne antimony, the LOAEL values were not recorded in Table 2-l or Figure 2-l. Alopecia was noted in rats exposed to 0.92 mg antimony/m^3 or greater as antimony trioxide for 13 weeks (Bio/dynamics 1985). Because high levels of antimony are measured in the skin or hair of animals following nose-only exposure to antimony aerosols, this effect may not be the result of dermal contact to airborne antimony (Felicetti et al. 1974a, 1974b).

Other Systemic Effects. No studies were located regarding other systemic effects in humans after inhalation exposure to antimony. Hyperplasia

of the reticuloendothelial cells in the peribronchiolar lymph nodes was observed in rats exposed to 0.07 mg antimony/m^3 antimony trioxide for 1 year with a 1 year recovery period (Bio/dynamic 1990).

2.2.1.3 Immunological Effects

No studies were located regarding immunological effects in humans or animals after inhalation exposure to antimony.

2.2.1.4 Neurological Effects

A causal relationship between exposure to airborne antimony and neurological effects in humans has not been established. Nerve tenderness and a tingling sensation were reported in workers exposed to antimony oxide at a concentration of 10.07 mg antimony/m^3 (Renes 1953). However, the factory workers were also exposed to arsenic, lead, copper, and possibly hydrogen sulfide and sodium hydroxide. Thus, it is difficult to determine if this effect was the result of antimony exposure.

No studies were located regarding neurological effects in animals after inhalation exposure to antimony.

2.2.1.5 Developmental Effects

An increased incidence of spontaneous abortions, compared to a control grow, were reported in women working at an antimony metallurgical plant. The women were exposed to a mixture of antimony trioxide, antimony pentasulfide, and metallic antimony (Belyaeva 1967). The level of airborne antimony and presence of other compounds is not known. In addition, a description of the control group was not given; thus, it is unclear if the controls had jobs comparable to those of the exposed group.

A decreased number of offspring was born to rats exposed to 209 mg antimony/m^3 as antimony trioxide prior to conception and throughout gestation. No difference in fetal body weights was observed (Belyaeva 1967). This LOAEL for developmental effects in rats is presented in Table 2-l and Figure 2-1.

2.2.1.6 Reproductive Effects

Disturbances in the menstrual cycle were reported in women exposed to airborne metallic antimony, antimony pentasulfide, and antimony trioxide in a metallurgical plant. No other details were provided (Belyaeva 1967).

In rats exposed to 209 mg antimony/m3 as antimony trioxide for 63 days, 67% failed to conceive. Metaplasia in the uterus and disturbances in the ovum-maturing process were obsewed in the animals that failed to conceive,. These effects were not observed in the rats that conceived (Belyaeva 1967).

This LOAEL value for reproductive effects in rats is presented in Table 2-l and Figure 2-l.

2.2.1.7 Genotoxic Effects

No studies were located regarding genotoxic effects in humans or animals after inhalation exposure to antimony.

Genotoxicity studies are discussed in Section 2.4.

2.2.1.8 Cancer

Inhalation exposure to 8.87 mg antimony/m^3 as antimony oxide did not affect the incidence of cancer in workers employed for 9-31 years (Potkonjak and Pavlovich 1983).

Antimony can be carcinogenic in rats. Lung tumors were observed in rats exposed to 4.2 or 36 mg antimony/m3 as antimony trioxide (Groth et al. 1986; Watt 1980, 1983; Wong et al. 1979) or 17.48 mg antimony/m^3 as antimony trisulfide for 1 year (Groth et al. 1986; Wong et al. 1979). An increased incidence of lung tumors was not observed in rats exposed to 4.01 mg antimony/m^3 as antimony trioxide (Bio/dynamics 1990) or in pigs exposed to 4.2 mg antimony/m^3 as antimony trioxide (Watt 1983). The carcinogenic potential of antimony may be related to the deposition and clearance of antimony from the respiratory tract. Further discussion is presented in Section 2.4. The cancer effect levels are recorded in Table 2-1 and Figure 2-l.

2.2.2 Oral Exposure

Health effects have been observed in humans and animals following oral exposure to a variety of antimony compounds. Adverse effects following exposure to potassium antimony tartrate (an organic form of antimony), antimony trichloride, antimony trioxide, and metallic antimony are discussed below.

2.2.2.1 Death

No studies were located regarding death in humans after oral exposure to antimony.

Mortality was not observed in rats following a single exposure to 188-16,714 mg antimony/kg or lower as inorganic antimony (Fleming 1982; Myers et al. 1978; Smyth and Carpenter 1948; Smyth and Thompson 1945) or to a 7,000 mg antimony/kg dose of metallic antimony (Bradley and Frederick 1941). However, a lower single dose of organic antimony (300 mg antimony/kg dose as potassium antimony tartrate) resulted in death in rats (Bradley and Frederick 1941). Death was attributed to myocardial failure. These NOAELS and LOAELs

for death in animals suggest that organic antimony is more lethal than the inorganic compounds, probably due to increased absorption of the potassium antimony tartrate.

Chronic administration of a low dose of potassium antimony tartrate (0.262 mg antimony/kg/day) resulted in decreased lifespan in rats (Schroeder et al. 1970). No effect on longevity was observed in mice exposed to 0.35 mg antimony/kg/day as potassium antimony tartrate (Kanisawa and Schroeder 1969; Schroeder et al. 1968).

The highest NOAEL values for each antimony compound and all reliable LGAEL values are presented in Table 2-2 and plotted in Figure 2-2.

2.2.2.2 Systemic Effects

Cardiovascular, gastrointestinal, hematological, hepatic, and other systemic effects observed following oral exposure to antimony are presented below. No studies were located regarding respiratory, musculoskeletal, renal, or dermal/ocular effects in human and animals after oral exposure to antimony. The highest NOAEL values and all reliable LOAELs for each systemic effect in each species and duration are presented in Table 2-2 and plotted in Figure 2-2.

Cardiovascular Effects. No studies were located regarding cardiovascular effects in humans after oral exposure to antimony. No effect on blood pressure or heart rate was observed in rats exposed to antimony as antimony trichloride (Marmo et al. 1987) or antimony trioxide (Gross et al. 1955). Pre- and postnatal exposure or only postnatal exposure alone to 0.0748 mg antimony/kg/day as antimony trichloride appears to affect the development of certain cardiovascular reflexes in rats that are important for regulating systemic arterial blood pressure. In rats exposed to antimony trichloride pre- and postnatally or postnatally, a decreased pressor response to 1-noradrenaline and a decreased hypotensive response to 1-isoprenaline and acetylcholine was observed (Marmo et al. 1987). The occurrence of the effect is duration related.

Gastrointestinal Effects. Shortly after drinking an average of 10 ounces of lemonade contaminated with potassium antimony tartrate (equivalent to 0.53 mg antimony/kg for a 70 kg man), workers began to vomit (Dunn 1928). Gastrointestinal effects have also been reported in factory workers after exposure to airborne antimony dust. As discussed in Section 2.2.1.2, the gastrointestinal effects probably resulted from swallowing the antimony dust.

Vomiting and diarrhea have also been observed in animals following acute exposure to antimony trioxide or potassium antimony tartrate (Houpt et al.

TABLE 2-2. Levels of Significant Exposure to Antimony - Oral

Key to figure[a]	Species	Route	Exposure frequency/ duration	System	NOAEL (mg/kg/day)	LOAEL (effect) Less serious (mg/kg/day)	LOAEL (effect) Serious (mg/kg/day)	Reference	Form
ACUTE EXPOSURE									
Death									
1	Rat	(GW)	1 d 1x/d				300 (decreased survival)	Bradley and Frederick 1941	Potassium tartrate
2	Rat	(GO)	1 d 1x/d		7,000			Bradley and Frederick 1941	Metallic antimony
3	Rat	(F)	1 d 1x/d		16,714			Smyth and Thompson 1945	Trioxide
Systemic									
4	Human	(W)	1 d	Gastro		0.529 (vomiting)		Dunn 1928	Potassium tartrate
5	Rat	(GO)	1 d 1x/d	Gastro		16,714 (diarrhea)		Myers et al. 1978	Trioxide
6	Rat	(GW)	1 d 1x/d	Gastro Hepatic	376 376			Fleming 1982	Trioxide
7	Dog	(W)	1 d	Gastro		13.2 (vomiting)		Houpt et al. 1984	Potassium tartrate
INTERMEDIATE EXPOSURE									
Systemic									
8	Rat	(F)	24 wk	Hemato	500	1,000 (decreased hemotocrit and hemoglobin)		Sunagawa 1981	Metallic antimony
				Hepatic	250	500 (cloudy swelling in hepatic cords)			
9	Rat	(F)	12 wk	Hemato	418			Hiraoka 1986	Trioxide

TABLE 2-2 (Continued)

Key to figure[a]	Species	Route	Exposure frequency/ duration	System	NOAEL (mg/kg/day)	LOAEL (effect) Less serious (mg/kg/day)	LOAEL (effect) Serious (mg/kg/day)	Reference	Form
10	Rat	(W)	30 d	Cardio	0.0748	0.748 (decreased hypotensive response in newborns)		Marmo et al. 1987	Trichloride
11	Rat	(F)	24 wk	Hemato		418 (decreased RBC count)		Sunagawa 1981	Trioxide
				Hepatic		418 (cloudy swelling in hepatic cords)			
12	Rat	(F)	12 wk	Hemato		500 (decreased total plasma protein)		Hiraoka 1986	Metallic antimony
13	Rat	(GW)	20 d 1x/d	Gastro	501			Fleming 1982	Trioxide
14	Rat	(W)	81 d Gd 0-21 birth to 60 days	Cardio		0.0748 (decreased hypotensive response in newborns)		Angrisani 1988; Marmo et al. 1987; Rossi et al. 1987	Trichloride
15	Rat	(W)	60 d	Cardio		0.0748 (decreased hypotensive response in newborns)		Marmo et al. 1987	Trichloride
16	Rat	(W)	21 d Gd 0-21	Other		0.0748 (decreased maternal weight gain)		Rossi et al. 1987	Trichloride
17	Rat	(F)	30 d	Hemato	226	894 (increased RBC count)		Smyth and Thompson 1945	Trioxide
18	Dog	(GW)	32 d 1x/d	Gastro			84 (severe diarrhea)	Fleming 1982	Trioxide
				Other			6,644 (weight loss)		
Neurological									
19	Dog	(GW)	32 d 1x/d				6,644 (muscle weakness)	Fleming 1982	Trioxide

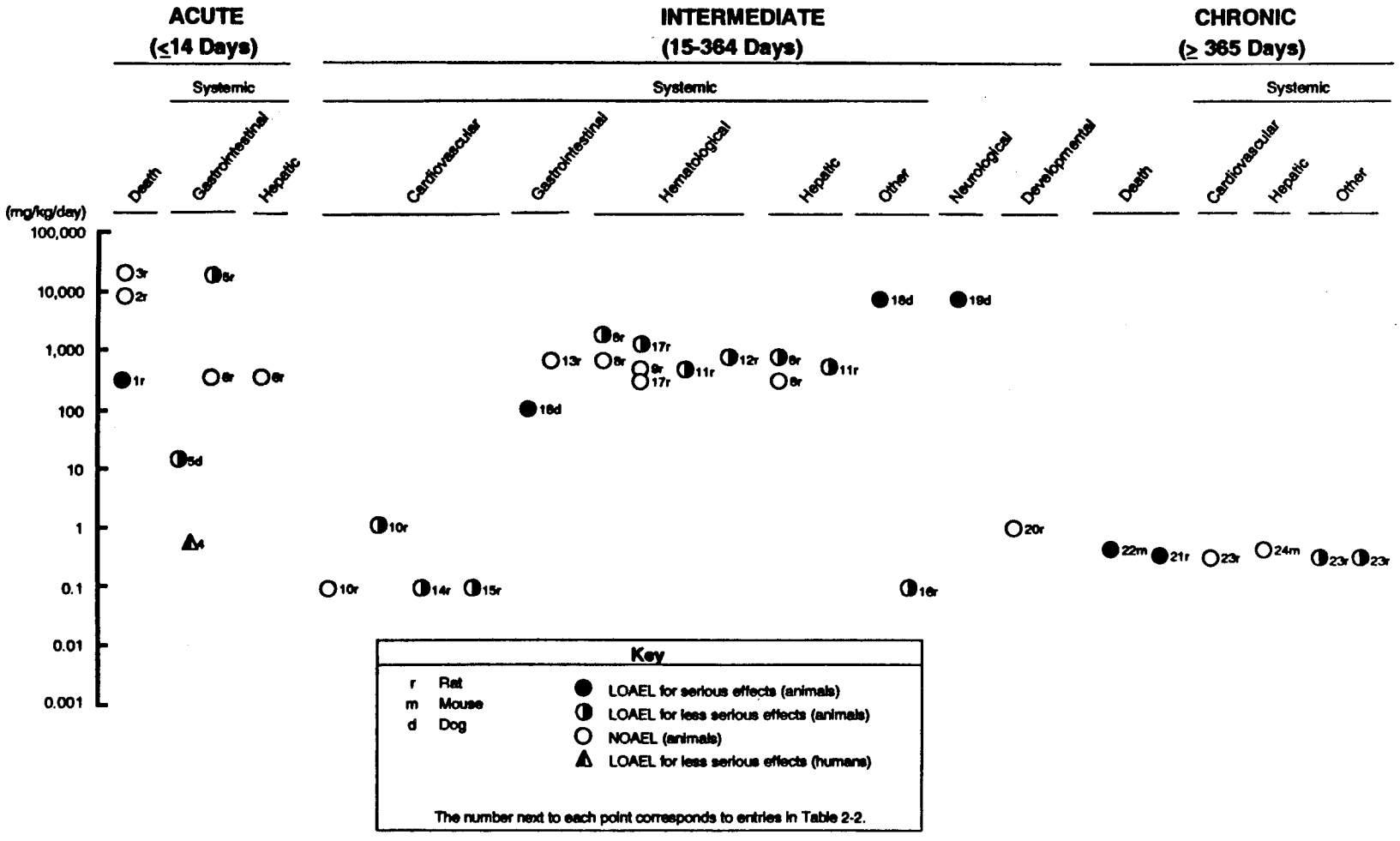
TABLE 2-2 (Continued)

Key to figure[a]	Species	Route	Exposure frequency/ duration	System	NOAEL (mg/kg/day)	LOAEL (effect) Less serious (mg/kg/day)	LOAEL (effect) Serious (mg/kg/day)	Reference	Form
Developmental									
20	Rat	(W)	21 d Gd 0-21		0.748			Rossi et al. 1987	Trichloride
CHRONIC EXPOSURE									
Death									
21	Rat	(W)	746-1,342 d				0.262 (decreased lifespan)	Schroeder et al. 1970	Potassium tartrate
22	Mouse	(W)	542-909 d				0.35 (decreased lifespan - females)	Kanisawa and Schroeder 1969; Schroeder 1968	Potassium tartrate
Systemic									
23	Rat	(W)	746-1,342 d	Cardio	0.262			Schroeder et al. 1970	Potassium tartrate
				Other		0.262 (decreased nonfasting serum glucose)			
				Other		0.262 (increased serum cholesterol)			
24	Mouse	(W)	542-909 d	Hepatic	0.35			Schroeder et al. 1968	Potassium tartrate

[a]The number corresponds to entries in Figure 2-1.

Cardio = cardiovascular; d = day; (F) = food; Gastro = gastrointestinal; Gd = gestation day; (GO) = gavage oil; (GW) = gavage water; Hemato = hematological; LOAEL = lowest-observed-adverse-effect level; NOAEL = no-observed-adverse-effect level; RBC = red blood cell; (W) = water; wk = week

FIGURE 2-2. Levels of Significant Exposure to Antimony - Oral



1984; Myers et al. 1978). Severe diarrhea was observed in dogs administered 84 mg antimony/kg/day as antimony trioxide for 32 days. No gastrointestinal effects or gross abnormalities were noted in rats exposed to 501 mg antimony/kg/day or less as antimony trioxide for 20 days (Fleming 1982).

Hematological Effects. No studies were located regarding hematological effects in humans after oral exposure to antimony.

Mild hematological alterations are observed in animals exposed to 418 mg antimony/kg/day or greater. Increased red blood cell count was observed in rats exposed to 894 mg antimony/kg/day as antimony trioxide for 30 days (Smyth and Thompson 1945). Exposure to metallic antimony resulted in decreased hematocrit and hemoglobin levels and decreased plasma protein levels in rats exposed to 500-1,000 mg antimony/kg/day for 12-24 weeks (Hiraoka 1986; Sunagawa 1981). Decreased red blood cell count was observed in rats exposed to 418 mg antimony/kg/day as antimony trioxide for 24 weeks (Sunagawa 1981).

Hepatic Effects. No studies were located regarding hepatic effects in humans after oral exposure to antimony.

Cloudy swelling of the hepatic cords has been observed in rats exposed to 418 mg antimony/kg/day as antimony trioxide or 500 mg antimony/kg/day as metallic antimony (Sunagawa 1981). Hepatic effects have not been observed at lower concentrations of antimony trioxide or potassium antimony tartrate (Fleming 1982; Schroeder et al. 1968).

Other Systemic Effects. No studies were located regarding other systemic effects in humans after oral exposure to antimony.

Severe weight loss was observed in dogs administered 6,644 mg antimony/kg/day as antimony trioxide. Severe diarrhea and vomiting were also observed in these dogs (Fleming 1982).

Increased serum cholesterol and decreased nonfasting serum glucose levels were observed in rats exposed for a lifetime to low levels of potassium antimony tartrate in drinking water (Schroeder et al. 1970). The toxicologic significance of these effects is not known.

2.2.2.3 Immunological Effects

No studies were located regarding immunological effects in humans or animals after oral exposure to antimony.

2.2.2.4 Neurological Effects

No studies were located regarding neurological effects in humans after oral exposure to antimony.

Muscle weakness and difficulty in moving hind limbs were observed in a dog exposed to 6,644 mg antimony/kg/day as antimony trioxide for 32 days (Fleming 1982). This LOAEL value for neurological effects in dogs is recorded in Table 2-2 and Figure 2-2.

2.2.2.5 Developmental Effects

No studies were located regarding developmental effects in humans after oral exposure to antimony.

No developmental effects (differences in the number of newborn pups per litter and macroscopic teratogenic effects) were observed in the offspring of rats treated during gestation with 0.748 mg antimony/kg/day as antimony trichloride (Rossi et al. 1987). As discussed in the cardiovascular effects section, pre- and postnatal or postnatal exposure impaired the development of certain cardiovascular reflexes that are important in regulating systemic arterial blood pressure (Angrisani et al. 1988; Marmo et al. 1987; Rossi et al. 1987). Because comparisons were not made between the hypotensive response in pups exposed prenatally and the response in pups exposed postnatally, the potential of antimony trichloride to produce developmental cardiovascular effects cannot be assessed.

2.2.2.6 Reproductive Effects

No studies were located regarding reproductive effects in humans or animals after oral exposure to antimony.

2.2.2.7 Genotoxic Effects

No studies were located regarding genotoxic effects in humans or animals after oral exposure to antimony.

Genotoxicity studies are discussed in Section 2.4.

2.2.2.8 Cancer

No studies were located regarding cancer effects in humans after oral exposure to antimony.

No change in the incidence of cancer was observed in rats (Schroeder 1970) or mice (Kanisawa and Schroeder 1969; Schroeder 1968) fed 0.262 or 0.35 mg antimony/kg/day, respectively, as potassium antimony tartrate for a lifetime. The use of these studies to assess carcinogenicity is limited because only one exposure level was used, which was below the maximum tolerated dose.

2. HEALTH EFFECTS

2.2.3 Dermal Exposure

The dermal toxicity of antimony compounds is discussed below. Data were located on the health effects following application of antimony trioxide, antimony thioantimonate (a mixture of antimony trfsulfide, and antimony pentasulfide) and antimony oxide to the skin or eye.

2.2.3.1 Death

No studies were located regarding death in humans after dermal exposure to antimony.

Death was observed in rabbits following a single application of antimony oxides at a level of 6,685 mg antimony/kg (Myers et al. 1978). The cause of death was not reported. Two out of four rabbits died after 6-8 topical applications of antimony trioxide paste. The antimony trioxide was combined with a mixture formulated to resemble acidic sweat. The application area was not occluded; thus, there is a possibility of oral ingestion of the paste (Fleming 1982). Death was not reported in rabbits after 13 weeks of application of a 5% solution of antimony thioantimonate (a mixture of antimony trisulfide and antimony pentasulfide) (Horton et al. 1986). The highest NOAEL and all reliable LOAEL values for death for rabbits for each duration are recorded in Table 2-3.

2.2.3.2 Systemic Effects

Respiratory, cardiovascular, gastrointestinal, and dermal/ocular effects following dermal or ocular exposure are presented below. No studies were located regarding respiratory, gastrointestinal, hematological, musculoskeletal, hepatic, or renal effects in humans and animals following dermal exposure to antimony. The highest NOAEL for each antimony compound and all reliable LOAEL values for each systemic effect for each species are recorded in Table 2-3.

Respiratory Effects. No studies were located regarding respiratory effects in humans following dermal exposure to antimony. Hyperemia in the lungs was observed in two rabbits that died after 6-8 applications of an antimony trioxide paste to shaven and abraded skin. The antimony trioxide (concentration not reported) was combined with a mixture resembling acidic sweat (Fleming, 1982). The application area was not occluded; thus, the ingestion of the paste may have occurred.

Cardiovascular Effects. No studies were located regarding cardiovascular effects in humans following dermal exposure to antimony. Application of a 5% solution of antimony thioantimonate did not change EKG readings or heart pathology in rabbits (Horton et al. 1986).

TABLE 2-3. Levels of Significant Exposure to Antimony - Dermal

Species	Exposure frequency/ duration	System	NOAEL	LOAEL (effect) Less serious	LOAEL (effect) Serious	Reference	Form
ACUTE EXPOSURE							
Death							
Rabbit	1 d				6,685 mg/kg (1/6 died)	Myers et al. 1978	Trioxide
Systemic							
Rabbit	1 d 1x/d	Ocular		79.2 mg (mild eye irritation)		Wil Research Laboratories 1979	Oxide
Rabbit	1 d 1x/d	Ocular		100 mg (eye irritation)		Horton et al. 1986	Trisulfide and pentasulfide
Rabbit	1 d	Dermal		6,685 mg/kg (edema)		Myers et al. 1978	Trioxide
Rabbit	1 d 1x/d	Ocular	209 mg			Myers et al. 1978	Trioxide
Rabbit	1 d 1x/d	Dermal	20,900 mg			Gross et al. 1955	Trioxide
Neurological							
Rabbit	1 d			6,685 mg/kg (abnormal gait)		Myers et al. 1978	Trioxide
INTERMEDIATE EXPOSURE							
Death							
Rabbit	13 wk 5 d/wk		5%			Horton et al. 1986	Trisulfide and pentasulfide

TABLE 2-3 (Continued)

Species	Exposure frequency/ duration	System	NOAEL	LOAEL (effect)		Reference	Form
				Less serious	Serious		
Systemic							
Rabbit	13 wk 5 d/wk	Cardio Derm/oc Other	5% 5% 5%			Horton et al. 1986	Trisulfide and pentasulfide

Cardio = cardiovascular; d = day; Derm/oc = dermal/ocular; Gn pig = guinea pig; hr = hour; LOAEL = lowest-observed-adverse-effect level; NOAEL = no-observed-adverse-effect level; wk = week

Gastrointestinal Effects. No studies were located regarding gastrointestinal effects in humans following dermal exposure to antimony. Hemorrhages in the cardiac portion of the stomach were observed in two rabbits that died after 6-8 applications of an antimony trioxide-acidic sweat paste (Fleming 1982). Because the application area was not occluded, ingestion of the paste is possible.

Dermal/Ocular Effects. No studies were located regarding dermal/ocular effects in humans following dermal exposure to antimony.

In rabbits, edema was noted in the area where an antimony trioxide patch (6,685 mg antimony/kg) was applied for 1 day (Myers et al. 1978).

Instillation of 79-100 mg antimony.as antimony oxide or antimony thioantimonate into the eyes of rabbits resulted in eye irritation (Horton et al. 1986; Wil Research Laboratories). However, instillation of antimony trioxide (34.5-83.6 mg antimony) did not result in eye irritation (Gross et al. 1955; Myers et al. 1978).

Dermal and ocular effects have been observed in humans and animals exposed to airborne antimony. The effects include ocular onjunctivitis, eye irritation, and dermatosis. Further information on these effects is provided in Section 2.2.1.2.

2.2.3.3 Immunological Effects

No studies were located regarding immunological effects in humans and animals following dermal exposure to antimony.

2.2.3.4 Neurological Effects

No studies were located regarding neurological effects in humans after dermal exposure to antimony.

Abnormal gait was observed in rabbits following application of a lethal concentration of antimony trioxide (6,685 mg antimony/kg/day) (Myers et al. 1978). This LOAEL value for neurotoxicity in rabbits is recorded in Table 2-3.

No studies were located regarding the following effects in humans or animals after dermal exposure to antimony:

2.2.3.5 Developmental Effects
2.2.3.6 Reproductive Effects
2.2.3.7 Genotoxic Effects

Genotoxicity studies are discussed in Section 2.4.

2. HEALTH EFFECTS

2.2.3.8 Cancer

No studies were located regarding cancer effects in humans or animals after dermal exposure to antimony.

2.3 TOXICOKINETICS

2.3.1 Absorption

2.3.1.1 Inhalation Exposure

Quantitative data on the absorption of antimony from the lungs in humans were not located. Elevated blood and urine antimony levels were observed in workers exposed to antimony, suggesting that antimony is absorbed (Cooper et al. 1968; Ludersdorf et al. 1987). However, there is a possibility that some of the antimony detected in the urine and blood was swallowed.

The International Commission on Radiological Protection (ICRP 1981) considers oxides, hydroxides, halides, sulfides, sulfates, and nitrates of antimony to be class W chemicals. All other common compounds of antimony are assigned to class D. Class W and D chemicals are considered to have respiratory tract clearance rates of weeks and days, respectively. The ICRP classifications are based on animal data (Felicetti et al. 1974a, 1974b; Thomas et al. 1973). Data from deceased antimony smelter workers suggest that the elimination half-time of some forms of antimony in the lungs may be longer than weeks (Gerhardsson et al. 1982).

The absorption of antimony from the respiratory tract is a function of particle size. Exposure to antimony tartrate with a particle size of 1.6 μm resulted in a greater deposition of antimony in the upper respiratory tract than exposure to 0.7 or 0.3 μm particles (Felicetti et al. 1974a; Thomas et al. 1973). Furthermore, the antimony deposited in the upper respiratory tract was cleared after several hours via mucociliary clearance. Particles of the two smaller sizes were relatively insoluble in the lung and were slowly absorbed over several weeks (Thomas et al. 1973). No difference in the body burden, 1 day after exposure to trivalent or pentavalent antimony tartrate, was observed (Felicetti et al. 1974b). Although no information on differences in absorption rates between antimony compounds was located, differences related to solubility probably exist.

2.3.1.2 Oral Exposure

No quantitative data on the absorption of antimony from the gastrointestinal tract in humans were located. However, results of studies in animals suggest that at least certain forms of antimony are probably absorbed from the gastrointestinal tract. Estimates of the absorption of antimony tartrate and antimony trichloride in animals range from 2% to 7% (Felicetti et al. 1974b; Gerber et al. 1982), suggesting that absorption of trivalent

antimony salts in humans is probably less than 10%. Gastrointestinal absorption of antimony is likely to be affected by numerous factors, including chemical form of the ingested antimony, particle size and solubility, age, and diet. Although quantitative information on the absorption of antimony is not available for all forms, ICRP (1981) has recommended 10% for antimony tartrate and 1% for all other forms of antimony as reference values for gastrointestinal
absorption in humans.

2.3.1.3 Dermal Exposure

No studies were located regarding absorption of antimony in humans following dermal exposure.

Exposure to high levels of antimony trioxide or a mixture of antimony trioxide and pentoxide resulted in death in rabbits (Myers et al. 1978). The application area was occluded, suggesting that at least some forms of antimony can be absorbed through the skin.

2.3.2 Distribution

Very low levels of antimony are found in unexposed humans. Autopsy data on Japanese adults (Sumino et al. 1975) and other data on selected body fluids are presented in Table 2-4. The mean body burden of antimony is 0.7 mg (Sumino et al. 1975). The skin and hair had the highest levels of antimony. A somewhat higher estimate of 7.9 mg for total body burden is reported by ICRP (1981). ICRP (1981) has recommended'reference values of 5.9 mg of antimony in soft tissue and 2.0 mg in skeletal tissue.

2.3.2.1 Inhalation Exposure

Information on the distribution of antimony in humans following inhalation exposure was not located. Blood is the main vehicle for the transport of absorbed antimony to various tissue compartments of the body. The relative partitioning between erythrocytes and plasma is a function of valency. Following exposure to trivalent antimony, erythrocyte levels are elevated, compared to the elevated plasma antimony levels after inhalation exposure to pentavalent antimony (Felicetti et al. 1974b). The clearance of antimony from the blood appears to differ among animal species. Elevated blood antimony levels persist longer in rats than in mice and dogs (Felicetti et al. 1974a; Thomas et al. 1973).

Valence-state differences also exist in the distribution of antimony to the rest of the body. In hamsters, the levels of trivalent antimony increase more rapidly in the liver than pentavalent antimony. Skeletal uptake is greater following exposure to pentavalent antimony than trivalent antimony (Felicetti et al. 1974b). Outside of the respiratory tract, antimony accumulates in the liver, thyroid, skeleton, and fur; with the largest burden of antimony in the fur (Felicetti et al. 1974a, 1974b).

TABLE 2-4. Levels of Antimony Found in Various Tissues of Unexposed Humans

Tissue	Concentration (μg/g)	Reference
Hair	0.12	Muramatsu and Parr 1988
	0.096	Takagi et al. 1986
Adrenal gland	0.073	Sumino et al. 1975
Skin	0.096	Sumino et al. 1975
Lung	0.062	Sumino et al. 1975
Large intestine	0.047	Sumino et al. 1975
Trachea	0.045	Sumino et al. 1975
Cerebellum	0.030	Sumino et al. 1975
Kidney	0.043	Sumino et al. 1975
	Not detected	Muramatsu and Parr 1988
Small intestine	0.039	Sumino et al. 1975
Heart	0.032	Sumino et al. 1975
Pancreas	0.030	Sumino et al. 1975
Spleen	0.029	Sumino et al. 1975
Liver	0.023	Sumino et al. 1975
	Not detected	Muramatsu and Parr 1988
Ovary	0.021	Sumino et al. 1975
Testicle	0.017	Sumino et al. 1975
Cerebrum	0.016	Sumino et al. 1975
Blood	0.016	Sumino et al. 1975
	0.34	Mansour et al. 1967
Saliva	0.003	Olmez et al. 1978

2.3.2.2 Oral Exposure

Data on the distribution of antimony in humans following oral exposure to antimony were not located.

Following oral exposure in animals, the major sites of accumulation, outside of the gastrointestinal tract, are the liver, kidney, bone, lung, spleen, and thyroid. However, the rise in antimony levels in these tissues is not dose-related (Sunagawa 1981). This lack of dose-responsiveness may be a reflection of decreased absorption at higher antimony concentrations. Antimony levels tend to reach a plateau in the livers and lungs of voles fed a diet containing antimony trioxide (Ainsworth 1988).

Some species differences in animals exist in the elimination of antimony from the tissues. In rats, antimony is cleared slowly from the thyroid, with an elimination half-time of approximately 40 days (Gross et al. 1955); however, more than 50% of liver, lung, and kidney antimony is removed after 15 days following exposure in voles (Ainsworth 1988).

Evidence is insufficient to determine if there are valency differences in the distribution of orally administered antimony. Based on the inhalation data and the fact that higher liver concentrations were found in rats fed metallic antimony than those fed antimony trioxide (Sunagawa 1981), it is assumed that there are differences.

Pregnancy results in a higher antimony body burden in mice. However, transplacental transport of antimony appears limited. Exposure to antimony during lactation results in high antimony levels in newborns (Gerber et al. 1982).

2.3.2.3 Dermal Exposure

No information on the distribution of antimony in humans or animals following dermal exposure to antimony was located. However, judging from studies of the distribution of antimony following inhalation, oral, and parenteral exposure in animals, the major sites of accumulation are likely to include the liver, kidney, skeleton, spleen, and fur.

2.3.2.4 Other Routes of Exposure

No information on the distribution of antimony in humans following parenteral exposure was located. In animals, antimony is recovered primarily in the liver, with smaller amounts in the spleen, heart, lungs, and muscle (Gellhorn et al. 1946; Gerber et al. 1982).

Two hours after intraperitoneal injection of trivalent antimony, 95% of the antimony in the blood is incorporated into the erythrocytes, mainly in the hemoglobin fraction (Edel et al. 1983; Lippincott et al. 1947). Pentavalent

antimony is primarily distributed into the plasma fraction of blood (Edel et al. 1983).

Following intraperitoneal administration of trivalent antimony, a larger percentage of the administered dose is recovered in the liver than in the spleen. However, a smaller difference in antimony levels between the liver and spleen was observed when pentavalent antimony was administered (Gellhorn and van Dyke 1946).

2.3.3 Metabolism

Antimony is a metal and, therefore, does not undergo catabolism. Antimony can covalently interact with sulfhydryl groups and phosphate, as well as numerous reversible binding interactions with endogenous ligands (e.g., proteins). It is not known if these interactions are toxicologically significant. No information was located on the in vivo interconversion of trivalent and pentavalent antimony.

2.3.4 Excretion

2.3.4.1 Inhalation Exposure

Increased levels of urinary antimony have been noted in workers exposed to antimony trioxide (Cooper et al. 1968; Ludersdorf et al. 1987). In animals, antimony is excreted via the urine and feces. Some of the fecal antimony may represent unabsorbed antimony that is cleared from the lung via mucociliary action into the esophagus to the gastrointestinal tract. Based on studies in which antimony was parenterally administered to animals, the urine/feces ratio of antimony depends on valence state. Antimony is excreted predominantly in the urine following pentavalent antimony injection and in the feces after trivalent antimony administration (Edel et al. 1983; Felicetti et al. 1974b).

In animals, whole-body clearance of trivalent antimony tartrate occurs in two phases. Ninety percent of the initial body burden of antimony tartrate was excreted within the first 24 hours. The half-life of the slow phase was 16 days (Felicetti et al. 1974b).

2.3.4.2 Oral Exposure

Information on the excretion of antimony in humans following oral exposure was not located. However, information obtained from human and animal studies in which antimony was administered parenterally provides some insight regarding the routes and rates of excretion that can be anticipated after oral exposure in humans. Animal studies have shown that ingested antimony is only partially absorbed from the gastrointestinal tract (Felicetti et al. 1974b; Gerber et al. 1982). Assuming that this is also true for humans, fecal excretion is probably an important route of excretion of ingested antimony in

humans. Antimony absorbed from the gastrointestinal tract appears to be excreted in the urine and feces to a variable degree, depending on the chemical form. Pentavalent antimony injected parenterally into humans or animals is excreted predominantly in the urine, whereas injected trivalent antimony is excreted in the feces (Edel et al. 1983; Goodwin and Page 1943; Rees et al. 1980).

2.3.4.3 Dermal Exposure

No information on the excretion of antimony following dermal exposure in humans or animals was located. However, information obtained from human and animal studies in which antimony was administered parenterally provides some insight regarding the routes and rates of excretion that can be anticipated after dermal exposure in humans. Antimony that is absorbed through the skin will be excreted in urine and feces to a variable degree, depending on the chemical species. Pentavalent antimony injected parenterally into humans or animals is excreted predominantly in urine, whereas injected trivalent antimony is excreted in feces (Edel et al. 1983; Goodwin and Page 1943; Rees et al. 1980).

2.3.4.4 Other Routes of Exposure

Pentavalent antimony is rapidly excreted in humans following intravenous or intramuscular administration, with greater than 50% excreted in the urine 6 hours after injection (Goodwin and Page 1943; Rees et al. 1980). Trivalent antimony is predominantly excreted in the feces and not as rapidly excreted in the urine as pentavalent antimony. Twenty-four hours after injection, approximately 25% was excreted in the urine (Goodwin and Page 1943).

Twenty-four hours following intraperitoneal administration of trivalent antimony in rats, 33% of the compound was excreted via the feces and 6% in the urine. In contrast, 88% of the pentavalent antimony was excreted in the urine and 1% in the feces (Edel et al. 1983j.

Following repeated intramuscular administration of trivalent antimony in humans, approximately 15% was excreted per day at the beginning of treatment and 25% at the end of treatment. Fecal antimony excretion ranged from 4% in the beginning of treatment to 15.4% of the daily administered dose toward the end of treatment (Lippincott et al. 1947).

The elimination of pentavalent antimony following intramuscular injection fits into a two-compartment pharmacokinetic model. The half-life of the rapid phase of elimination was 2 hours; the slower phase was 76 hours (Chulay et al. 1988).

2. HEALTH EFFECTS

2.4 RELEVANCE TO PUBLIC HEALTH

Adverse health effects have been observed in humans and animals following exposure to antimony and antimony compounds. Metallic antimony, organic forms, and inorganic forms of antimony were included in this profile. The organic forms of antimony discussed are potassium antimony tartrate, sodium antimony tartrate, and antimony acetate. Also included are the antimony-containing drugs stibocaptate (sodium antimony-2,3-meso-di-mercapto succinate) also referred to as astiban, and stibophen (bis[4,5-dihydroxy- 1,3-benzenedisulfonato(4)-O^4, O^5]-antimonate (5-) pentasodium heptahydrate) also called fuadin. Trivalent inorganic antimony compounds (antimony trioxide, antimony trichloride, antimony trisulfide), pentavalent inorganic compounds (antimony pentoxide, antimony pentachloride, and antimony pentasulfide), and stibine are also discussed. The toxicity data for antimony and compounds have been summarized across compounds; if differences in the toxicity between the various antimony compounds are known, this information will be presented in a compound specific discussion.

The toxicological effects of antimony in humans following inhalation or oral exposure are pneumoconiosis, altered EKG readings, increased blood pressure, abdominal distress, ulcers, dermatosis, and ocular irritation. No effects were found in humans after dermal exposure to antimony. There are several beneficial uses of antimony. Antimony and its compounds are among the oldest known remedies in the practice of medicine. Currently, antimony compounds are used to treat two parasitic diseases, schistosomiasis and leishmaniasis. Toxic side effects in humans following intraperitoneal, intravenous, or intramuscular injection of an antimony-containing drug have been reported. These effects include altered EKG, anemia, vomiting, diarrhea, joint and/or muscle pain, and death.

Similar toxicological effects have been reported in animals following inhalation, oral, or dermal exposure to antimony. These effects include fibrosis in the lung, altered EKG readings, myocardial damage, vomiting and diarrhea in dogs, parenchymatous degeneration in the liver and kidney, muscle weakness, difficulty in moving, developmental effects, and lung cancer. In addition, degeneration of the myoneural junction has been observed in animals following parenteral administration of antimony.

Inhalation and oral MRLs for antimony and compounds were not derived. Damage to the lungs and myocardium has been observed in several species of animals following acute, intermediate, and chronic inhalation exposure (Brieger et al. 1954; Bio/dynamics 1985, 1990; Gross et al. 1952; Groth et al. 1986; Watt 1983). These effects have also been observed in humans chronically exposed to airborne antimony (Brieger et al. 1954; Potkonjak and Pavlovich 1983). At the lowest exposure levels tested, the adversity of the effects was considered to be serious. Thus, the data were inadequate for the derivation of an acute-, intermediate-, and chronic-duration inhalation MKL values.

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The lowest LGAEL for acute oral exposure is from-a human report (Dunn 1928). Gastrointestinal disturbances were reported in workers who drank lemonade contaminated with potassium antimony tartrate. If the dose was administered throughout the day rather than consumed as a bolus administration, it is likely that the gastrointestinal disturbances would not be observed. Thus, this study would not be an appropriate basis for an acuteduration oral MRL. The intermediate-duration inhalation data suggest that the myocardium is a target of antimony toxicity. The intermediate oral studies did not examine sensitive end points (e.g., EKG) of myocardial damage. This deficiency precludes derivation of an intermediate duration oral MRL. Two chronic oral studies were identified (Schroeder et al. 1968, 1970). At the lowest dose tested, decreased lifespan was observed in rats; this is not an appropriate basis for a chronic-duration oral MRL.

Acute-, intermediate-, and chronic-duration dermal MRLs were not derived for antimony due to the lack of an appropriate methodology for the development of dermal MRLs.

Death. Death has not been reported in humans following inhalation, oral, or dermal exposure to antimony. However, acute exposure to approximately 2 mg antimony/kg/day as stibocaptate (a drug used to treat parasitic disease) administered intramuscularly resulted in the death of an adult and a child (Rugemalila 1980). Therefore, antimony may be lethal at sufficiently high exposure levels. Animal studies have provided some information about the relative lethality of various forms of antimony. Based on data from studies on parenterally administered antimony, relative lethality can be ranked as follows: antimony tartrate > metallic antimony > inorganic trivalent antimony (Bradley and Frederick 1941).

Systemic Effects

Respiratory Effects. The respiratory tract is a target in humans following inhalation exposure to antimony. Pneumoconiosis, impaired pulmonary function (airway obstruction, bronchospasm, and hyperinflation) and respiratory irritation (coughing and wheezing) have been observed in factory workers exposed to antimony dust (Cooper et al. 1968; Potkonjak and Pavlovich 1983). A relationship between exposure level and effect cannot be established from this data because the workers were also exposed to other compounds, including arsenic oxide, iron oxide, hydrogen chloride, and hydrogen sulfide.

Information on the health effects in animals following inhalation exposure to antimony supports the finding in humans that the respiratory tract is a target. Most of the respiratory effects observed in animals are associated with the physiological response to dust accumulation in the respiratory tract. Because of the large amount of antimony that is deposited in the lung during chronic inhalation, the proliferation of macrophages observed in rats exposed to 0.07 mg antimony/m^3 or greater continues several

months after the exposure termination (Bio/dynamics 1990). This increase in the number of alveolar macrophages may contribute to the development of fibrosis. Fibrosis and lipoid pneumonia have been reported in rats chronically exposed to 1.6 mg antimony/m^3 or higher as antimony trioxide or to 17.48 mg antimony/m3 as antimony trisulfide (Bio/dynamics 1990; Gross et al. 1952; Groth et al. 1986; Watt et al. 1980,1983; Wong et al. 1979). Respiratory effects have not been reported in humans or animals following oral or dermal exposure to antimony.

Although serious antimony-related lung disease has not been observed in humans, antimony-induced pneumoconiosis is associated with serious lung pathology in animals. Therefore, it is likely that, with sufficiently high or prolonged exposures, serious lung disease would occur in humans. In addition, the toxicity of inhaled antimony compounds may be greater for smaller particle sizes.

Cardiovascular Effects. The heart is another target organ in humans. Alterations in EKG readings and increased blood pressure have been reported in workers exposed to airborne antimony trisulfide (Brieger et al. 1954). In addition, altered EKG readings have been reported in individuals exposed to repeated injections of antimony (Dancaster et al. 1966; Honey 1960; Pandey et al. 1988). The antimony injections were part of a therapeutic treatment for parasitic disease. In some of these individuals, the EKG did not return to normal until 6 weeks after the last dose (Dancaster et al. 1966). Pentavalent antimony appears to be less cardiotoxic than the trivalent form. Altered EKG readings were observed after 4 days of trivalent antimony treatment (0.98 mg antimony/kg/day) (Dancaster et al. 1966); however, a change in EKG readings was not observed until after 3 weeks of pentavalent antimony injections (7.2 mg antimony/kg/day) (Pandey et al. 1988).

Altered EKG readings have also been observed in animals. In addition, decreased blood pressure, increased heart rate, and decreased contractile force have been observed following injection of trivalent antimony (Bromberger-Barnea and Stephens 1965; Cotten and Logan 1966). The decreased blood pressure contrasts with the increased blood pressure observed in humans (Brieger et al. 1954). Studies on isolated dog hearts suggest that antimony exerts its effect on the myocardium directly, and that the effect persists after exposure is terminated (Bromberger-Barnea and Stephens 1965).

Gastrointestinal Effects. Historically, antimony has been known for its emetic properties. Vomiting, diarrhea, gastric discomfort, and ulcers have been reported in humans following inhalation or oral exposure to antimony. Amounts as low as 0.529 mg antimony/kg have resulted in vomiting. The gastrointestinal effects following inhalation exposure may have resulted from antimony being swallowed. Gastrointestinal effects have also been observed in humans receiving intramuscular injections of antimony (Harris 1956; Zaki et al. 1964). Similar gastrointestinal effects have been reported in animals following oral exposure to antimony.

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Hematological Effects. Hematological effects in humans following inhalation, oral, or dermal exposure to antimony have not been reported. However, hematological parameters were not measured in the human studies. Hemolytic anemia was reported in one subject following repeated injections of fuadin (stibophen) (Harris 1956). Fuadin is an antimony-containing compound used in the treatment of schistosomiasis. Alterations in hematological parameters have not been reported in animals exposed to antimony via inhalation or dermal routes. Decreased hemoglobin and hematocrit and altered erythrocyte count were observed in animals following oral exposure to metallic antimony or antimony trioxide (Smyth and Thompson 1945; Sunagawa 1981). The potential of antimony to cause hematological effects in humans is not known.

Musculoskeletal Effects. Musculoskeletal effects have not been reported in humans or animals following inhalation, oral, or dermal exposure to antimony. However, muscle and/or joint pain was reported in 30-50% of subjects injected with fuadin or astiban, which were administered as part of the therapeutic treatment of schistosomiasis. The joint pain was more severe in subjects receiving fuadin, although' the dose was four times less than the astiban dose (Zaki et al. 1964). This suggests differences in the toxicity of the different antimony compounds, which might explain why musculoskeletal effects have not been observed in humans by the other routes of exposure. Myoneural junction swelling was observed in mice following injection with potassium antimony tartrate (Mansour and Reese 1965). A more complete description of the myopathy observed in these mice is given in the neurological section. Because of the limited human and animal data, it is difficult to determine the significance of this effect to human health.

Hepatic Effects. Hepatic effects have not been observed in humans exposed to antimony. Parenchymatous degeneration in the liver was observed in rats and guinea pigs exposed to airborne antimony trioxide for 30 weeks or to antimony trisulfide for 5 days (Brieger et al. 1954; Dernehl et al. 1945). However, liver effects have not been observed in more recent intermediate-chronic-duration inhalation studies (Bio/dynamics 1985, 1990; Groth et al. 1986; Watt 1983; Wong et al. 1979). Swelling of the hepatic cords has been observed in rats orally exposed to metallic antimony or antimony trioxide (Hiraoka 1986). Since hepatic effects have not been observed in humans and animal data are inconsistent, it is not known if liver damage will occur in humans exposed to antimony.

Renal Effects. Renal effects have not been reported in humans following inhalation, oral, or dermal exposure to antimony. Tubular dilation and degeneration of the tubular epithelium have been observed in rats, rabbits, and guinea pigs acutely exposed to airborne antimony trisulfide or stibine gas (Brieger et al. 1954; Price et al. 1979). Kidney effects have not been reported in animals exposed to airborne antimony for an intermediate or chronic duration (Bio/dynamics 1985, 1990; Groth et al. 1986; Watt 1983; Wong et al. 1979). The kidneys were not examined in the oral and dermal exposure studies. The relevance of this effect to human health is not known.

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Dermal/Ocular Effects. Dermatosis and ocular irritation have been reported in humans following exposure to airborne antimony and antimony via injection (Potkonjak and Vishnijich 1983; R&es 1953; Stevenson 1965; Zaki et al. 1964). The dermatitis associated with exposure to airborne antimony was seen more often during the summer months and in workers exposed to high temperatures. It is probably the result of antimony being dissolved in sweat and penetrating the sweat glands (Stevenson 1965). Dermal and ocular exposure to antimony has resulted in minimal skin and eye irritation in animals and the formation of cataracts (Bio/dynamics 1985, 1990).

Other Systemic Effects. Hyperplasia of the reticuloendothelial cells in the peribronchiolar lymph nodes was observed in rats chronically exposed to airborne antimony (Bio/dynamics 1990). This effect is probably the result of the clearance of antimony particles from the lungs, and thus it is an effect that is likely to occur in humans.

Immunological Effects. Immunological effects have not been studied in humans or animals following inhalation, oral, dermal, or parenteral exposure to antimony.

Neurological Effects. Neurological effects have not been observed in humans following inhalation, oral, dermal, or parenteral exposure to antimony. Muscle weakness, difficulty in moving, and abnormal gait have been observed in animals follotring oral and dermal exposure to antimony trioxide (Fleming 1982; Myers et al. 1978). Decreased motor efficiency and dystonic torsion of the limbs were observed in mice receiving intraperitoneal injections of potassium antimony tartrate (Mansour and Reese 1965). Degenerative changes in the anterior horn cells of the lumbar cord, edema with hydropic degeneration in the sciatic nerve, and swelling of the myoneural junction were also observed in this mouse study. Because neurological effects have been observed in three species of animals (dogs, rats, and mice), these effects may also occur in humans exposed to high levels of antimony.

Developmental Effects. An increase in the number of spontaneous abortions was observed in women exposed to airborne antimony in the workplace. The exposure level was not reported in this study. No overt developmental effects were observed in the children of these women (Belyaeva 1967). No gross abnormalities were observed in the offspring of rats exposed to low levels of antimony trichloride in the drinking water (Rossi et al. 1987). The likelihood of antimony-induced developmental effects occurring in humans is not known.

Reproductive Effects. Human exposure to antimony dust in the workplace has resulted in disturbances in menstruation (Belyaeva 1967). In animals, the failure to conceive and metaplasia in the uterus have been observed following inhalation exposure to antimony trioxide (Belyaeva 1967). No information on the potential of antimony to cause reproductive effects in animals following

oral or dermal exposure was located. These data suggest a potential for antimony to cause reproductive effects in humans.

Genotoxic Effects. No in vivo genotoxicity studies were located. The results of in vitro genotoxicity studies are presented in Table 2-5. Positive results for chromosome breakage in human leukocytes were found (Paton and Allison 1972). Positive results were also found for DNA damage, viral transformation, and chromosomal aberrations. Gene mutation and transformation tests were negative. Because of the limited in vitro genotoxicity data and the lack of in vivo tests, the genotoxicity of antimony in humans cannot be determined.

Cancer. No information on the carcinogenic potential of antimony in humans was located. Inhalation exposure to antimony trioxide or antimony trisulfide produced lung tumors in rats (Groth et al. 1986; Watt 1980, 1983; Wong et al. 1979). Lung tumors were not observed in the Bio/dynamics (1990) study. The Watt (1980, 1983) and Bio/dynamics (1990) studies used similar concentrations of antimony trioxide. However, lung cancer was observed only in the Watt (1980, 1983) study. A possible explanation for the conflicting results is differences in the amount of antimony that was deposited in the lungs. Bio/dynamics (1990) asked the pathologist who examined the histopathology slides from the Watt (1980, 1983) study to also examine the slides from the Bio/dynamics (1990) study. The pathologist determined that the degree of pigmentation in the lungs (indicative of the amount of antimony in the lungs) was greater in the lungs of rats from the Watt (1980, 1983) study compared to those from the Bio/dynamics study (1990). However, why there were differences in antimony deposition and/or clearance between the studies is not known. The deposition and clearance of antimony depends on particle size (Felicetti et al. 1979b; Thomas et al. 1973). The smaller particles are deposited in the lower respiratory tract and are slowly cleared from the lung. The larger particles are deposited in the upper airways and are cleared more efficiently from the lung. Thus, antimony with smaller particle sizes come into contact with the lung tissue for a longer period of time; this may influence the carcinogenic potential. Because of differences in the methods used to assess particle size distribution between these two studies, a comparison of particle size distribution between the studies can not be made. The carcinogenicity of inhaled antimony also may vary with the chemical form of antimony, which will affect the solubility of antimony and, thereby, lung retention. The carcinogenicity of inhaled antimony is probably related to its deposition in the respiratory tract and the resulting reactive processes induced by its presence in the lung tissue. These include macrophage infiltration and fibrosis, typical of pneumoconiosis. The lung carcinogenicity of inhaled antimony may not, therefore, predict carcinogenic potential for other routes of exposure. Antimony has not produced cancer in rats or mice exposed by the oral route (Kanisawa and Schroeder 1969; Schroeder et al. 1968, 1970). There may be physiological differences in the deposition and clearance of antimony from the lungs between humans and rats. Thus, it is

TABLE 2-5. Genotoxicity of Antimony *In Vitro*

Species (test system)	End point	Results: With activation	Results: Without activation	Reference	Form
Prokaryotic organisms:					
Bacillus subtilis	DNA damage	No data	+	Kanematsu et al. 1980	Trioxide; trichloride; pentachloride
Mammalian cells:					
Syrian hamster embryo cells	Viral transformation	No data	+	Casto et al. 1979	Acetate
Chinese hamster ovary cells	Gene mutation	-	-	Tu and Sivak 1984	Thioantimonate
	Chromosomal aberrations	+	+	Tu and Sivak 1984	Thioantimonate
BALB/c-3T3	Transformation	No data	-	Tu and Sivak 1984	Thioantimonate
Human leukocytes	Chromosome breakage	No data	+	Paton and Allison 1972	Sodium tartrate

+ = positive
- = negative

difficult to assess carcinogenic potential of antimony in humans. No information of carcinogenic potential of antimony following dermal application of antimony was located.

2.5 BIOMABKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

A biomarker of exposure is a xenobiotic substance or its metabolite(s) or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s) or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time biologic samples can be taken. It may be difficult to identify individuals exposed to hazardous substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as copper, zinc, and selenium). Biomarkers of exposure to antimony are discussed in Section 2.5.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are often not substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by antimony are discussed in Section 2.5.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, biologically effective dose, or target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 2.7, "POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE."

2. HEALTH EFFECTS

2.5.1 Biomarkers Used to Identify and/or Quantify Exposure to Antimony

Elevated blood, hair, urine, and fecal levels of antimony indicate high exposure to antimony. A significant correlation exists between the level of pentavalent antimony (N-methylglucamine antimonate) administered intraperitoneally to humans and antimony levels in hair (Dorea et al. 1989). However, Dorea et al. (1989) only tested two levels of antimony (10 and 20 mg antimony/kg/day). Factory workers exposed to antimony trioxide (0.042-0.70 mg antimony/m^3) had elevated urine and blood antimony levels (Ludersdorf et al. 1987). Antimony levels in the urine and blood were 1.1 and 0.9-5.0 μg/L, respectively, compared to 0.6 μg/L urine levels and 0.4 μg/L blood levels in unexposed workers. Animal data suggest that urine and blood levels remain elevated several days after exposure (Felicetti et al. 1974b).

No effect biomarkers that could be used to implicate exposure to antimony were found.

2.5.2 Biomarkers Used to Characterize Effects Caused by Antimony

No toxic symptoms specific to antimony exposure have been identified. Toxic effects that reportedly occur in humans include pneumoconiosis, altered EKG readings, and gastrointestinal effects. No quantitative biomarkers associated with these effects are known.

2.6 INTERACTIONS WITH OTHER CHEMICALS

No information on the influence of other compounds on the toxicity of antimony was located.

2.7 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

Individuals with existing chronic respiratory or cardiovascular disease or problems would probably be at special risk, since antimony probably exacerbates one or both types of health problems. Because antimony is excreted in the urine, individuals with kidney dysfunction may be unusually susceptible.

2.8 MITIGATION OF EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to antimony. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to antimony. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice.

2. HEALTH EFFECTS

Adverse health effects in humans following antimony exposure appear to target on the respiratory and cardiovascular systems. Eye and skin irritation have also been noted.

Human exposure to antimony may occur by inhalation, ingestion, or by dermal contact. Mitigation approaches to reduce absorption of antimony have included general recommendations of separating contaminated food, water, air, and clothing from the exposed individual. Externally, exposed eyes and skin are flushed with a clean neutral solution such as water or normal saline. Administration of water or milk and a cathartic such as magnesium sulfate has been recommended by Stutz and Janusz (1988) for treatment following oral exposure to antimony. This would reduce the concentration of antimony in the stomach, but is not likely to affect its intestinal absorption. Administration of activated charcoal following exposure to organic compounds is thought to be effective in preventing absorption (Stutz and Janusz 1988).

Antimony may be found in the blood and urine several days after exposure. It also can be found in the hair (Dorea et al. 1989). Pentavalent antimony is rapidly excreted in humans following intravenous or intramuscular administration, with greater than 50% excreted in the urine 6 hours after injection (Goodwin and Page 1943; Rees et al. 1980). Trivalent antimony is not as rapidly excreted in the urine and is primarily excreted in the feces over a 24 hour period of time as noted after intraperitoneal administration in laboratory animals (Edel et al. 1983).

Chelation therapy with British anti-Lewisite (BAL) may be the most effective mitigation approach following absorption of trivalent antimony compounds into the blood stream (Ellenhorn and Barceloux 1988; Haddad and Inchester 1990). Antimony can covalently bind with sulfhydryl groups. BAL, a dithiol compound with two vicinal sulfur atoms, competes with the critical binding sites that may possibly be responsible for the toxic effects. There is no evidence that BAL is useful following stibine gas exposure (Ellenhorn and Barceloux 1988).

Dialysis may be the most effective method for mitigation of pentavalent antimony. Pentavalent antimony in the blood resides mainly in the plasma in an easily dialyzable form (Edel et al. 1983); Dialysis treatment following exposure to trivalent antimony may not be as effective. The majority of trivalent antimony found in blood is incorporated into the red blood cell fraction in a hard dialyzable form (Edel et al. 1983).

2.9 ADEQUACY OF THE DATABASE

Section 104(i)(S) of CERCLA as amended directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of antimony is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP),

is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of antimony.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

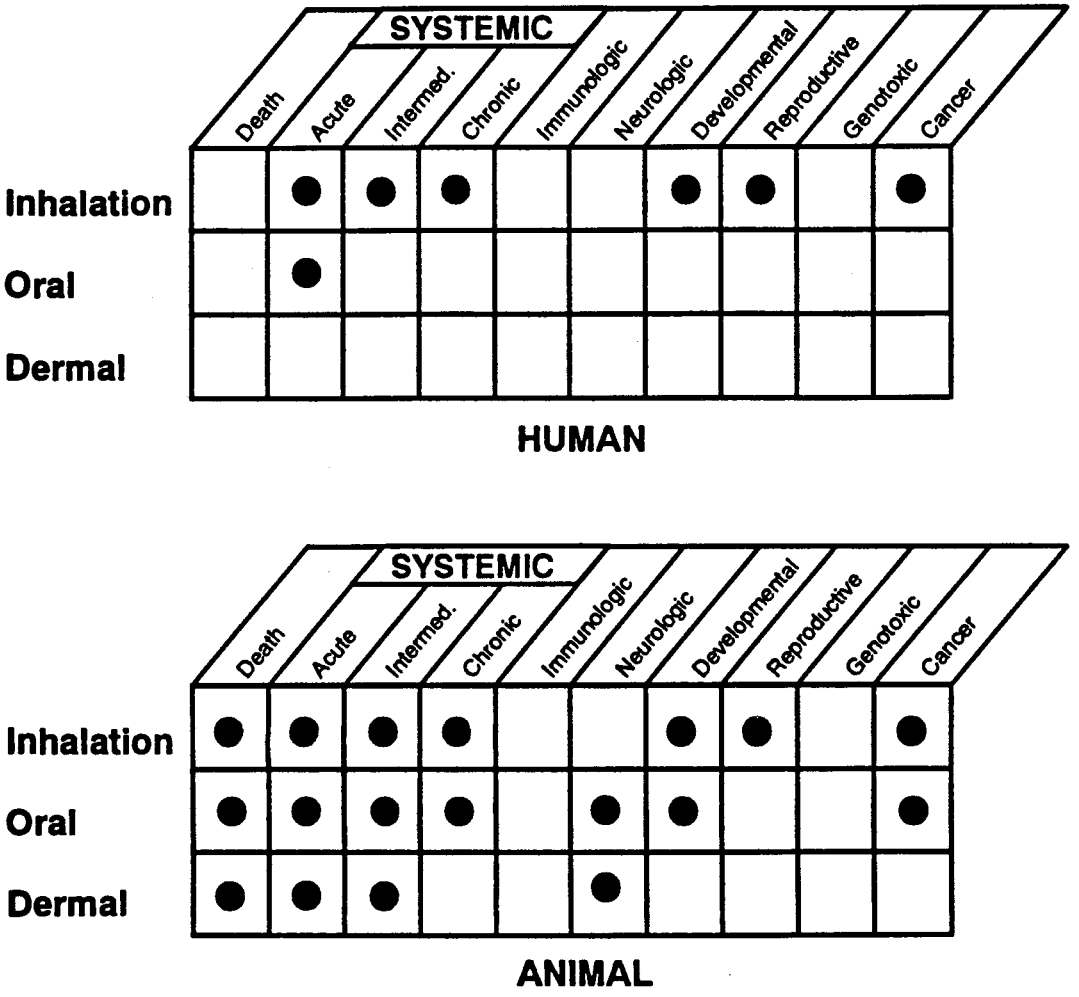
2.9.1 Existing Information on Health Effects of Antimony

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to antimony are summarized in Figure 2-3. The purpose of this figure is to illustrate the existing information concerning the health effects of antimony. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not imply anything about the quality of the study or studies. Gaps in this figure should not be interpreted as "data needs" information.

As seen in Figure 2-3, information on the health effects of antimony in humans following inhalation, oral, or dermal exposure is limited. The inhalation data consist of several reports of workers exposed to inorganic forms of antimony. However, most of these studies are incomplete because the workers were exposed to a variety of compounds or the exposure level was not reported. One oral study involving accidental drinking of lemonade contaminated with potassium antimony tartrate was located. The dermal data on humans is limited to a study in which antimony was applied to the skin of volunteers.

As compared to the human data, more complete information on the systemic health effects of antimony in animals was located. Although there are several reliable intermediate and chronic duration studies that examined numerous toxicological end points following exposure to airborne inorganic trivalent antimony (primarily antimony trioxide), most of the studies utilized rats. One inhalation reproductive/developmental study was located. Several studies that examined the toxicity of metallic antimony, antimony trioxide, antimony trichloride, and potassium antimony tartrate via oral exposure were located. Sensitive measurements of cardiovascular toxicity were not examined in most of these studies. One developmental toxicity study in rats was located; internal examination of pups was not located. The acute and intermediate toxicity of dermally applied antimony trioxide, antimony oxide, and antimony thioantimonate has been examined. However, these studies did not examine the systemic toxicity of antimony; they were designed to assess the dermal and/or ocular toxicity of antimony.

FIGURE 2-3. Existing Information on Health Effects of Antimony



● Existing Studies

2.9.2 Data Needs

Acute-Duration Exposure. Information on the target organs of acute exposure in humans to antimony is limited. Based on one human study, the gastrointestinal tract appears to be a target following inhalation exposure to antimony (Taylor 1966). Animal studies have shown that the respiratory tract and cardiovascular, hepatic, and renal effects occur after exposure to airborne antimony (Brieger et al. 1954; Price et al. 1979). The respiratory and cardiovascular effects occur at a lower exposure levels than those associated with gastrointestinal effects in humans. An acute inhalation MRL could not be derived from this animal data because serious myocardial effects were observed at the lowest exposure level tested. The gastrointestinal tract also appears to be a target in humans following oral exposure to antimony. This is based on a report of workers who accidentally drank lemonade contaminated with potassium antimony tartrate (Dunn 1928). An acute oral MRL could not be derived from this study. Acute animal data also suggest that the gastrointestinal tract is a target system (Fleming 1982; Houpt et al. 1984; Myers et al. 1978). However, two of the three acute animal studies did not perform complete histological examinations, thus there may be other target organs that have not been identified (Fleming 1982; Houpt et al. 1984; Myers et al. 1978). There is no information on the target organs in humans following dermal exposure to antimony. Application of antimony to the skin or eyes of animals results in mild irritation (Gross et al. 1955; Horton et al. 1986; Myers et al. 1978; Wil Research Lab 1979). A majority of the animal studies only examined the skin or eyes following dermal/ocular exposure to antimony. Toxicokinetic data that might allow route-to-route extrapolations of health effects were not found. Knowledge about the acute toxicity of antimony is important because people living near hazardous waste sites might be exposed to antimony for brief periods. Information about the toxicity of different antimony compounds, as well as differences in valence states, was not located. Additional acute-duration studies by the inhalation, oral, and dermal routes would provide information on differences in the potency of various antimony compounds, as well as on the thresholds for systemic toxicity due to acute-duration exposure to antimony.

Intermediate-Duration Exposure. Human target organs/systems following exposure to airborne antimony include the respiratory tract and gastrointestinal tract and skin (Brieger et al. 1954; Renes 1953; Stevenson 1965). No reports of health effects in humans following oral or dermal exposure were located. Animal data suggest that the heart and respiratory tract may be targets following inhalation exposure (Brieger et al. 1954; Bio/dynamics 1985). Developmental and reproductive effects have also been reported in animals (Belyaeva 1967). There is no information on human health effects following oral exposure to antimony. Oral exposure of animals to antimony has resulted in adverse health effects on the liver, cardiovascular system, gastrointestinal tract, and mild hematological effects (Angrisani 1988; Fleming 1982; Hiraoka 1986; Marmo et al. 1987; Rossi et al. 1987; Smyth

and Thompson 1945; Sunagawa 1981). No reports of human health effects following dermal exposure were located. No adverse health effects were observed in animals following intermediate duration dermal exposure (Horton et al. 1986). EKG readings are a sensitive indicator of myocardial damage; however, in the oral and dermal intermediate-duration studies this end point was not examined. Because the exposure levels tested were higher than the threshold levels for respiratory tract effects, and/or because EKG readings were not taken, inhalation and oral MKLs could not be derived. Toxicokinetic data that might allow extrapolation of health effects across routes of administration were not located. Information on the relative toxicity of the different antimony compounds has not been assessed. Intermediate-duration studies by inhalation, oral, and dermal routes would provide information on the thresholds for systemic toxicity, as well as on the differences in the potency of various antimony compounds. This information could be relevant to human exposure because people living near hazardous waste sites may be exposed to a variety of antimony compounds for an intermediate-duration.

Chronic-Duration Exposure and Cancer. There are several human and animal chronic inhalation studies that indicate the targets appear to be the respiratory tract, heart, eye, and skin (Brieger et al. 1954; Cooper et al 1968; Potkonjak and Pavlovich 1983). However, functional changes in the cardiovascular system were not assessed in the animal inhalation studies (Bio/dynamic 1990; Gross et al. 1952; Groth et al. 1986; Watt 1980, 1983; Wong et al. 1979). A no-effect level (NOEL) for respiratory or cardiovascular effects following exposure to antimony was not identified in the available literature. The NOEL is an important level in evaluating the risk of exposure to antimony, and it can be used along with protective uncertainty factors to help determine the amount of antimony humans can be exposed to without experiencing health effects. The chronic inhalation studies in animals examined only the toxicologic effects in rats; thus, interspecies differences could not be assessed. No target organs were identified in humans or animals following oral exposure to antimony (Kanisawa and Schroeder 1989; Schroeder et al. 1968, 1970). In addition, the data from the oral and inhalation studies were insufficient for deriving a chronic MEL. There is no information on the health effects in humans and animals following dermal exposure. Well-designed oral experiments, using several exposure levels and measuring all sensitive toxicological end points, would provide information on the health effects associated with long-term exposure to antimony. Chronic toxicity information is important because people living near hazardous waste sites might be exposed to antimony for many years.

No studies were located regarding the carcinogenicity of antimony in humans following inhalation, oral, or dermal exposure. Evidence for the carcinogenicity of inhaled antimony in animals is mixed. Two studies reported lung tumors in rats exposed to relatively low levels of antimony trioxide (Groth et al. 1986; Watt 1983; Wong et al. 1979). A study using a similar exposure level did not find evidence of carcinogenicity (Bio/dynamics 1990).

2. HEALTH EFFECTS

Differences in the amount of antimony deposited and/or cleared from the lungs were reported. It is not known if the conflicting results were due to differences in particle sizes. A study comparing the effects of different particle sizes would determine if the particle size of the inhaled antimony determines the carcinogenic potential of antimony. The increased incidence of lung tumors appears to be route specific. There is no evidence of increased incidence of cancer in humans as a result of oral exposure to antimony. The oral cancer data in animals are limited to studies that used very low levels of antimony (Kanisawa and Schroeder 1989; Schroeder et al. 1968, 1970). Oral studies have shown that antimony tends to accumulate in the liver and gastrointestinal tract (Ainsworth 1988; Sunagawa 1981); it is not known if this results in cancer. No dermal cancer studies in humans or animals was located. Oral and dermal studies in rodents using several exposure levels including the maximum tolerated level would provide useful information because prolonged exposure to antimony in humans may occur.

Genotoxicity. There are no in vivo genotoxicity studies in humans or animals. In vitro studies using human leukocytes were positive for chromosome breakage (Paton and Allison 1972). Results were mixed in in vitro studies using mammalian cells (Casto et al. 1979; Tu and Sivak 1984), and positive for DNA damage in Bacillus subtilis (Kanematsu et al. 1980). Additional in vitro and in vivo genotoxicity studies would enable better estimation of the actual genotoxic threat posed by antimony to people exposed in the environment.

Reproductive Toxicity. Women exposed to antimony in the workplace have reported menstrual disturbances and a higher incidence of spontaneous abortions as compared to nonexposed workers (Belyaeva 1967). From this report it is unclear what the exposure level was, whether the women were exposed also to other compounds, and whether the controls had comparable jobs. Reproductive effects (failure to conceive, uterine metaplasia) have been observed in rats exposed to airborne antimony (Belaeva 1967). In addition, studies on the distribution of antimony following oral administration in animals have shown high levels of antimony in the testes (Sunagawa 1981). It is not known whether these high levels of antimony could result in functional changes. There are no data on reproductive effects following oral or dermal exposure to humans and animals. There are insufficient toxicokinetic data to make route-to-route extrapolation. A well-designed study to assess the effects of orally or dermally administered antimony on reproductive performance would provide information on possible reproductive effects that might be relevant to humans.

Developmental Toxicity. An increased number of spontaneous abortions was observed in women exposed to antimony in the workplace (Belyaeva 1967). However, there are several limitations to this study, as discussed above in the reproductive toxicity section. No overt developmental effects were observed in the offspring of these women. Developmental effects were not observed in the offspring of rats exposed orally to antimony trichloride

(Rossi et al. 1987). An animal study has shown that antimony is not efficiently transported across the placenta (Gerber et al. 1982). However, there is evidence of high levels of antimony in unexposed newborn nursed by exposed female mice (Gerber et al. 1982). A study in which animals are exposed throughout gestation and lactation would provide information on the potential of antimony to result in developmental effects in humans.

Immunotoxicity. Immunotoxicity following inhalation, oral, or dermal exposure have not been studied in humans or animals. Immunological end points should be examined in the intermediate or chronic studies, especially since antimony has been shown to accumulate in the spleen (Sunagawa 1981).

Neurotoxicity. Neurotoxic effects have not been observed in humans following inhalation, intramuscular, and intraperitoneal exposure to antimony. Neuromuscular effects have been observed in animals following oral, dermal, and intraperitoneal administration (Fleming 1982; Mansour and Reese 1965; Myers et al. 1978). Furthermore, myopathy has been observed in mice exposed via intraperitoneal injection (Mansour and Reese 1965). Although this effect has not been observed by other routes of exposure there is no reason to suspect that it would not occur. Sensitive tests of neurophysiological function may detect early sign of neurotoxicity following inhalation, oral, or dermal exposure to antimony.

Epidemiological and Human Dosimetry Studies. There are several epidemiological occupational exposure studies (Brieger et al. 1954; Cooper et al. 1968; Potkonjak and Pavlovich 1983; RBnes 1953; Stevenson 1965). However, most of these studies are incomplete because the exposure level and/or particle size of the airborne antimony was not reported and/or the workers were often exposed to a variety of other compounds. In addition, cardiovascular toxicity, a sensitive end point of antimony toxicity, was not always assessed. No epidemiological or human dosimetry studies in which individuals were exposed to antimony orally or dermally were located. Epidemiological studies would be useful in order to determine the effects of long-term exposure on humans, with particular attention paid to cardiovascular and respiratory effects. If a cause/effect relationship was established between antimony exposure and health effects in humans, monitoring of individuals living near hazardous waste sites could be performed in order to verify that exposure levels do not exceed recommended limits and that body tissue and fluid levels remain below potentially hazardous levels.

Biomarkers of Exposure and Effect. Because antimony is not catabolized in the body, metabolites could not be used as a biomarker. Thus, the only biomarker of exposure would be measurement of antimony itself. Antimony levels are increased in the blood, urine, and feces following exposure to antimony (Cooper et al. 1968; Edel et al. 1983; Felicetti et al. 1974a, 1974b; Gerber et al. 1982; Goodwin and Page 1943; Ludersdorf et al. 1987; Rees et al. 1980). However, because antimony is poorly absorbed from the lung,

measurement of antimony levels in body fluids may not reflect the exposure level of airborne antimony (Felicetti et al. 1974a, 1974b; Thomas et al. 1973). The relationship between exposure level and concentration of antimony in various body fluids has not been established. Development of a biomarker with more exposure/dose data would aid in future medical surveillance that could lead to better detection of exposure.

No antimony-specific biomarkers of effects have been identified. Future studies on the toxicity of antimony should use several antimony exposure levels, this may lead to the identification of subtle biochemical or physiological biomarkers of effects.

Absorption, Distribution, Metabolism, and Excretion. There is some information on the toxicokinetic properties of antimony following oral or inhalation exposure in humans and animals (Ainsworth 1988; Cooper et al. 1968; Edel et al. 1983; Felicetti et al. 1974a, 1974b; Gerber et al. 1982; Gerhardsson et al. 1982; Goodwin and Page 1943; Ludersdorf et al. 1987; Rees et al. 1980; Sumino et al. 1975; Sunagawa 1981; Thomas et al. 1973). However, there is limited comparative information on the absorption, distribution, and excretion of different antimony compounds. Furthermore, the site and mechanism of antimony absorption from the gastrointestinal tract has not been elucidated. The influence of nutritional factors as well as the presence of food in the gastrointestinal tract on absorption are not known. Information on the absorption, distribution, or excretion of antimony following dermal application is not known. In addition, a study on the effect of oxidation state on the cellular uptake of antimony and the effect of water solubility of an antimony compound on lung retention/absorption would provide useful information on the toxicity of different antimony compounds. A study that examined these aspects of antimony would be useful in assessing the potential target organs following dermal exposure to antimony.

Comparative Toxicokinetics. Species differences in the toxicokinetics of antimony have been identified (Ainsworth 1988; Felicetti et al. 1974a; Gross et al. 1955; Thomas et al. 1973). However, the absorption, distribution, and excretion of antimony following oral or inhalation exposure in humans is not known. Thus, it is not possible to decide which animal species is the best model for assessing the toxicity of antimony. Information on the behavior of antimony in humans would be useful.

Mitigation of Effects. Chelation therapy with BAL has been shown to effectively mitigate the toxicity of trivalent antimony compounds in humans (Ellenhorn and Barceloux 1988; Haddad and Winchester 1990). Although BAL has been found to form stable chelates in vivo with antimony, it is not known if there are adverse side effects associated with the treatment. Studies examining the effectiveness of chelating agents and possible side effects would be helpful in determining the most effective treatment for antimony toxicity. Antimony is widely distributed throughout the body. The hair and

skin contain the highest levels of antimony. The adrenal glands, lung, large intestine, trachea, cerebellum, and kidneys also contain relatively high levels of antimony (Muramatsu and Parr 1988; Sumino et al. 1975). No information on methods of mitigating body stores were located. Studies that examined such methods would be useful in the treatment of antimony toxicity.

2.9.3 On-going Studies

NTP (1990) has recently completed a 14-day drinking water study in which groups of male and female Fischer 344 rats and B6C3Fl mice were given drinking water containing potassium antimony tartrate. The doses in rats were 0, 16, 28, 59, 94, and 168 mg/kg/day; for mice, they were 0, 59, 98, 174, 273, and 407 mg/kg/day. In rats, the only effects observed were an increase in relative liver and kidney (females only) weights in the high dose group. Focal areas of ulceration with necrosis and inflammation of the squamous mucosa of the forestomach were observed in the high dose mice. A final report of this study is currently not available.

NTP (1990) has also completed a 13-week intraperitoneal injection study. In this study, inflammation and/or fibrosis of the liver were observed in mice dosed with 60 mg/kg potassium antimony tartrate every other day. Degeneration was evident in the kidneys of male rats dosed with 24 mg/kg every other day. A final report of this study is currently not available.

Genotoxicity tests were negative in Salmonella typhimurium strains TAl00, TA1535, TA97, or TA98 for antimony potassium tartrate with and without metabolic activation (NTP, 1990).

NIOSH is conducting an epidemiological study using a cohort of antimony smelter workers to determine the possible association between exposure to antimony and the risk of developing lung cancer (Federal Research in Progress 1989).

3. CHEMICAL AND PHYSICAL INFORMATION

There are many compounds, complexes, and alloys of antimony that occur naturally or are man-made. The chemical identity and physical chemical properties of all of these forms of antimony cannot be discussed in detail.

3.1 CHEMICAL IDENTITY

Antimony is in the fourth row of group 5A in the periodic table, residing between arsenic and bismuth. It displays four oxidation states: Sb(-3), Sb(0), Sb(+3), and Sb(+5). The +3 state is the most common and stable. Antimony is sometimes referred to as a metalloid, indicating that it displays both metallic and nonmetallic characteristics.

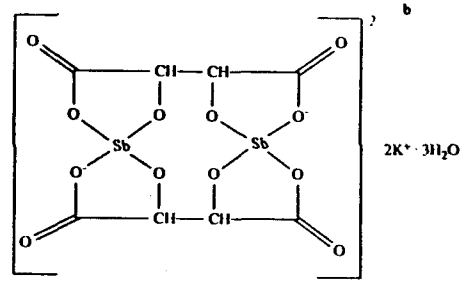
Metallic antimony is the only allotropic form of antimony that is stable under normal conditions. Two unstable allotropes exist: yellow and black amorphous forms (Herbst et al. 1985). Metallic antimony is a very brittle, moderately hard metal (Herbst et al. 1985). It is occasionally found uncombined in nature (Carapella 1978). Antimony has two stable isotopes with mass numbers 121 and 123, with natural abundances of 57.25% and 42.75%, respectively (Carapella 1978). One radioactive isotope, Sb125, is a fission product released in nuclear explosions or nuclear fuel reprocessing plants and has a half-life of 2.7 years (Weast 1988). Data on the chemical identity of antimony, antimony pentasulfide, antimony pentoxide, antimony potassium tartrate, antimony trichloride, antimony trioxide, antimony trisulfide, and stibine are shown in Table 3-1.

3.2 PHYSICAL AND CHEMICAL PROPERTIES

The physical and chemical properties of antimony, antimony pentasulfide, antimony pentoxide, antimony potassium tartrate, antimony trichloride, antimony trioxide, antimony trisulfide, and stibine are given in Table 3-2. Antimony metal is stable under ordinary conditions and is not readily attacked by air or water (Herbst et al. 1985). It is a poor conductor of heat and electricity (Weast 1988). Antimony is positioned after hydrogen in the electrochemical series and therefore will not displace hydrogen ions from dilute acids. It is not affected by cold, dilute acids (Windholz 1983). Simple antimony cations (i.e., Sb^{+3} and Sb^{+5}) do not occur in solution, but hydrolyzed forms (e.g., $Sb(OH)_6^-$) are found. The dominant species in the pH range typical of natural environments are $Sb(OH)_3$, in the case of trivalent antimony, and $Sb(OH)_6^-$ for pentavalent antimony (Bodek et al. 1988). In oxidizing environments, $Sb(OH)_6^-$, is the dominant species for pH greater than 3, whereas $Sb(OH)_3$ is dominant under relatively reducing conditions. The concentration of antimony is too low in natural water for Sb_2O_3 or Sb_2O_5 to precipitate out.

Antimony trioxide is dimorphic, existing as a cubic form, senarmontite, and an orthorhombic form, valentinite. The cubic form is stable at temperatures below 570°C (Freedman et al. 1978). Antimony trioxide is

TABLE 3-1. Chemical Identity of Antimony and Compounds

Characteristic	Antimony	Antimony pentasulfide	Antimony pentoxide	Antimony potassium tartrate
Synonym(s)	Antimony black; stibium; antimony regulus	Antimonial saffron; antimonic sulfide; antimony red; antimony; golden antimony sulfide; antimony persulfide[g]	Antimonic oxide; antimony pentaoxide; diantimony pentoxide; stibic anhydride; antimonic anhydride; antimonic acid[g]	Antimonyl potassium tartrate; potassium antimonyl tartrate; tartox; tartrated antimony; Potassium antimony tartrate; tartar emetic
Registered trade name(s)	No data	No data	No data	No data
Chemical formula	Sb[e]	S_5Sb_2[g]	O_5Sb_2[b]	$C_8H_4K_2O_{12}Sb_2 \cdot 3H_2O$[b]
Chemical Structure	Sb	No data	No data	
Identification Numbers				
CAS registry	7440-36-0	1315-04-4	1314-60-9	28300-74-5
NIOSH RTECS	CC4025000	CC6125000[g]	CC6300000[g]	CC6825000
EPA hazardous waste	No data	No data	No data	No data
OHM/TADS	7216595	No data	No data	7217219
DOT/UN/NA/IMCO shipping	UN 2871	No data	No data	UN 1551
HSDB	508	No data	No data	1428
NCI	No data	No data	No data	No data

[a]All information obtained from HSDB 1989,1991 except where noted
[b]Windholz 1983
[c]Freedman et al. 1978
[d]Avento and Touval 1980
[e]Weast 1989
[f]Dean 1985
[g]RTECS 1991
[h]Cotton and Wilkinson 1966

CAS = Chemical Abstracts Service; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; RTECS = Registry of Toxic Effects of Chemical Substances

TABLE 3-1 (Continued)

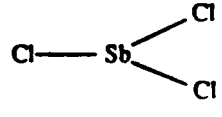
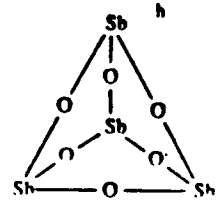
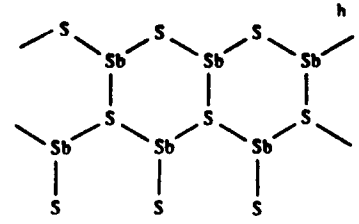
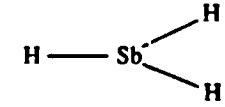
Characteristic	Antimony trichloride	Antimony trioxide	Antimony trisulfide	Stibine
Synonym(s)	Antimonous chloride; antimony butter; antimony(III) chloride; trichlorostibine; chlorid antimony	Antimonious oxide; antimony oxide; diantimony trioxide[b]; flowers of antimony[b]; antimony sesquioxide[c]; senmarmontite; valentinite; antimony white; antimony peroxide; timothox; exitelite	Antimonous sulfide; antimony glance; antimony orange; antimony crimsom; antimony sesquisulfide; antimony sulfide; antimony vermilion; stibite; antimony needles;	Antimony hydride; antimony trihydride; hydrogen antimonide
Registered trade name(s)	No data	HP[d]; LP[d]; KR[d]; White Star[d]; White Star M[d]; KR-LTS[d]; Thermoguard S[d]; Thermoguard L[d];H grade[d]; L Grade[d]; Fire Shield H[d]; Fire Shield L[d]; Montana Brand[d]	No data	No data
Chemical formula	Cl_3Sb	O_3Sb_2	S_3Sb_2	H_3Sb
Chemical structure				
Identification numbers:				
CAS registry	10025-91-9	1309-64-4	1345-04-6	7803-52-3
NIOSH RTECS	CC4900000	CC5650000	CC9450000	WJ0700000
EPA hazardous waste	No data	No data	No data	No data
OHM/TADS	7217220	7217222	No data	No data
DOT/UN/NA/IMCO shipping	UN 1733	UN1549; antimony compounds, inorganic, solid, NOS; NA 9201; antimony trioxide	UN 1549; antimony compounds, inorganic, solids, NA 1325 Antimony sulfide, solid	UN 2676
HSDB	439	436	1604	785
NCI	No data	C55152	No data	No data

TABLE 3-2. Physical and Chemical Properties of Antimony and Compounds

Property	Antimony	Antimony pentasulfide	Antimony pentoxide	Antimony potassium tartrate
Atomic/molecular weight	121.75	403.80	323.5 (anhydrous)	333.93
Color	Silvery white	yellow	yellow	colorless
Physical state	Solid	Solid	Solid	Solid
Valence state	0	+5	+5	+3
Melting point, °C	630.5	75°C decomposes	380°C decomposes[f]	100°C(-1/2H_2O)
Boiling point, °C	1,750; 1,325[b]; 1,635[d]	No data	No data	No data
Density (g/cm^3)	6.684 (25°C); 6.688 (20°C)[b]	4.12	3.78	2.6
Odor	No data	Odorless[d]	No data	Odorless[e]
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Taste	No data	No data	No data	Sweetish, metalic[d]
Taste threshold	No data	No data	No data	No data
Solubility:				
Water	Insoluble	Insoluble	Very slightly soluble	83 g/L (cold)
Organic solvents	No data	Insoluble in alcohol	No data	Insoluble in alcohol Soluble in glycerine
Partition coefficients:				
Log octanol/water	No data	No data	No data	No data
Log K_{oc}	No data	No data	No data	No data
Vapor pressure, mmHg	1 (886°C)[e]	No data	No data	No data
Henry's law constant	No data	No data	No data	No data
Autoignition temperature	No data	No data	No data	No data
flash point	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data
Conversion factors				
ppm to mg/m^3	None[g]	None[g]	None[g]	None[g]
Explosive limits	No data	No data	No data	No data

[a]All information obtained from Weast 1988 except where noted
[b]Herbst et al. 1985
[c]Freedman et al. 1978
[d]Windholz 1983
[e]HSDB 1989,1991
[f]SAX 1984
[g]Since these substances exist in the atmosphere in the particulate state, the concentration is expressed as mg/m^3.

TABLE 3-2 (Continued)

Property	Antimony trichloride	Antimony trioxide	Antimony trisulfide	Stibine
Atomic/molecular weight	228.11	291.50	339.69	124.77
Color	Colorless	White (senarmontite); Colorless (valentinite)	Black (stibnite) Yellow-red (amorphous)	Colorless[c]
Physical state	Solid	Solid	Solid	Gas
Valence state	+3	+3	+3	-3
Melting point, °C	73.4	656	550	-88
Boiling point, °C	283, 222.6[c]	1,550 sublimes; 1,425[c]	1,150	-17[c]
Density (g/cm^3)	3.140 (25°C)	5.2 (senarmontite); 5.67 (valentinite)	4.64 (stibinite) 4.12 (amorphorus solid)	2.204 (-17°C)[c]
Odor	Sharp, unpleasant	Odorless[e]	No data	Disagreeable, like hydrogen sulfide[c]
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Taste	No data	No data	No data	No data
Taste threshold	No data	No data	No data	No data
Solubility:				
Water	6,016 g/L (0°C)	Very slightly soluble	1.75 mg/L (18°C)	4.1 g/L (0°C)
Organic solvents	Soluble in ABS alcohol, tartaric acid, methylene chloride, benzene, acetone	Soluble in tartaric acid; acetic acid, hydrochloric acid	Soluble in alcohol Insoluble in acetic acid	Soluble in carbon disulfide ethanol[c]
Partition coefficients:				
Log octanol/water	No data	No data	No data	No data
Log K_{oc}	No data	No data	No data	No data
Vapor pressure, mmHg	1 mmHg (49.2°C, sublimes)	1 mmHg (574°C)[e]	No data	No data
Henry's law constant	No data	No data	No data	No data
Autoignition temperature	No data	No data	No data	No data
flash point	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data
Conversion factors				
ppm to mg/m^3	None[s]	None[s]	None[s]	1 ppm stibine = 5.1 mg/m^3
Explosive limits	No data	No data	No data	No data

amphoteric; it is soluble in bases and hydrochloric and some organic acids, but not dilute sulfuric or nitric acids (Cotton and Wilkinson 1966). Nitric acid and other strong oxidizing agents convert antimony trioxide to antimony pentoxide, Sb_2O_5, which is acidic (Carapella 1978; Cotton and Wilkinson 1966).

Antimony forms complex ions with organic and inorganic acids; one of the best known is the tartrate. In the presence of sulfur, stable complexes such as $Sb2S_4^{2-}$ may form (Bodek et al. 1988).

Stibine, SbH_3, is a gaseous antimony compound in which antimony is in the -3 valence state. It is formed by the action of acids on metal antimonides or antimony alloys, reduction of antimony compounds, or the electrolysis of acidic or basic solutions where antimony is present in the cathode. As such, there is a danger of stibine being liberated from overcharged lead storage batteries in which antimony is alloyed into the lead. Stibine slowly decomposes into metallic antimony and hydrogen. It is readily, and sometimes violently, oxidized by air to form antimony trioxide and water (Freedman et al. 1978).

4. PRODUCTION, IMPORT, USE, AND DISPOSAL

4.1 PRODUCTION

Antimony, while not abundant, occurs in over 100 minerals; the more important of these are sulfides and, to a lesser extent, oxides of Sb(III), and combinations with lead, copper, and silver. Stibnite (Sb_2S_3) is the predominant ore, followed in importance by valentinite (Sb_2O_3), senarmontite (Sb_2O_3), stibiconite ($Sb_2O_4.H_2O$), bindheimite ($Pb_2Sb_2O_7.nH_2O$), kermesite (Sb_2S_2O), tetrahedrite ($Cu_2Sb_2O_7$), and jamesonite ($2PbS.Sb_2S_3$). It also occurs uncombined as the metal (Herbst et al. 1985; Miller 1973). The antimony content of commercial ores ranges from 5% to 60% (Carapella 1978). The world's largest concentrations of antimony are found in China, Bolivia, U.S.S.R., Republic of South Africa, and Mexico (Miller 1973).

Between 1977 and 1984, the amount of antimony mined in the United States ranged from 311 to 760 metric tons (Llewellyn 1988; Plunkert 1982). The low of 311 metric tons occurred in 1980, and was the result of an 8-month work stoppage at the Sunshine Mine in the Coeur d'Alene district of Idaho. Data for 1985, 1986, and 1988 were withheld to avoid disclosing proprietary company data (Anonymous 1989a, 1989b; Llewellyn 1988). This reflects the fact that there were two or fewer active antimony mines in the United States. In 1987, the Sunshine Mine was closed, and there was no antimony mined in the United States. In recent years, the principal domestic ore producers have been the Sunshine Mining Company in Idaho and the United States Antimony Corporation in Montana. The Sunshine Mining Company principally mines tetrahedrite in conjunction with silver mining, and the United States Antimony Corporation principally mines stibnite. Antimony is also produced as a byproduct of the smelting of primary lead ores.

The primary antimony output from smelters has generally been rising in recent years; smelter outputs were 14,922, 16,309, 18,795, and 18,692 metric tons in 1985, 1986, 1987, and 1988, compared with 11,644 metric tons in 1977 (Anonymous 1989a; Llewellyn 1988; Plunkert 1982). According to the U.S. Bureau of Mines, nine companies produced primary antimony metal and metal oxide products in the United States in 1987. These were ASARCO Incorporated, Omaha, Nebraska; Amspec Chemical Corp., Gloucester City, New Jersey; Anzon America, Laredo, Texas; Chemet Co., Moscow, Tennessee; Laurel Industries Inc., La Porte, Texas; McGean Chemical Co., Inc., Cleveland, Ohio; M&T Chemicals Inc., Baltimore, Maryland; Sunshine Mining Co., Kellogg, Idaho; and U.S. Antimony Corp, Thompson Falls, Montana (Llewellyn 1988). Most of the primary antimony generated in the United States was generated as the oxide. In 1985 and 1986, 13,969 and 15,898 metric tons of antimony oxide were produced, compared with 855 and 343 metric tons of metal, respectively. In 1987 and 1988, 18,758 and 18,226 metric tons of the oxide were produced, respectively; production figures for the metal were withheld to maintain business confidentiality (U.S. Bureau of Mines 1989a). In 1988, U.S. primary antimony consumption was 12,060 metric tons, of which 2,121 metric tons were metal, 9,432 metric tons were oxide, and 42 metric tons were sulfide (U.S. Bureau of

Mines 1989a). Consumption trends have generally paralleled those of production. Table 4-l lists the number of facilities in each state that produced, imported, processed, or used antimony and its compounds in 1987, according to reports made to EPA under requirements of Section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 and subsequently published in the Toxic Chemical Release Inventory (TRI). Companies are required to report if they produced, imported, or processed 75,000 or more pounds of antimony and its compounds or used more than 10,000 pounds. Also included in Table 4-l is the maximum amount of antimony and its compounds these facilities have on site and whether the antimony is produced, processed, or used at the site. The quality of the TRI data must be viewed with caution since 1987 data represent first-time, incomplete reporting by these facilities. Not all facilities that should have reported have done so.

Almost as much antimony is produced from scrap as from ore. Antimony produced from secondary sources is primarily derived from "old scrap," generally consisting of lead battery plates, type metal, and bearing metal. "New scrap," which is derived from drosses and scrap generated during fabrication, constituted 8.6% of the secondary antimony in 1987 (Llewellyn 1988). Secondary antimony is chiefly consumed as antimonial lead; a small percentage goes into the production of other lead- and tin-based alloys. Secondary antimony production has stabilized and recovered slightly after a long decline; it was 27,780 metric tons in 1977, 12,886 metric tons in 1983, and 13,635, 14,082, 15,189, and 16,172 metric tons in 1985, 1986, 1987, and 1988, respectively (U.S. Bureau of Mines.1989a; Llewellyn 1988; Plunkert 1982).

The method of treating antimony ore after mining depends on the type of ore and its antimony content. High grade (45-602;) sulfide ore that is free from lead and arsenic can be extracted by melting, a technique known as liquation. In this process, the ore is heated to 550-660°C in a crucible or reverberatory furnace in a reducing atmosphere. High-grade sulfide **ores** can also be reduced to the metal by iron precipitation, a technique in which the ore is heated with iron scrap, which replaces the antimony. High-grade oxide ores are reduced with charcoal in a reverberatory furnace. An alkaline flux is used to reduce volatilization losses, which may be as high as 12-20X. The method of choice for low-grade (less than 20%) sulfide ores is volatilizing roasting. In this process, the ore is heated to about 5OO"C, and the amount of oxygen is controlled, so that the antimony trioxide formed is volatilized and then recondensed. Intermediate-grade sulfide or oxide ores are generally handled by smelting (Carapella 1978; Herbst et al. 1985). The impure metal may be refined by pyrometallurgical techniques or electrolysis. For further details on antimony mining, ore processing, recovery, and refining, see Carapella (1978) or Herbst et al. (1985).

Antimony trioxide is produced by oxidizing antimony sulfide ore or antimony metal in air at 600-800°C (Avento and Touval 1980).

TABLE 4-1. Facilities That Manufacture, Process, or Use Antimony and Compounds[a]

State[b]	No. of facilities	Range of maximum amounts on site in thousands of pounds[c]	Activities and uses[d]
AL	5	10-9,999	2, 3, 7, 8
AR	3	1-999	3, 8, 9, 13
AZ	3	1-999	1, 2, 3, 4, 5, 6, 7, 9
CA	16 (1)[e]	0-999	1, 2, 3, 4, 7, 8, 9, 12
CO	2	10-99	1, 2, 4, 7, 8, 9
CT	5	10-999	1, 2, 3, 4, 7, 8, 9
DE	1	0.1-0.9	2, 9
FL	3 (1)[e]	1-9	3, 8, 11, 13
GA	8	0.1-99	1, 3, 4, 7, 8, 9
ID	3	1-9,999	1, 5, 8, 9, 12
IL	11 (1)[e] 11	1-999	2, 3, 7, 8, 9, 10,
IN	12	0.1-999	8, 9, 11
KS	4	10-99	8, 11
KY	12	0-9,999	1, 2, 3, 4, 5, 6, 7, 8, 9, 11
LA	8 (1)[e]	0.1-999	7, 8, 9, 10, 11
MA	10 (1)[e]	1-999	1, 2, 3, 4, 7, 8, 9
MD	4	1-9,999	1, 2, 3, 4, 7, 8
MI	7 13	1-999	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11,
MN	5	0-99	6, 8, 9
MO	8 (1)[e]	1-499,999	1, 2, 3, 4, 5, 7, 8, 9
MS	5 (1)[e]	0-99	8, 9
MT	1	10,000-49,999	1, 2, 3, 4, 7
NC	9 13	0-999	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11,
NE	3	1-9,999	1, 2, 3, 4, 5, 7, 8, 9
NJ	22 (3)[e]	0.1-999	2, 3, 4, 6, 7, 8, 9, 10, 11

TABLE 4-1 (Continued)

State[b]	No. of facilities	Range of maximum amounts on site in thousands of pounds[c]	Activities and uses[d]
NM	1	10-99	8, 9
NY	8	0.1-49,999	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11
OH	34 (2)[e]	1-9,999	1, 2, 3, 4, 7, 8, 9, 10, 13
OK	4	0.1-99	2, 6, 8, 9, 11
OR	1	10-99	7, 9
PA	21	0.1-999	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12
RI	2	0.1-99	8, 9
SC	12	0.1-99	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11,
	12		
TN	7	10-999	1, 2, 6, 8, 9, 13
TX	24 (1)[e]	1-49,999	1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 12
VA	4	1-99	3, 8, 9
VT	4	1-99	8, 9, 12
WA	2 (1)[e]	10-99	9, 11
WI	9	1-999	3, 7, 8, 9
WV	2	10-999	8, 9, 11

[a]TRI 1989
[b]Post office state abbreviations
[c]Data in TRI are maximum amounts on site at each facility.
[d]Activities/Uses:

1. produce
2. import
3. for on-site use/processing
4. for sale/distribution
5. as a byproduct
6. as an impurity
7. as a reactant
8. as a formulation component
9. as an article component
10. for repackaging only
11. as a chemical processing aid
12. as a manufacturing aid
13. ancillary or other use

[e]Number of facilities reporting "no data" regarding maximum amount of the substance on site.

4 . PRODUCTION, IMPORT, USE, AND DISPOSAL

4.2 IMPORT/EXPORT

The United States is not self-sufficient in antimony and depends heavily on imports of both ore and metal. In 1987 and 1988, 24,248 and 30,027 metric tons of antimony, respectively, were imported into the United States for consumption. Of this, 55.3% was as the metal, 31.9% as the oxide, 12.4% as ore and concentrate, and 0.4% as the sulfide in 1988 (U.S. Bureau of Mines 1989a). The respective percentages for 1987 were 36.3, 42.4, 21.1, and 0.3. In 1987, China, Hong Kong, Mexico, and the Republic of South Africa supplied over 72% of this antimony (U.S. Bureau of Mines 1989a). China is, by far, our largest supplier of antimony, most of which is imported as antimony metal. Importation of antimony has generally increased in recent years. In comparison to the figures for 1987 and 1988 given above, imports ranged from 12,098 to 20,086 metric tons between 1977 and 1981 (Plunkert 1982). States that have companies that imported more than 10,000 pounds of antimony and its compounds in 1987 are indicated in Table 4-1.

The United States exported 624 metric tons of antimony metal, alloys, and scrap and 1,227 metric tons of antimony oxide in 1988 (U.S. Bureau of Mines 1989a). Canada is the largest recipient of these exports. No clear trend in antimony exports was evident in the last decade (Llewellyn 1988; Plunkert 1982).

4.3 USE

Antimony is a brittle metal that is not readily fabricated and has no significant use in its unalloyed state. It is alloyed with lead and other metals to increase their hardness, mechanical strength, corrosion resistance, and electrochemical stability or decrease their coefficient of friction. Some antimony alloys expand slightly upon cooling, a valuable property for use in type metal and other castings. Most primary antimony metal, 55% in 1988, as well as most of secondary antimony, goes into antimonial lead, which is used primarily in grid metal for lead acid storage batteries. In this application, the antimony imparts fluidity and electrical stability, and increases the fatigue strength and creep resistance of the lead (Carapella 1978). Other uses in decreasing order of importance are solder, sheet and pipe, bearing metal, and bearings, castings, and type metal. Antimony is also used in ammunition and cable sheathing. Other uses, including nonantimonial lead alloys (e.g., pewter), accounted for 21.7% of 1988 metal consumption (U.S. Bureau of Mines 1989a). The level of antimony in grid metal ranges from 2.5% to 5% (Carapella 1978). Antimony levels in other antimony alloys range up to 23%. High-purity antimony is used as a dopant in semiconductors. Intermetallic compounds of antimony such as aluminum antimonide (AlSb), gallium antimonide (GaSb), and indium antimonide (InSb) are used for thermoelectric devices such as infrared detectors and diodes (Gudzovskij 1983; Herbst et al. 1985).

4 . PRODUCTION, IMPORT, USE, AND DISPOSAL

The most common end-use of antimony compounds is antimony trioxide for fire retardation. In 1985, 15,500 metric tons of antimony oxides, amounting to 85% of production, were consumed for this use (Sutker 1988). Antimony trioxide in a suitable organic solvent is used as a fire retardant for plastics, textiles, rubber, adhesives, pigments, and paper (U.S. Bureau of Mines 1989a). According to Bureau of Mine estimates, 56% of the end-use consumption of primary antimony in the United States was for flame retardants, as opposed to 23% in metal products and 21% in nonmetal products (U.S. Bureau of Mines 1989a). Nonmetal products include enamels for plastics, metal, and ceramics, decolorizing and refining agents in special optical glass and other glasses, stabilizers in plastics, pigments in paints and ceramics, vulcanization agents, ammunition primers, and fireworks (U.S. Bureau of Mines 1989a; Herbst et al. 1985; Ludersdorf et al. 1987). The number of companies in each state that used more than 10,000 pounds of antimony and its compounds in 1987 is included in Table 4-l. The most common general use of antimony and its compounds is as a formulation or article component.

Some trivalent organic antimony compounds (e.g., potassium or sodium antimony tartrate) are used to treat bilharziasis (schistosomiasis) (Swellengrebel and Sterman 1961).

4.4 DISPOSAL

Much of the antimony used in antimonial lead, most of which comes from auto batteries, is recycled. This is evident from the large amount of secondary antimony production. Little information concerning the disposal of antimony and its compounds has been found in the literature. Wastes from mining and smelting are generally disposed of in landfills. This is evident from the amounts of releases to land from companies that produce antimony and its compounds (Section 5.2.1). In addition, many companies transfer their antimony wastes to publicly-owned treatment works or to off-site facilities for disposal. Plastics and articles of clothing that contain small amounts of antimony oxide flame retardants will generally be placed in landfills or incinerated along with normal industrial or municipal trash.

Antimony and its compounds have been designated as priority pollutants by EPA (1988). As such, persons who generate, transport, treat, store, or dispose of antimony-containing material must comply with regulations of the federal Resource Conservation and Recovery Act (RCRA). No limitations on the disposal of antimony ore from mines and mills have been promulgated in the Code of Federal Regulations (EPA 1988).

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Antimony and its compounds are naturally present in the earth's crust. Releases to the environment occur from natural discharges such as windblown dust, volcanic eruption, sea spray, forest fires, and biogenic sources, as well as from anthropogenic activities. Therefore, it is important to consider the background levels that are due to natural sources and distinguish these from higher levels that may result from anthropogenic activities. According to the SARA Section 313 TRI, an estimated total of 3,061,036 pounds of antimony were released to the environment from manufacturing, processing, and antimony-using facilities in the United States in 1987 (TRI 1989). Of these releases, 92.9% was to land, 4.4% was to air, 2.0% was to water, and 0.6% was to underground injection. Table 5-1 lists releases of antimony to air, water, and land from these facilities. Companies above a minimum size are required to report if they produce, import, or process over 75,000 pounds of antimony and its compounds or use in excess of 10,000 pounds. The quality of the TRI data must be viewed with caution since the 1987 data represent first-time, incomplete reporting of estimated releases from these facilities. Not all sources of chemical waste are included, and not all facilities that should have reported have done so.

Most antimony released to the atmosphere from anthropogenic sources results from metal smelting and refining, coal-fired power plants, and refuse incineration. Since antimony is a fairly volatile metal, it will volatilize during combustion processes and subsequently condense on suspended particulate matter that is predominantly less than 1 μm in size. Such fine particles are less efficiently trapped by pollution control devices than are larger particles. In the atmosphere, they tend to settle out slowly; they are also removed by dry and wet deposition. A model that relates particle size to volatility estimates average atmospheric half-lives of 1.9 and 3.2 days for antimony and antimony trioxide, respectively (Mueller 1985). Submicron particles may have atmospheric half-lives as long as 30 days (Schroeder et al. 1987). The long atmospheric half-life and monitoring data indicate that antimony can be transported far from its source (Dutkiewicz et al. 1987; Hillamo et al. 1988). Antimony concentrations in air particulate matter in remote, rural, and U.S. urban areas are 0.00045-1.19, 0.6-7, and 0.5-171 ng/m^3, respectively (Austin and Millward 1988; Schroeder et al. 1987).

The speciation and physicochemical state of antimony are important to its behavior in the environment and availability to biota. For example, the antimony incorporated in mineral lattices is inert and unlikely to be bioavailable. Most analytical methods for antimony do not distinguish the form of antimony. While the total amount of antimony may be known, the nature of the antimony compounds and whether they are adsorbed to other material are not. This information, which is critical in determining antimony's lability and availability, is apt to be site-specific.

TABLE 5-1. Releases to the Environment from Facilities That Manufacture, Process, or Use Antimony and Compounds[a]

State[c]	No. of facilities	Range of reported amounts released in thousands of pounds[b]						
		Air	Underground injection	Water	Land	Total Environment[d]	POTW[e] transfer	Off-site waste transfer
AL	5	0-0	0-0	0-3	0-29	0-30	0-0	0-16
AR	3	0-0	0-0	0-0	0-0	0-0	0-0	0-8
AZ	3	0-14	0-0	0-0	0-1,562	0-1,576	0-0	0-0
CA	16	0-1	0-0	0-0	0-0	0-1	0-5	0-52
CO	2	0-1	0-0	0-0	0-0	0-1	0-0	1-37
CT	5	0-0	0-0	0-0	0-0	0-0	0-0	0-2
DE	1	0-0	0-0	0-0	0-0	0-0	0-0	0-0
FL	3	0-0	0-0	0-0	0-0	0-0	0-0	0-1
GA	8	0-1	0-0	0-0	0-0	0-1	0-1	0-22
ID	3	0-1	0-0	0-3	0-140	0-144	0-0	0-0
IL	11	0-1	0-1	0-0	0-0	0-1	0-0	0-5
IN	12	0-2	0-0	0-0	0-2	0-2	0-32	0-122
KS	4	0-0	0-0	0-0	0-0	0-0	0-0	0-9
KY	12	0-2	0-0	0-1	0-0	0-2	0-0	0-112
LA	8	0-1	0-9	0-26	0-10	0-26	0-0	0-22
MA	10	0-1	0-0	0-0	0-0	0-1	0-0	0-9
MD	4	0-7	0-0	0-0	0-0	0-7	0-3	0-7
MI	7	0-1	0-0	0-1	0-0	0-2	0-0	0-13
MN	5	0-0	0-0	0-0	0-20	0-20	0-0	0-12
MO	8	0-0	0-0	0-0	0-11	0-12	0-0	0-10
MS	5	0-0	0-0	0-0	0-0	0-0	0-0	0-25
MT	1	3-3	0-0	0-0	224-224	226-226	0-0	0-0
NC	9	0-0	0-0	0-0	0-1	0-1	0-0	0-14
NE	3	0-30	0-0	0-0	0-1	0-31	0-5	0-112
NJ	22	0-1	0-0	0-0	0-0	0-1	0-0	0-10
NM	1	0-0	0-0	0-0	0-0	0-0	0-0	0-0
NY	8	0-0	0-0	0-0	0-0	0-0	0-0	0-56
OH	34	0-9	0-0	0-0	0-13	0-13	0-13	0-35
OK	4	0-0	0-0	0-0	0-0	0-0	0-3	0-1
OR	1	0-0	0-0	0-0	0-0	0-0	0-0	0-0
PA	21	0-1	0-0	0-1	0-0	0-1	0-0	0-25
RI	2	0-0	0-0	0-0	0-0	0-1	0-0	0-0
SC	12	0-1	0-0	0-1	0-1	0-1	0-16	0-38
TN	7	0-0	0-0	0-0	0-33	0-33	0-3	0-47
TX	24	0-8	0-8	0-5	0-470	0-475	0-2	0-83
VA	4	0-1	0-0	0-0	0-0	0-1	0-0	0-12

TABLE 5-1 (Continued)

State[c]	No. of facilities	Range of reported amounts released in thousands of pounds[b]						
		Air	Underground injection	Water	Land	Total Environment[d]	POTW[e] transfer	Off-site waste transfer
VT	4	0-0	0-0	0-0	0-0	0-0	0-0	0-0
WA	2	0-0	0-0	0-9	0-54	0-63	0-0	0-13
WI	9	0-1	0-0	0-1	0-31	0-33	0-0	0-89
WV	2	0-0	0-0	0-0	0-0	0-0	0-0	0-10

[a]TRI 1989
[b]Data in TRI are estimated annual releases by each facility.
[c]Post office state abbreviation
[d]The sum of all releases of the chemical to air, land, water, and underground injection wells by a given facility.
[e]publicly owned treatment works

5. POTENTIAL FOR HUMAN EXPOSURE

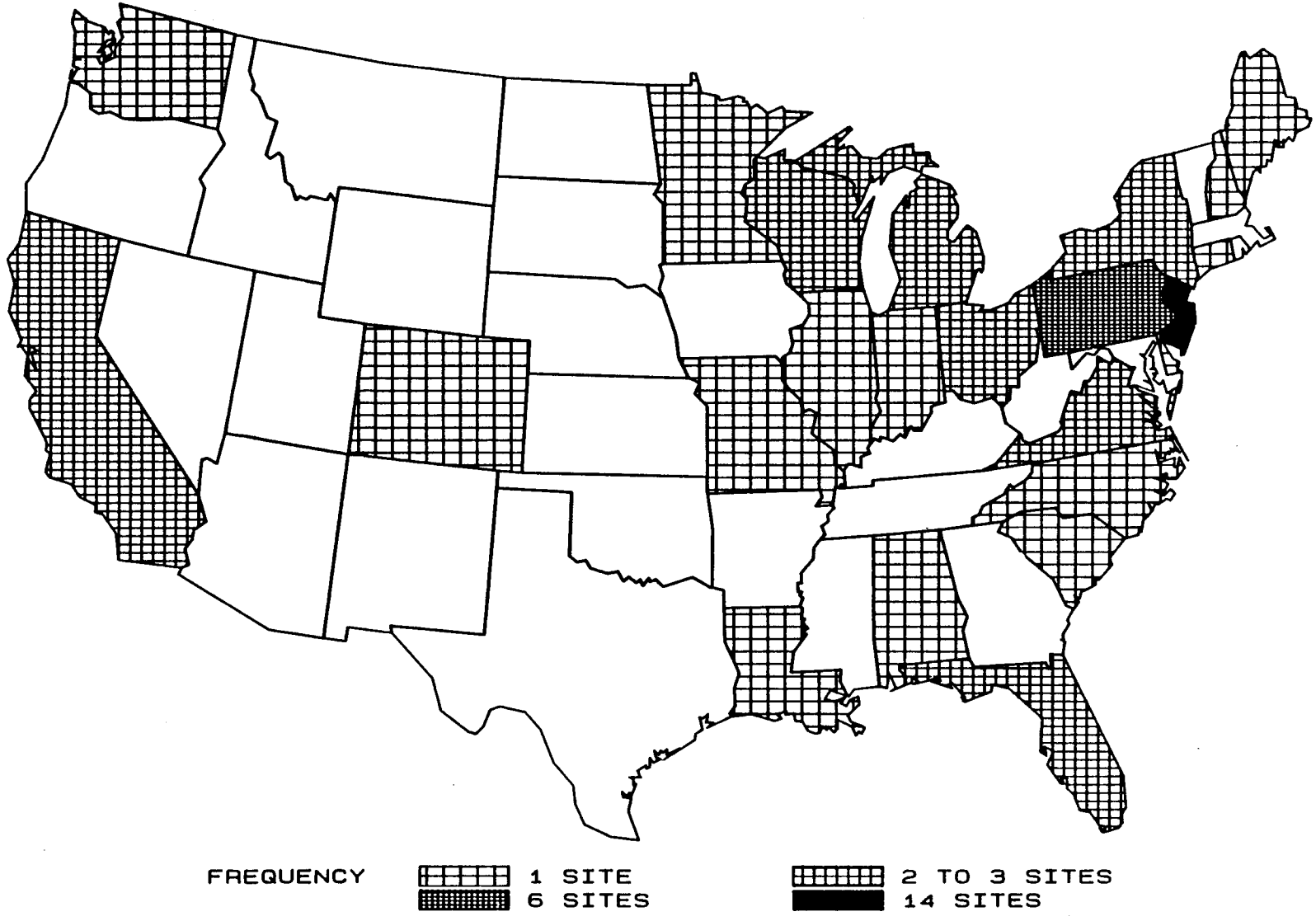
Antimony occurs in soil and rock in very low concentrations; the range of concentration in soil ranges from less than 1 to 8.8 ppm, with a mean of 0.48 ppm (Shacklette and Boerngen 1984). This is the third lowest of 50 elements surveyed by the U.S. Geological Survey. The forms of antimony in various soils and the transformations between these forms is poorly understood. The available data indicate that the lability of antimony may vary considerably according to its environment. In one study, three-quarters of the soil near a smelter site was in a residual (extractible with aqua regia) fraction (Ainsworth 1988). While the concentration of antimony was much lower at control sites, it was in a more labile form; none of the antimony was in the residual fraction. Little is known about the adsorption of antimony to soil. Limited studies indicate that antimony may be fairly mobile under diverse environmental conditions (Rai and Zachara 1984). Since antimony forms anionic species, adsorption should be greatest under weakly acidic conditions. Antimony's adsorption to soil and sediment is primarily correlated with the iron, manganese, and aluminum content; it coprecipitates with hydroxylated oxides of these elements.

As a natural constituent of soil, antimony is transported into streams and waterways from natural weathering of soil as well as from anthropogenic sources. Antimony has a low occurrence in ambient waters. In a survey of dissolved antimony in ambient waters performed by the U.S. Geological Survey, only 6% of 1,077 survey measurements were above the probable detection limit of 5 ppb (Eckel and Jacobs 1989). Antimony concentrations in groundwater appear to be similar to that in surface water. Mean antimony concentrations in surface and groundwater at hazardous waste sites were 27 and 35 ppb, respectively (CLPSD 1989). The forms of antimony and the chemical and biochemical process that occur in the aquatic environment are not well understood. Antimony in both aerobic freshwater and seawater is largely in the +5 oxidation state, although antimony in the +3 oxidation state also occurs in these waters. Trivalent antimony is the dominant oxidation state of antimony in anaerobic water. Antimony can be reduced and methylated by microorganisms in anaerobic sediment, releasing volatile methylated antimony compounds into the water. Methylstibonic acid and dimethylstibonic acid occur in natural water; the monomethyl species is the more abundant one (Andreae 1983; Andreae and Froelich 1984).

EPA has identified 1,177 NPL sites. Antimony and its compounds have been found at 52 of the sites evaluated for the presence of these chemicals (View 1989). However, we do not know how many of the 1,177 NPL sites have been evaluated for these chemicals. As more sites are evaluated by EPA, the number may change. The maximum concentrations of antimony reported at these sites are 2,100 ppb in groundwater, 1,000 ppb in surface water, and 2,550 ppm in soil. The frequency of these sites within the United States can be seen in Figure 5-1.

The general population is exposed to low levels of antimony in ambient air and food. The average intake of antimony from food and water is roughly

FIGURE 5-1. FREQUENCY OF NPL SITES WITH ANTIMONY CONTAMINATION *



* Derived from View 1989

5 μg/day (Iyengar et al. 1987). The intake from breathing air is generally a small fraction of that from ingestion. Exposure from antimony trioxide, which is used extensively in textiles and plastics as a fire retardant, is not expected to be significant. EPA estimates that approximately 4,000 workers may be exposed to antimony and antimony trioxide in production facilities and first-level processing facilities. These workers will have the highest levels of exposure to antimony. The highest air concentration of antimony reported in workplace surveys was 6.2 mg/m^3.

5.2 RELEASES TO THE ENVIRONMENT

Most analytical methods for antimony in environmental samples do not distinguish between antimony metal, antimony trioxide, or other compounds of antimony. More sophisticated methods are required to determine the oxidation state of antimony or the nature of its binding to soil and particulate matter; therefore, it is generally impossible to say with certainty what forms of antimony are released from natural and anthropogenic sources, what forms are deposited or occur in environmental samples, and to what form of antimony people are exposed. The form of antimony will have significant consequences as far as its transport, transformations, and bioavailability are concerned.

5.2.1 Air

Antimony and its compounds are natural components of the earth's crust and releases to the atmosphere result from natural as well as anthropogenic sources. A recent assessment of natural sources of atmospheric trace metals paid special attention to biologic origins of these metals. Nriagu (1989) estimated that 41% of antimony emissions to the air are from natural sources. The natural sources and their median percentage contribution are: wind-borne soil particles, 32.5%; volcanoes, 29.6%; sea salt spray, 23.3%; forest fires, 9.2%; and biogenic sources, 12.1%. Previous assessments indicated that natural inputs were minor compared with anthropogenic ones; in one estimate, anthropogenic sources contributed 39 times more antimony than did natural sources (Lantzy and Mackenzie 1979; Yocom 1983).

Anthropogenic sources of antimony releases to the atmosphere include nonferrous metal mining, nonferrous metal primary and secondary smelting and refining (Crecelius et al. 1974; Pacyna et al. 1984), coal combustion (Gladney and Gordon 1978), and refuse and sludge combustion (Greenberg et al. 1978). Table 5-1 lists the air releases by state from facilities that produce, process, and use antimony and its compounds according to the 1987 TRI (1989). Releases to air total 135,627 pounds. The highest annual release reported by a single company is 29,900 pounds. The industries that contribute the bulk of releases are those that produce antimony and antimony trioxide. Since the TRI does not include emissions from power plants and refuse and sludge incinerators, their estimate of antimony emissions is not complete. European emissions of antimony were estimated at 380 tons for 1979 (Pacyna et al. 1984). Volatile elements and chalcophilic elements (those elements showing an

association with sulfur), like antimony, show large enrichment over crustal abundances in particulate matter emitted from smelting, coal combustion, and refuse combustion. The ranges of enrichment factors reported for these sources are 10,300-l,000,000, 20-140, and 3,000-10,000, respectively (Gladney et al. 1978; Gordon and Sheffield 1986; Small et al. 1981). On a global basis, metal smelting is estimated to make more than double the contribution to atmospheric emissions as other sources, but long-term coal combustion is anticipated to be a dominant factor in future tropospheric antimony levels (Austin and Millward 1988). The antimony that is associated with fine particles (less than 1 pm) tends to result from combustion and other high temperature sources, while that associated with large particles (greater than 10 pm) is likely to originate from wind-blown soil and dust (Schroeder et al. 1987).

It is estimated that 3 g of antimony are released from copper smelters for each ton of copper produced (Pacyna 1984). Typical concentrations of antimony observed in plumes of five copper smelters ranged from 58 to 370 ng/m^3 versus an average background level of 2 ng/m^3 in ambient air (Small et al. 1981). It was found that most of the antimony deposited close to one smelter originated from ground-level emissions (e.g., fugitive emissions) rather than stack emissions (Ainsworth 1988). It was determined that between 57% and 66% of the antimony in the stack of a plant that recycled lead storage batteries was in the vapor form (Craig et al. 1981). This antimony will recondense onto small particles.

The antimony content of 166 American coal samples ranged from 0.1 to 8.9 ppm, with a mean content of 1.15 ppm (Sabbioni et al. 1983). Therefore, it would be expected that coal-fired power plants are a significant source of antimony emissions. A typical, modem coal-fired power plant emits about 31 μg of antimony per kilogram of fuel burned, compared with 3.9 μg/kg for an oil-fired plant (Hasanen et al. 1986). Heavy fuel oil has an antimony content of about 0.067 ppm. Emissions from two units of the Columbia Station coal-burning power plant in Portage, Wisconsin, ranged from 220 to 1,300 ng antimony/m^3 when sampled over 1.5 years (Bauer and Andren 1988). Another investigator reported that a coal-fired power plant with pollution control had stack emissions of 6,800 ng antimony/m^3 (Lee et al. 1975). Antimony in these emissions tends to be associated with fine particles and the surface of particulate matter, consistent with their formation by volatilization and subsequent condensation (Hansen and Fisher 1980). In a modern coal plant, 69% of antimony emissions were associated with particles less than 3 μm in diameter (Sabbioni et al. 1984). Two other studies found that 34-52% of emissions from coal-fired power plants were associated with particles less than 2 μm, and that the mass medium diameter (MMD) of particles from a plant with pollution control devices was 0.6 μm (Gladney et al. 1978; Lee et al. 1975).

A study of emissions from two municipal incinerators in Washington, D.C., showed that refuse incineration can account for the major

portion of antimony in urban aerosols (Greenburg et al. 1978). At least 90% of this antimony is associated with respirable, fine particles that are less than or equal to 2 μm in diameter. The concentration range of antimony in suspended particles from these incinerators was 610-12,600 ppm, with a mean concentration of 2,400 ppm. In performance tests conducted under the Canadian National Incinerator Testing and Evaluation Program, 2.3 g antimony/ton of refuse was emitted under normal operating conditions. Under a range of operating conditions, the amounts ranged from 1.9 to 9.6 g antimony/ton (Hay et al. 1986). Respective stack antimony concentrations were 0.6 and 0.5-2.6 mg/Nm^3 at 12% CO_2, where Nm3 indicates standard cubic meter (1 atmosphere, 25°C). A European study gave emission factors for refuse and sewage sludge incinerators as 4.55 and 1.9 g antimony/ton, respectively (Pacyna 1984). All of the antimony from the stack of a refuse-burning plant was in particulate rather than gaseous form (Braun et al. 1983).

Antimony is a component of ammunition, and therefore antimony may be emitted during the discharge of firearms. This source of emission is inconsequential outdoors. However, in indoor firing ranges, it is a significant source of antimony emission (Dams et al. 1988; Olmez et al. 1985).

An air monitoring study was conducted in 1982 at three sites surrounding the Anaconda Minerals Company smelter facility in Montana. This company had closed 2 years earlier after 8 decades of operation. The study was performed under Superfund to ascertain whether the accumulated heavy metals released during the smelting operations and from tailing ponds might become reentrained by wind and pose a health hazard (Ives et al. 1984). While no antimony was reported to have been produced at the Anaconda Minerals Company facility, many of the metals that were extracted, (e.g., copper, lead, arsenic) are found in association with antimony. The atmospheric levels of heavy metals were very low, indicating that there was not any significant reentrainment of heavy metals from tailing ponds or smelter deposits. The particulate matter examined was generally crustal or carbonaceous in character. Antimony was detected on only 3 of 85 air sampling filters at the three sites.

Stibine may be produced in lead acid battery plants during the formation process. During this process, an electric current is passed through the battery plates, reducing PbO to Pb at the negative plate, and oxidizing PbO to PbO_2 at the positive plate. Hydrogen gas is released that can react with the antimony in the grid metal to form stibine (Jones and Gamble 1984). Stibine may also be formed during remelting of mixed lead-calcium and lead-antimony battery scrap, the former being used for starter batteries. In this process, the intermetallic compound calcium antimonide may be produced in the dross or scum. This compound releases stibine when it comes in contact with water (Ayhan et al. 1982).

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5.2.2 Water

Antimony is a natural constituent of soil and is transported into streams and waterways in runoff either due to natural weathering or disturbed soil. Much of this antimony is associated with particulate matter. In the EPA-sponsored National Urban Runoff Program in which 86 samples of runoff from 19 cities throughout the United States were analyzed, antimony was found in 14% of runoff samples at concentrations ranging from 2.6 to 23 ppb (Cole et al. 1984).

Estimated releases of antimony to water from facilities in the United States that produced, processed, and used antimony in 1987 according to the TRI are found in Table 5-1. These releases totaling 62,328 pounds are much lower than those to air or land. A survey of raw and treated waste water from 20 industrial categories indicates that antimony is commonly found in some waste waters. Those industries with mean effluent levels exceeding 1 ppm in raw waste water were (industry [mean level in ppm]): foundries (l.l), porcelain enameling (1.4), and nonferrous metal manufacturing (5.7) (EPA 1981). The maximum levels in discharges from these industries were 3.4, 22, and 80 ppm, respectively. Additionally, four other industrial categories had maximum concentrations exceeding 1 ppm. These were laundries (2.4 ppm), inorganic chemical manufacturing (1.4 ppm), ore mining and dressing (3.8 ppm), and paint and ink formulation (2.2 ppm). For treated waste water, only porcelain enameling had mean antimony levels in excess of 1 ppm. The levels reached 4.3 ppm.

Domestic waste water is a potential source of antimony in waterways. Concentrations of antimony in influents to 11 municipal waste water treatment plants (POTWs) (155 observations) ranged from 0.0003 to 2.1 ppm; the median value was approximately 0.1 ppm (Minear et al. 1981). Antimony is not well removed in POTWs, and releases from these facilities may contribute to releases of antimony to water (Aulenbach et al. 1987; EPA 1981). The outfall of a sewage treatment plant in Seattle, however, did not appear to make a significant contribution to the antimony levels in the sediment of Puget Sound (Crecelius et al. 1975).

Waste water generated from mining and smelting operations comes from seepage, runoff from tailing piles, or utility water used for mine operation. In addition to liquid effluent from smelting operations, slag may be dumped directly into receiving waters (Crecelius et al. 1975). These discharges largely contain insoluble silicates and sulfides which readily settle out. Total antimony in effluent from a primary aluminum production facility was 40 ppb (Rawlings 1980). Sixty percent of this antimony was subsequently removed by lime coagulation.

One of the potentially dangerous sources of chemical release at waste sites is from leachate. Leachate from three municipal landfills in New Brunswick, Canada, each contained 0.01 ppm of antimony (Cyr et al. 1987).

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The antimony concentration in sediment at two sites below the leachate outfalls was 23.9 ppm (dry weight) and nondetectable, respectively.

5.2.3 Soil

Most of the antimony released to the environment is released to land. According to Table 5-1, which shows the estimated releases in 1987 of domestic industries that produce, process, or use antimony, 2,845,131 pounds of antimony are released to land, constituting 93% of environmental releases reported to TRI (TRI 1989). The industries that release the largest amount of antimony are the smelters that produce antimony and antimony trioxide. Much of this release is slag, which is the residue from smelting operations. Other releases to land include sludge from POTWs and municipal refuse.

An analysis of the concentration of antimony at hazardous waste sites at the Contract Laboratory Program Statistical Database (CLPSD) shows that antimony was reported in 153 of 1,307 soil samples, with geometric mean and maximum levels in positive samples of 8.0 and 330 ppm, respectively (Eckel and Langley 1988). An analysis of these data indicates that 7.3% of the CLPSD samples exceed the number expected to be above the 95% upper confidence limit for background U.S. soils (Eckel and Langley 1988). The CLPSD includes both NPL and non-NPL data. A more recent update of the CLPSD reports a 12.8% occurrence of antimony and a geometric mean concentration of 16.86 ppm (CLPSD 1989). No analysis was performed on these results to indicate what percentage exceed the background levels of antimony normally found in soil.

5.3 ENVIRONMENTAL FATE

It is not always possible to separate the environmental fate processes relating to transport and partitioning from those relating to transformation for a metal and its various compounds and complexes. Part of this problem is that the form of a metal is rarely identified. A change of mobility may result from a transformation of a metal to a more or less soluble form. Adsorption may be the result of the formation of strong bonds (transformation) as well as weak bonds. Information regarding the deposition and general adsorption of antimony is in Section 5.3.1 and information regarding the areas of environmental fate where speciation is discussed is in Section 5.3.2.

5.3.1 Transport and Partitioning

Antimony is released to the atmosphere in the form of particulate matter or adsorbed to particulate matter. It is dispersed by wind and removed by gravitational settling and dry and wet deposition (Schroeder et al. 1987). The removal rate and distance traveled from the source will depend on source characteristics (e.g., stack height), particle size and density, and meteorological conditions.

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Gravitational settling governs the removal of large particles (greater than 5 μm), whereas smaller particles are removed by the other forms of dry and wet deposition. Removal of coarse particles may occur in a matter of hours. Very small particles may have an atmospheric half-life as long as 30 days (Schroeder et al. 1987). Antimony is predominantly associated with small, submicron particles (Stoessel and Michaelis 1986). This is frequently the case with the more volatile metals, such as antimony, that may volatilize during combustion and condense when cooled. A model which relates particle size to volatility estimates an average atmospheric half-life for antimony of 1.9 days; for the more volatile antimony trioxide (see Table 3-2), the estimated half-life is 3.2 days (Mueller 1985). With such an atmospheric half-life, antimony may be transported far from its source. Evidence of this was reported by Animoto and Duce (1987), who stated that the antimony levels in aerosols at the Enewatak Atoll in the tropical North Pacific were higher than those expected from seawater or crustal material.

Metal deposition is characterized by large temporal and spatial variability. Estimated antimony deposition rates in urban areas are 0.006 and 0.004 kg/hectare/year (60 and 40 ng/cm^2/year) for dry and wet deposition, respectively (Schroeder et al. 1987). For remote areas, bulk (wet plus dry) deposition may be as little as 0.00016 kg/hectare/year (1.6 ng/cm^2/year). Rates of air-sea transfer of antimony are similar to the rates of accumulation of antimony in sediment (Arimoto and Duce 1987).

The partitioning between dry and wet deposition depends on the intensity and duration of precipitation, the element in question, its form in the particulate matter, and its particle size. The ratio of wet to dry deposition generally increases with decreasing particle size; therefore, a larger proportion of antimony will be found in rain compared with most other metals. A study of the wet and dry deposition over an 8-week period on an island in the German Bight, which was presumably far from sources, found 87% of deposited antimony dissolved in rain, 11% in particulate matter in rain, and only 2% as dry deposition (Stoessel and Michaelis 1986). In other studies conducted in areas removed from sources of antimony emissions, half of the antimony deposition was in the form of wet deposition (Ainsworth 1988). The total antimony deposition annualized from a B-month study in an industrial area of England where a number of ferrous and nonferrous metal smelting and manufacturing works were concentrated was 1,000 ng/cm^2-year (a factor of 20-40 above nonurban deposition rates) (Pattenden et al. 1982). Of this, 42% represented wet deposition, of which 58% was dissolved antimony.

Antimony released into waterways is generally associated with particulate matter; it is transported to and settles out in areas of active sedimentation such as where a river empties into a lake or bay (Beijer and Jernolov 1986). Similarities in the composition of suspended river sediment and the sediment in bays indicate that the rivers transport the suspended sediment and deposit it in the bottom sediment (Crecelius et al. 1975). Additionally, when a river feeds into an estuary, the salinity changes that

are encountered may affect adsorption to sediment and particulate matter, complexation, and coprecipitation.

Little is known of the adsorptive behavior of antimony, its compounds, and ions. The binding of antimony to soil is determined by the nature of the soil and the form of antimony deposited on the soil. Some forms of antimony may bind to inorganic and organic ligands. On the other hand, a mineral form would be unavailable for binding. Some studies suggest that antimony is fairly mobile under diverse environmental conditions (Rai and Zachara 1984), while others suggest that it is strongly adsorbed to soil (Ainsworth 1988; Foster 1989; King 1988). Since antimony has an anionic character (e.g., Sb(OH)i), it is expected to have little affinity for organic carbon. No information could be found about antimony's adsorption to clay minerals. It is not expected that cation exchange, which generally dominates adsorption to clay, would be important for anionic antimony. Antimony is known to form coprecipitates with hydrous iron, manganese, and aluminum oxides in soil and sediment (Callahan et al. 1978).

The capacity of soil to adsorb antimony and the nature of the bound antimony were evaluated by incubating 200 ppm of antimony potassium tartrate with 5 g samples of soils for 6 days (King 1988). Thirteen soils and subsoils (21 samples) from the southeastern United States (10 mineral and 3 organic) were included in the study. Antimony adsorbed strongly to most soils. The amount of adsorbed antimony ranged from 50% in Lakeland surface soil to 100% in several soils; the median percent adsorption was 93%. The percentage of nonexchangeable (i.e., that not removed with KCl) antimony adsorbed paralleled that of total antimony and ranged from 57% to 99%. Both sorbed and nonexchangeable antimony were negatively correlated to sand content in mineral soil. The soil/water partition coefficient (mmol/kg soil)/(mol/m^3) was 81 and greater than 185 for organic and mineral soils, respectively. Several mineral soils adsorbed 100% of the antimony and were excluded from the averaging. It is not clear what species of antimony was adsorbed in this study. If it was the antimony tartrate ion, the study may not be particularly relevant to other forms of antimony. The mobility of antimony in clay, sandy loam, silt loam, and sand soils was investigated using soil thin-layer chromatography (TLC) (Foster 1989). The antimony was applied as antimony trioxide in a water or 1% HCl suspension and developed with water in 8 hours or less. Despite experimental difficulties, the results demonstrated that there is no general mobility of antimony in any soil. The experimental problems and the fact that small amounts of antimony were found in all zones is possibly due to an unsuitable soil digestion (Ainsworth 1988).

A Superfund site study at a battery reclamation plant showed that while soil and sediment contained high levels of antimony, an aquifer 3 m below the surface contained 0.1 ppm of antimony; no antimony was detected in two deeper aquifers (Trnovsky et al. 1988). Antimony adsorbs strongly to colloidal material in soil. The partition coefficient of antimony to 0.05-0.003 μm

colloids was 1,300. Antimony adsorbed to such material can be transported with the colloids in groundwater (Buddenmeier and Hunt 1988).

Leaching experiments performed with river sediment samples from a mintng district in Idaho indicated that Sb(V) was the major species released during leaching (Mok and Wai 1990). The fraction of antimony leached from sediment with deionized water after 10 days was highly correlated with the free iron and manganese oxide content of the sediment (correlation coefficients of 0.90 and 0.75). Experiments were also performed in which the pH dependence of leaching was determined. The release of antimony from the sediment increased at low pH and increased sharply at high pH. The form of released antimony was also sensitive to pH. At pH 2.7, the bulk of antimony released was as Sb(II1); at pH 4.3, the concentrations of tri- and pentavalent antimony were comparable; and at pH 6.3 and above, Sb(V) was the predominant species.

In order to evaluate the potential for leaching of elements from landspread sewage sludge, Gerritse et al. (1982) studied the adsorption of elements from water, salt solutions, and sludge solutions to sandy and sandy loam top soils. They used metal levels that occur in the solution phase of sewage sludge, lo-100 ppb in the case of antimony. The results indicate that antimony is fairly mobile in these soils. The adsorption constants were approximately 2-16 in the sandy soil and 20 in the sandy loam soil. Although the presence of sludge increases the mobility of many trace elements because of complexation with dissolved organic compounds or increased ionic strength, this did not appear to be the case with antimony (Gerritse et al. 1982). It is not easy to reconcile these results with those of Foster (1989), Ainsworth (1988), Trnovsky et al. (1988), or Van der Sloot et al. (1982). These studies indicated that antimony deposited on the soil surface accumulates primarily in the surface layer, and that aquifers beneath antimony waste piles are not grossly contaminated.

Mobilization of elements deposited on soil in fly ash is a potential source of terrestrial and aquatic pollution. When the alkaline fly ash from a coal-fired power plant was packed in a column and subject to leaching with dilute sulfuric acid, antimony was partially dissolved and removed from the upper layers of ash and deposited and retained on lower sections of ash in the column (Warren and Dudas 1988). It was thought that extractable, surfaceadsorbed antimony in the upper layers of ash was removed by the acid, subsequently precipitated by iron oxyhydroxides, and retained lower down in the column. Other column leaching and shake-flask experiments with coal ash are too complex to summarize; they basically indicate that leaching of antimony is low. Low concentrations of antimony found in groundwater beneath precipitator ash ponds lend field confirmation to the laboratory results (Van der Sloot et al. 1982).

When saline sediment is oxidized, such as when dredged sediment is exposed to oxygen, the pH can become very low (pH 3.1 in a lab experiment), and antimony and other toxic metals may be released (DeLaune and Smith 1985).

This occurs because sediments in estuaries often contain pyrite and other readily oxidizable sulfur compounds; sulfuric acid may be produced and overwhelm the buffering capacity of the sediment. An analogous pH decrease following oxidation was not observed in a freshwater sediment.

Antimony does not appear to bioconcentrate appreciably in fish and aquatic organisms. No detectable bioconcentration occurred during a 28-day test in bluegills (EPA 1980). Only low levels of antimony have been reported in fish and aquatic organisms collected off the coast of Africa, Australia, and the Danube River in Austria (Callahan et al. 1978; Maher 1986). Bioconcentration factors for antimony ranged from 0.15 to 390 (Acquire 1989; Callahan 1978). A study of the distribution of antimony around a smelter site indicated that antimony occurring in plants results from surface deposition. Uptake from soil is minor and appears to be correlated with the amount of available antimony (that which is soluble or easily exchangeable) (Ainsworth 1988). Antimony bioconcentration was measured in voles, shrews, rabbits, and invertebrates around a smelter. Analysis of antimony in organs of the small mammals, compared with estimates of their antimony intake from food, showed that, although the amount of antimony in the organs was elevated, it was low compared to the amount ingested. The results suggest that antimony does not biomagnify from lower to higher trophic levels in the food chain.

5.3.2 Transformation and Degradation

5.3.2.1 Air

Little is known about the chemical forms and physical and chemical transformations of trace elements in the atmosphere. This is primarily because analytical methods provide information concerning the metal content rather than the specific compounds or species. Studies at an antimony smelter suggest that emissions consist of antimony oxide (Ainsworth 1988). In the absence of specific information, it is generally assumed that elements of anthropogenic origin, especially those emanating from combustion sources, are present as the oxide. Windblown dust particles may contain antimony in mineral species, such as sulfides and oxides, and are associated with silicates.

When released into the atmosphere as an aerosol, antimony is believed to be oxidized to antimony trioxide by reaction with atmospheric oxidants. Antimony trioxide particles do not undergo changes in chemical composition, particle size, or morphology after emission; however, a surface coating of sulfate may form (Ainsworth 1988).

5.3.2.2 Water

There is relatively little information available regarding the behavior of antimony in the aquatic environment. Since the dissolved state is the phase in which transfers to suspended matter, organisms, and sediment occur,

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it is especially important to know the oxidation state and forms of the antimony that is dissolved. This is particularly difficult for antimony because the levels of total antimony in water are so low. Thermodynamically, most dissolved antimony in natural waters under aerobic conditions should be present in the +5 oxidation state as antimonate species. At 0.001 M total antimony, the dominant species were $Sb(OH)_6^-$ and $Sb(OH)_5^0$ (Rai et al. 1984). A small quantity of polymeric hydroxy species were found, but these will be less significant when the total antimony concentration is low, such as in natural water. While industrial inputs will commonly contain antimony in the +3 oxidation state (e.g., antimony trioxide), it is not known how fast antimonite would oxidize to antimonate under natural conditions. Under reducing conditions, trivalent species such as $Sb(OH)_3^0$, $Sb(OH)_4^-$, and $Sb_2S_4^{4-}$ may be significant (Andreae and Froelich 1984; Rai et al. 1984).

Antimony compounds may undergo photochemical reactions, but these do not appear to be significant in determining their aquatic fate (Callahan et al. 1978). Antimony trioxide suspensions strongly absorb ultraviolet radiation below 325 nm and darken. The process is reversible, and when the light is removed, the white color slowly returns (Markham et al. 1958). The effect is believed to be due to peroxide radical formation on the crystal surface. Both water and oxygen seem to be necessary for the reoxidation of the reduced antimony.

Antimony can be reduced and methylated by microorganisms in the aquatic environment, similar to arsenic, and become mobilized (Andreae et al. 1983; Austin and Millward 1988). This reaction is most likely to occur in reducing environments, such as in bed sediment. In the case of arsenic, this reaction may be mediated by fungi and bacteria (Beijer and Jernelov 1986), but it is not known whether this is the case with antimony. The resulting trimethylstibine is initially oxidized by atmospheric oxygen to a mixture of trimethylstilbine oxide ($(CH_3)_3SbOH$) and trimethylstibinic acid ($(CH_3)_2SbO_3H$), and then to antimony oxides and insoluble polymers (Parris and Brinckman 1976). The rate constant is estimated to be of the order of 0.1 to 0.2 L/mol-sec. Trimethylstibine has a high vapor pressure, 103 mmHg at 25°C, and might volatilize before it is completely oxidized. The oxidation product, $(CH_3)_3SbO$, is much more soluble than trimethylstibine; therefore, oxidation will reduce volatilization (Callahan et al. 1978). Oxidation of trimethylstibine in the gas phase is very rapid; the rate is 0.11/mmHg-sec or 2000 L/mol-sec. Trimethylstibine has been shown to react with alkyl iodides and bromides; this results in the formation of quaternary salts (Parris and Brinckman 1975). Should antimony occur in a landfill with alkyl halides, the formation of quaternary salts should greatly enhance antimony's mobility.

The chemical and biochemical transformations of antimony in natural waters are not well understood. There are only a few studies that describe the antimony species present in various systems and their transformations. A study of the waters of the Ochlockonee River estuary revealed the presence of Sb(III), Sb(V), methylstibonic acid, and dimethylstibonic acid (Andreae 1983).

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The concentration of all four species increased with the salinity of the water. For freshwater, the concentrations were about 18, 3.3, and 1 ng antimony/L (ppt) for Sb(V), Sb(III), and methylstibonic acid, respectively; dimethylstibonic acid was not detectable. The concentration of Sb(V) and Sb(II1) increased linearly with salinity, reaching 135 and 11 ppt, respectively, in the ocean. An analogous increase in the level of the methylated forms with salinity was nonlinear, suggesting that these forms are consumed in the estuary. In seawater, levels of methylstibonic acid and dimethylstibonic acid were 12.5 and 1.5 ng antimony/L (ppt), respectively. It was reported that the Sb(III) levels were approximately 2% that of Sb(V) in samples of sea water and river water (Mok and Wai 1987). In a sample of river water from the Kellogg mining district in Idaho, the contribution of Sb(III) was only 0.4% (0.03 ppb compared with 7.03 ppb of Sb(V)). More recent studies in Idaho indicated that 1-4% of antimony was in the trivalent form in a river receiving mining leachate, while at a site on an unpolluted fork of the same river, the fraction was 17% (Mok and Wai 1990).

The depth profile of antimony species in the Baltic sea showed that Sb(V) was the most abundant species in the oxic zone, although Sb(III) was detectable throughout the water column (Andreae and Froelich 1984). A maximum for Sb(III) in the oxic zone was sometimes noted in the surface layer and is believed to result from biological activity. There is evidence that phytoplankton can reduce Sb(V) to the Sb(III). Sb(III) decreases to very low levels at the base of the seasonal thermocline and remains low down to the sediment where increasing levels are again observed. Sb(III) only accounts for 44% of the inorganic antimony in the anoxic zone, and speciation in this region is unclear. Thermodynamically, the antimony should be in the trivalent state. Thiocomplexes are thought to account for some of the antimony in this zone. Methylated antimony species existed throughout the water column and made up 10% of total antimony. Monomethyl antimony species were more abundant in surface waters and in the anoxic zone. There was no sharp increase in methyl antimony near the sediment, which would be expected if these species were formed biosynthetically. Since the highest antimony concentration is at the surface, it is unlikely that antimony is taken up by phytoplankton, as is the case with arsenic. A decrease in antimony concentration with depth suggests scavenging by particulate matter and, at lower depths, by iron hydroxyoxides.

Sea water samples off the Belgian coast were analyzed using oxidation, UV irradiation, and anodic stripping voltammetry to distinguish bound antimony and to identify its oxidation state (Gillian and Brihaye 1985). The concentration of total antimony ranged from 0.05 to 0.38 ppb. The study showed that antimony was mainly present as Sb(V), and the percentage complexed to organic matter varied between 20% and 60%. This results is surprising because antimony occurs as anionic species in water and these are not expected to complex with organic matter. These results have not been confirmed by other investigators. The concentration of Sb(II1) was below the detection limit (0.005 ppb) at almost all sites. The exception was the coastal sites

where it ranged up to 0.039 ppb. Antimony found in rain and snow is predominantly in the +5 oxidation state (Metzger and Braun 1986).

Information concerning the behavior of antimony in sediment is extremely limited. Investigators would like to know how strongly antimony is bound in sediment and what the potential is for long-term mobilization. A study was conducted of sediments in Puget Sound, Washington, where a copper smelter discharges large amounts of antimony (Crecelius et al. 1975). In 23 noncontaminated sediment samples, antimony concentrations correlated with organic carbon and fine-grained particles; however, since these sediments are also associated with hydrous-iron oxides, further investigations on the association of antimony in sediment were conducted and showed that less than 10% of the antimony in both contaminated and uncontaminated sediment was bound to readily oxidizable organic matter. Extraction with oxalate and citratedithionite-bicarbonate suggested that roughly half of the antimony in uncontaminated sediment and less than 20% of that in contaminated sediment was bound to extractable iron or aluminum compounds. Most of the antimony in the polluted water was bound to chemically stable slag.

Experiments were performed in which the forms of antimony in sediment were evaluated after the sediment was incubated under anaerobic conditions for 45 days (Brannon and Patrick 1985). Ten dredged, contaminated sediments that were obtained from various locations in the United States were used as is or amended with 75 ppm antimony potassium tartrate. An extraction procedure was used that identified the antimony in interstitial water and in "exchangeable," "easily reducible," and "moderately reducible" sediment fractions. Essentially all antimony in the unamended sediment was in a "moderately reducible" phase (oxalate extraction). The same was generally true for 7 of the 10 sediments that were amended with potassium antimony tartrate. In the other three sediments, the greatest proportion of antimony was in the "easily reducible" fraction. A small fraction of the antimony-amended sediment (but none of the unamended sediment) was contained in the more potentially mobile interstitial water and "exchangeable" fraction. It should be stressed that since the amended samples had higher antimony levels, small percentages in different fractions were more readily detectable than for the unaziended sediment. The high correlation of antimony with the "moderately reducible" fraction indicates that hydrous iron and aluminum oxides were affecting the fixation of antimony. These hydrous oxides are positively charged under environmental conditions and bind the anionic antimony. The samples were subjected to 6 months of aerobic leaching. Unamended samples released antimony very slowly, compared with amended samples, indicating the higher amounts of mobile antimony in the latter samples. Antimony-amended sediments lost from 3.6% to 32% of their antimony during leaching; unamended sediments lost from 0% to 23% of their antimony. The sediment/water distribution coefficient ranged from 3.3 to 27.5 in amended sediments, compared with values up to 1,183 in unamended sediments. The distribution coefficient correlated with iron in both amended and unamended sediment; additionally, it correlated with calcium carbonate in amended sediment. After aerobic leaching, there was

an increase in antimony in the "moderately reducible" phase and a decrease in the "easily reducible" phase that paralleled changes in iron concentrations. Volatile antimony compounds were formed in seven of the amended sediments but in none of the unamended sediments; they escaped through the overlying water independently of the redox state of the water (i.e., aerobic or anaerobic).

When 10 and 100 ppm antimony trioxide with added nutrients was incubated with natural bottom sediment from Puget Sound under aerobic or anaerobic conditions for up to 120 days, three organoantimony biotransformation products were found in solution after 60 days (Martinson 1988). Two of these were identified as methylstibonic acid and dimethylstibonic acid. No determination of rate or conditions affecting the transformation was made. However, it was estimated that much less than 0.1% of the antimony present was transformed.

Few data are available on the removal of antimony in the activated sludge process used in water treatment plants. In one laboratory simulation, mixture of metals at levels considered typical of industrial/domestic sewage (0.1 ppm antimony) was continuously added to the influent of the treatment system. No antimony removal was observed (Kempton et al. 1983).

5.3.2.3 Soil

Little is known about the behavior of antimony on soil during weathering. In aerobic surface soils, oxidation generally occurs. Antimony trisulfide in ore deposits is known to be oxidized by soil bacteria (Ainsworth 1988). Methylated antimony compounds, similar to those formed in sediment, may be formed in waterlogged soil.

The form and availability of antimony in soil is determined by measuring antimony's extractability with different solvents. A sequential extraction procedure was used to determine the form of antimony in soil around a stibnite smelter and to compare it with that found at a control site (Ainsworth 1988). The extraction procedure used could identify the following fractions of antimony: soluble or bound to ion-exchange sites and, therefore, available; bound to carbonates; bound to manganese oxides that are easily reduced; bound to iron oxides that are less easily reduced; bound to organic matter; and residual antimony that was not incorporated into silicates (Ainsworth 1988). Results of the study showed that the distribution pattern among the various fractions was different at the smelter and control sites. Three quarters of the total extracted antimony in surface (0-5 cm) soil near the smelter was in the residual fraction; none of the antimony in the control site was in this fraction. The remainder of antimony in soil from the smelter site was more or less equally distributed among the other fractions. Higher proportions of the antimony at the control site were in the readily available fraction, bound to manganese oxides, or complexed with organic matter, compared to the smelter site. Because of the low concentration of antimony in the control-site soil, fractional determinations are less accurate than for sites near the smelter. For subsoil (greater than 15 cm depth) from the smelter site, less antimony

was found in the residual fraction (62%), and more antimony was in the available fraction, bound to carbonates, or bound to iron oxides than in the surface sample. The factors determining the distribution of antimony between fractions is unclear. The absence of any residual fraction at the control site has been explained by assuming that antimony-containing mineral has been completely broken down.

Near the smelter, antimony deposits have a different character than further away since they are derived from fugitive emissions rather than stack deposition. In a 3-month study of deposition of antimony from the smelter, about one-third to one-half of the deposited antimony at the smelter site was soluble, compared with about one-half to two-thirds at other sites. Since antimony in rain near the smelter site was soluble, it appears that once deposited on soil, antimony rapidly converts to more insoluble forms.

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

5.4.1 Air

There are insufficient data regarding antimony concentrations in the atmosphere for representative general mean or median concentrations to be reported. Antimony concentrations in air particulate matter in remote, rural, and U.S. urban areas are 0.00045-1.19, 0.6-7, and 0.5-171 ng/m^3, respectively (Austin and Millward 1988; Schroeder et al. 1987). No vapor-phase antimony has been reported. Antimony concentrations over the North Atlantic and North Pacific are 0.086 and 0.0037 ng/m^3, respectively (Arimoto and Duce 1987; Austin and Millward 1988). Two values reported for antimony in aerosols in clean continental and marine environments are 0.2 ng/m^3 at the Jungfraujoch in the Swiss Alps and 0.00045 ng/m^3 at American Samoa (Austin and Millward 1988). The mass median aerodynamic diameter of antimony-containing aerosols from a range of areas remote from anthropogenic sources was 0.86 μm (Milford and Davidson 1985). The mass size distribution is bimodal, with the larger peak at about 0.6 μm and a smaller one at about 3 μm. An example of the size distribution of antimony-containing particles removed from anthropogenic sources was obtained in an 8-week study on an island in the German Bight. The concentration of antimony in a size fraction increased as the size decreased. The antimony concentration ranged from 0.03 ng/m^3 for particles greater than 7.2 μm to 0.3 ng/m^3 for particles less than 0.5 μm (Stoessel and Michaelis 1986).

Several studies show that antimony can travel long distances, and that ambient levels may reflect the origin of the air masses. The geometric mean antimony concentration in aerosols at three rural/remote locations in New York state was 1.0, 0.72, and 0.33 ng/m^3 (Dutkiewicz et al. 1987), and the enrichment over crustal abundance ranged from 920 to 1,650. The enrichment factor is smaller but similar to the mean enrichment factor of 1,880 for antimony in 29 cities (Gladney et al. 1984). The high enrichment indicates that the antimony is of anthropogenic origin. An analysis of the New York

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State data using backward-in-time air trajectories is consistent for the Midwest being the dominant source of antimony. An analysis of European sources and wind trajectories further illustrate that antimony may be transmitted over long distances. The average concentration at a city in southern Norway was 0.54 ng/m^3 when the air masses came from the United Kingdom, and 0.07 ng/m^3 when they came from over the Atlantic (Hillamo et al. 1988).

Twenty-four-hour samples collected at 10 locations in Washington, D.C., yielded average antimony concentrations ranging from 1.1 to 3.0 ng/m^3 (Kowalczyk et al. 1982). As a result of a chemical element balance analysis, the three major contributing sources in order of decreasing significance are believed to be refuse incineration, motor vehicles, and coal combustion. In a Houston study, the range of antimony concentrations in fine (0.1-2.5 μm) aerosols was 0-12 ng/m^3, whereas that in particles greater than 2.5 μm was 0-4 ng/m^3 (Johnson et al. 1984). Median, mean, and maximum concentrations of antimony in aerosols at three sites in Quebec, Ontario, and Nova Scotia were 0.05-0.10, 0.11-0.23, and 0.37-2.17 ng/m^3, respectively (Hopper and Barrie 1988). According to the Texas Air Control Board, the first- and secondhighest annual average antimony concentration in Texas between 1978 and 1982 was 452 and 50 ng/m3 at Laredo and Dallas, respectively. The statewide 1978-1982 average was below the minimum detectible mean of 90 mg/m^3 (Wiersema et al. 1984).

Concentrations of antimony in 24-hour air samples at Kellogg, Idaho, which is the site of a large and active nonferrous metal industry, ranged from 5.21 to 1,210 ng/m^3 with a mean of 146 ng/m^3 (Ragaini et al. 1977). Air particulate matter in Tacoma, Washington, 40 km downwind of a copper smelter often have antimony concentrations in excess of 300 ppm (Crecelius et al. 1974). The 6-month average concentration of antimony in air in an industrial area of England where a number of ferrous and nonferrous metal smelting and manufacturing works were concentrated was 40 ng/m^3. This is a factor of 50 higher than that found in rural areas (Pattenden et al. 1982). Antimony was reported in air at one site on the NPL (View 1989). The maximum concentration at the site was 69 ng/m^3.

The mean monthly concentration of antimony in precipitation at Birkenes in southern Norway ranged from 0.2 to 2.3 ppb with a mean of 0.6 ppb (Pacyna et al. 1984). During the same period, the respective air concentrations were 0.19-0.80 and 0.43 ng/m^3. Rain samples were collected during two storms upwind and downwind of a copper smelter in Tacoma, Washington. Antimony in rainwater originated primarily from the smelter. The mean total antimony concentration in rainwater downwind from the smelter was 1.3 ppb; the concentration upwind was 0.03 ppb (Vong et al. 1988). Eighty percent of the antimony in rainwater was dissolved (i.e., passed through a 0.45 μm filter).

Antimony is almost entirely found in the particulate, as opposed to the dissolved fraction of snow (Landsberger et al. 1983). The antimony content of

snow particulate matter in samples from Montreal, Canada, ranged from 4 to 145 ppm. A more recent sampling of snow around Montreal found total antimony concentrations of l-8.7 ppb and enrichment factors of 39-590 (Zikovsky and Badillo 1987).

Antimony is a component of ammunition, and studies have been performed to ascertain the elemental concentrations of antimony in the air of indoor shooting ranges. Antimony might be expected in such situations because it is alloyed with lead in bullets, and lead stibnite and antimony sulfides are used as primers (Dams et al. 1988). After an intensive 3-hour shooting exercise, levels of antimony reached 119 $\mu g/m^3$ or four orders of magnitude over ambient levels (Vandecasteele et al. 1988). An instructor at the shooting range had a time-weighted average (TWA) inhalable antimony concentration of 12.0 $\mu g/m^3$, compared with the threshold limit value (TLV) of 500 $\mu g/m^3$. An American study conducted at the National Guard Armory in Washington, D.C., during routine daytime and gun club use, had antimony concentrations ranging from 57 to 216 $\mu g/m^3$ versus background air ranging from 1.5 to 2.3 $\mu g/m^3$, an enrichment of 9,900 over District of Columbia air) (Olmez et al. 1985). More than 60% of the antimony was associated with respirable particles with an aerodynamic diameter less than 3.5 pm.

5.4.2 Water

Antimony has a low occurrence in ambient waters, and there are few monitoring data with which one can establish a mean value of antimony in surface waters. Eckel and Jacob (1989) gathered water monitoring data from the Water Resources Division of the U.S. Geological Survey covering the period from about 1960 to September, 1988, and found that all but 70 of 1,077 entries for dissolved antimony were below 5 ppb, which was the probable detection limit. The geometric mean and standard deviation of the 70 values above 5 ppb were 12 and 1.93 ppb, respectively. By applying a technique known as censoring, and assuming a log normal distribution for the monitoring data, these investigators determined the population geometric mean and standard deviation for antimony to be 0.25 and 7.16 ppb, respectively. The concentration of dissolved antimony in other rivers reported in the literature include: St. Lawrence River at Massena, New York, 1.62 nM (0.197 ppb); Yukon River 2.73 nM (0.332 ppb); and European rivers less than 0.03-4.43 nM (0.004-0.539 ppb) (Andreae and Froelich 1984). Few rivers have dissolved antimony concentrations below 1 nM (0.120 ppb) (Andreae and Froelich 1984).

The major antimony mining area in the United States was the Kellogg district in northern Idaho, and mining and smelting wastes have been dumped into the South Fork of the Coeur d'Alene River for over 80 years (Mok and Wai 1990). The South Fork joins with the North Fork of the river to form the Main Stem of the Coeur d'hlene River somewhat below Kellogg. Mean and maximum total dissolved antimony concentrations at two sites on the South Fork are 4.3 and 8.2 ppb, respectively. Mean and maximum concentration at six stations on the Main Stem ranged from 0.6 to 1.0 and 0.8 to 1.9 ppb, respectively.

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Those at a station on the unpolluted North Fork were 0.09 and 0.2 ppb, respectively.

The concentration of dissolved antimony in a polluted estuary in Portugal was found to increase with salinity up to 30 parts per thousand and then rapidly decrease (Andreae et al. 1983). The total antimony content of seawater samples off the Belgian coast ranged from 0.05 to 0.38 ppb (Gillain and Brihaye 1985). Filtered and unfiltered coastal marine waters from the North Adriatic contained 0.31 and 45 ppb, respectively (Strohal et al. 1975).

Little information is available concerning the concentration of antimony in groundwater. The range of antimony concentrations reported for antimony in groundwater in Switzerland (0.3-1.0 ppb) was essentially the same as that reported for the nearby Glatt River (0.5-1.2 ppb) (von Gunten and Ku11 1986). The concentration of antimony in groundwater under four retention-recharge basins receiving urban runoff water in Fresno, California, were all less than the 1 ppb detection limit (Nightingale 1987).

Antimony was found in 5.7% and 8.5% of surface waters and groundwaters at hazardous waste sites on the CLPSD (CLPSD 1989). The geometric means of antimony found in positive samples of these media were 40 and 50 ppb, respectively (CLPSD 1989). The CLPSD includes both NPL and non-NPL sites.

Since antimony is used in solder, there has been interest as to whether antimony will leach from pipes soldered with antimony-containing solder into drinking water. Leaching of antimony from tin/antimony (Sn/Sb) solder when it comes in contact with water with pH of 5.2-8.6 was evaluated using loops of pipe containing 20 solder joints (Murrell 1987). Antimony was undetectable (less than 4 ppb) in the water at first, but rose to 10 ppb after 4 days and 68 ppb (at pH 7.4) after 4 weeks. A study was conducted at the University of Washington to evaluate the potential for leaching of metals into drinking water from 95/5 Sn/Sb solder (Herrera et al. 1982). After a series of static and continuous-flow laboratory tests and evaluation of field samples from university buildings, it was concluded that increases in antimony concentration as a result of corrosion and leaching were minimal and would not contribute significantly to dietary antimony intake. Only one of the field samples of standing water from university buildings containing Sn/Sb solder joints was above the detection limit of 0.6 ppb. The sample contained 2 ppb of antimony, one-half of which was dissolved. Examination of the solder joints indicated that a double passivation film of tin monoxide (SnO) and tin dioxide (SnO_2) forms and inhibits leaching.

Sediment is a significant sink for antimony. No information was found on the levels of antimony in pristine sediment. Background antimony concentrations in sediment cores from open water of Lake St. Clair ranged from 0.032 to 0.098 ppm with a mean concentration of 0.061 and 0.068 ppm in sand and silty-clay sediment, respectively (Rossmann 1988). The range of antimony levels in 10 sediments obtained from all over the United States by workers

engaged in research on contaminated, dredged sediment was 0.5-17.5 ppm, and the median concentration was 2.9 ppm (Brannon and Patrick 1985). Sediment samples taken from Puget Sound in Washington (the site of a copper smelter) were analyzed for antimony. This was the only known anthropogenic source of antimony in the area. While the antimony concentration in sediment from noncontaminated areas ranged from 0.3 to 1.0 ppm, these levels rose to 2-3 times background within 8-15 km of the smelter, and up to 12,500 ppm within 1 km of the smelter where considerable amounts of slag were dumped (Crecelius et al. 1975). One hundred and seven core samples of sediment were collected in the delta area of the Coeur d'Alene river in northern Idaho, a primary antimony mining and smelting area in the United States. The sediment was mostly fine silt, which is typical of mine tailings. The top layer of sediment contained 270-900 ppm of antimony with a mean of 512 ppm (Maxfield et al. 1974). More recent monitoring data reported antimony concentrations in sediment of 137, 49-72, and 1.9 ppm on the South Fork, Main Stem, and North Fork of the Coeur d'Alene River, respectively (Mok and Wai 1990). The South Fork receives mining and smelting wastes, and the North Fork is essentially uncontaminated. A sediment profile on one sample showed that the antimony concentration decreased with depth and was between 2 and 3 ppm between 8.5 and 21.5 cm depth.

5.4.3 Soil

A survey of soils throughout the conterminous United States conducted by the U.S. Geological Survey showed that antimony concentrations ranged from less than 1 to 8.8 ppm with an average concentration of 0.48 ppm. This was the third lowest concentration of the 50 elements surveyed (Shacklette and Boerngen 1984). In this survey, samples were taken at a depth of 20 cm at 1,318 sampling sites. Soils not derived from ore-bearing rock or close to industrial sources do not generally contain more than 1 ppm of antimony. Antimony concentrations in igneous rock, shales, limestone, and sandstone have been reported to be 0.2, 1.5, 0.2, and 0.05 ppm, respectively (Ainsworth 1988). Antimony concentrations in 57 sludge-treated soils in an agricultural area west of Toronto in Ontario, Canada, ranged from 0.16 to 0.37 ppm (dry weight) (Webber and Shamess 1987).

A study of the effects of an antimony smelter on soil found that antimony levels exceeding 50 ppm were found only within 2 km of the smelter (Ainsworth 1988); the background antimony concentration was 6.9 ppm. Antimony concentrations in surface soil near the Kellogg Valley, Idaho, the site of one of the nation's largest and richest mining districts, were considerably elevated at seven contaminated sites, with mean and maximum levels of 111 and 260 ppm, respectively (Ragaini et al. 1977). These values represent an enrichment of 1,000 or more over crustal antimony levels. The concentration profiles in core samples sharply decreased with depth. This indicates that the antimony contamination resulted from air deposition. Soil samples taken in Tacoma, Washington, 40 km downwind of a copper smelter, often

had antimony concentrations in the range of 11-109 ppm (dry weight). Natural levels are believed to be 3-5 ppm (Crecelius et al. 1974).

The range of maximum antimony concentration in soil at sites on the NPL was 0.084-2,550 ppm (View 1989). The geometric mean and the maximum concentration of antimony found in soil at hazardous waste sites on the CLPSD is 8.0 and 330 ppm, respectively (Eckel and Langley 1988). Thirteen percent of sites on an updated version of CLPSD contain antimony in soil (CLPSD 1989). The geometric mean of positive samples is 17 ppm. The CLPSD includes both NPL and non-NPL sites. The concentration of antimony in surface soil at the Sapp Battery Superfund site in northern Florida, which housed a facility for recovering lead from auto batteries from 1970 to 1980, ranged from 0.46 to 857.0 ppm (Trnovsky et al. 1988).

A New Zealand study showed that the mean level of antimony in street dust was comparable to that in soil (4.69 ppm versus 5.94 ppm) (Fergusson et al. 1986). The antimony content of household dust, however, was enriched approximately two-fold to 10.0 ppm.

5.4.4 Other Environmental Media

A determination of nutrients in a human diet was conducted by the U.S. Food and Drug Administration (FDA) using mixed diet composites representative of the intake of a 25- to 30-year-old U.S. male. The average concentration of antimony in the diet was 9.3 ppb (dry weight). This corresponds to a daily dietary intake of 4.6 μg of antimony assuming a 3,075 g diet/day (wet weight with a total dry matter of 16.2%) (Iyengar et al. 1987). Another study of antimony in food using a highly sensitive neutron activation procedure found that the average antimony concentration in 12 table-ready foods ranged from 0.22 to 2.81 ppb (Cunningham 1987). The food items used in the study were primarily prepared for FDA's Total Diet Studies program in Kansas City and included meats, vegetables, and seafood. The mean concentration ranges of antimony in meats, seafoods, and vegetables were 0.46-1.15, 0.22-1.81, and 1.09-2.81 ppb, respectively. The results of an earlier investigation of trace elements in food in an FDA basket survey reported that median levels of antimony in eight food groups were less than 10 ppb (wet weight) (Tanner and Friedman 1977). In a separate study, the concentration of antimony in pooled human milk was 13 ppb (dry weight) (Iyengar et al. 1982).

In a comprehensive survey of the presence of heavy metals in sewage sludge, 30 sludge samples from 23 American cities were analyzed (Mumma et al. 1984). The antimony concentration in the sludge samples ranged from 1.3 to 55.7 ppm (dry weight) and had a median value of 7.35 ppm. The highest concentration of antimony was in a sludge sample from Baltimore. This level was more than double that of the second highest sludge sample analyzed. In comparison with the above values, the concentration of antimony in cow manure was 0.43 ppm.

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The concentration of antimony in grass from representative sites in the Kellogg Valley, Idaho (the site of the heavy-metal industry), ranged from 6.2 to 111 ppm. Grass from background sites in the valley that were located 3.3 and 7.8 miles from a smelter contained from 3.5 to 4.5 ppm of antimony (Ragaini et al. 1977). Similar results were found around an antimony smelter in England. The antimony content of grass close to the smelter was 50-300 ppm. The content at control sites ranged from 0.1 to 0.3 ppm (Ainsworth 1988). In comparison with the above values, the concentration of antimony in forage crops was about 0.1 ppm (Ragaini et al. 1977).

Concentrations of antimony in selected species of algae, mollusc tissue, crustacean tissue, and fish muscle from southeastern Australia were 0.094-0.193, 0.031-0.060, 0.018-0.116, and less than 0.009-0.010 ppm (dry weight), respectively. The water collected at the site contained 0.17 ppm of antimony (Maher 1986).

A French study of the metallic content in soaps, shampoos, body oils, and cosmetics found that of all products tested only lacquer contained significant amounts of antimony (1.7 ppm) (Demanze et al. 1984). Antimony was found in high concentrations in certain composite resins used in dentistry. Two materials analyzed in England had mean antimony levels of 288 and 403 ppm (Molokhia and Lilley 1986).

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Antimony occurs naturally in the earth's crust, and the general population is exposed to low levels of antimony in ambient air and food. The average daily intake of antimony from food or water was estimated at 100 μg/day (Wiersema et al. 1984). According to the recent results of Iyengar (1987), the average daily dietary intake is 4.6 μg, ,and, because of the low antimony levels in water, the average daily intake of antimony (by ingestion) is probably not much greater than 5 pg. Laredo, Texas, has the highest annual average concentration of antimony in ambient air (452 ng/m^3). If a person is assumed to inhale 20 m^3 of air/day, this would amount to an average antimony intake of 9.0 μg/day. For a city such as Washington, D.C. (average antimony concentration about 2 ng/m^3), the inhalation intake would be 0.04 μg/day. Only in an extreme situation would the amount of antimony inhaled compare to the amount that is ingested; the amount inhaled is generally much less. Those people who reside near industrial sources of antimony such as smelters, coalfired power plants, and refuse incinerators are exposed to higher levels of atmospheric antimony. People who spend time in shooting galleries are also exposed to higher antimony levels.

EPA does not believe that the antimony found in such consumer products as car batteries and flame retardants in plastics and textiles results in significant consumer exposure (EPA 1983a). When antimony oxide is used as a fire retardant, it is tightly bound into the material; release and subsequent exposure during use is unlikely (EPA 1983a). No antimony leached from several

glass containers used for injectable solutions into distilled water, saline, sodium bicarbonate solution, or hydrochloric acid (Pradeau et al. 1988). This glass contained up to 5 ppm of antimony, and the detection limit for the analytical procedure was 10 ppb. In another study, no antimony was detected in water (pH 3, 7, or 10) kept in a canteen for 24 hours (Augustson 1976).

A National Occupational Exposure Survey (NOES) conducted by NIOSH from 1981 to 1983 estimated that 373,460 workers were potentially exposed to antimony (molecular formula unknown) in the United States in 1981-1983 (NIOSH 1989). The number of workers exposed to antimony trioxide, antimony sulfide, antimony oxide, antimony pentoxide, antimony dialkyldithiocarbamate, and other antimony compounds is estimated to be 226,645. The total estimated number of workers exposed to antimony and all of its compounds is 486,347. Since all of the data for trade-name products that may contain antimony have not been analyzed, this estimate is preliminary. The NOES was based on field surveys of 4,490 facilities. It was designed as a nationwide survey based on a statistical sample of virtually all workplace environments in the United States where eight or more persons are employed in all standard industrial codes (SIC) except mining and agriculture. The NOES database does not contain information on the frequency, concentration, or duration of exposure of workers to any of the chemicals listed therein. These surveys provide only estimates of the number of workers potentially exposed to chemicals in the workplace. EPA states that the NOES figures substantially overestimate occupational exposure to antimony and compounds (EPA 1983a). Most antimony in this country is either smelted from imported ore or impure metallic antimony or recycled from antimony scrap. According to EPA, "mining, hauling, and crushing of ore will be of minor consequence," because ore crushing is done in closed systems, and ore processing is done under wet conditions to minimize dust (EPA 1983a). Following a membership survey, the Antimony Oxide Industry Association (AOIA) reported that 230-240 production workers and l,000-2,000 workers using antimony were exposed to antimony (EPA 1983a). This represented the entire population.of workers potentially exposed to antimonial substances. An independent survey of the three facilities producing and processing antimony metal in 1979 estimated that 2,249 workers were exposed to antimony (EPA 1983a). This estimate included producers and first-level processors of antimony metal into products such as batteries and alloys. Alloys usually contain small amounts of antimony that are most often combined with much larger amounts of lead. Occupational exposure controls that are employed to mitigate lead exposure also protect workers from antimony. Much of the estimated exposure to antimony metal may actually be to antimony oxide; fumes formed when heating the metal (e.g., for carting) are oxidized. The survey also estimated that 1,710-1,880 workers were employed at facilities that manufacture and process antimony trioxide. EPA believes that 200-2,000 workers may be exposed to stibnite, which is used in small quantities in smokes and in priming mixtures for igniting explosives (EPA 1983a). This stibnite is expected to form antimony trioxide during use, and exposure will be primarily to the oxide.

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There have not been any systematic and representative surveys of occupational exposure levels to antimony in industry; however, some data are available from walk-through surveys of selected companies conducted by NIOSH and other investigators. In some of these surveys, only a few samples were analyzed. In a facility where antimony oxide was produced from the sulfide ore, breathing-zone samples from five antimony oxide production workers ranged from 0.21 to 3.2 mg antimony/m^3; four of these samples were above 0.5 mg antimony/m^3. Air samples from the bagging area ranged from 0.43 to 0.83 mg antimony/m^3 (Cassady and Etchison 1976). In another facility that produced antimony and antimony oxide from ore, breathing-zone samples from 55 employees ranged from 0.05 to 6.21 mg antimony/m^3, and area air samples ranged from 0.14 to 2.12 mg antimony/m^3 (Donaldson 1976). The mean exposure for the antimony oxide operation was 2.23 mg/m^3, and this was the highest in the plant. Two personal air samples in a third antimony oxide production facility were 2.7 and 5.0 mg antimony/m^3, and general area samples ranged from 1.8 to 5.6 mg antimony/m^3 (Donaldson and Gentry 1975). In a secondary lead smelter where scrap batteries were reclaimed, breathing zone samples in 2 of 21 workers were quantifiable; these TWAs were 0.037 and 0.051 mg/m^3 (Craig et al. 1981). TWA antimony concentrations in the compounding area of a rubber company ranged from 0.01 to 0.15 mg/m^3, and the mean in an iron foundry was 0.00015 mg/m^3 (Salisbury 1980; Zhang et al. 1985). Antimony levels in a glass production facility were 0.005 mg/m^3, and this represents 1% of the NIOSH-recommended maximum level (Burroughs and Horan 1985). Antimony may also be released during injection molding of ignitionresistant polystyrene in which fire retardant additives that contain antimony are used. In one such study, antimony levels ranged from less than the detection limit of 0.0003 to 0.2 mg/m^3 (Willetts et al. 1982).

Since antimony trioxide is used in many materials as a fire retardant, it is likely that antimony will be released during fires. Antimony was present in soot and in tracheal specimens of people who perished in fires (Willetts et al. 1982). In 18 cases that were analyzed, soot antimony concentrations ranged from 0.1 to 543 ppm, and 50% of tracheal antimony concentrations exceeded the normal range of 0.1-124.0 ppm. These results indicate that firemen and other people at fires may be exposed to increased antimony levels in smoke.

Stibine may be produced in lead-acid battery plants during the formation process (Jones and Gamble 1984). In a study involving five battery plants, stibine concentrations ranged from not detectable to 2.5 mg/m^3. In three other surveys of battery plants, stibine concentrations ranged from not detectable to 0.35 mg/m^3 (Young 1979a), 0.007 mg/m^3 (Young 1979b), and 0.031 mg/m^3 (Young et al. 1979). Stibine was also reported in a company that manufactured glass for hypodermic syringes at levels up to 0.5 mg/m^3 (Burroughs and Horan 1985).

Antimony trioxide is used in the glass industry as a refining agent and colorant. In an exposure assessment in the German glass industry, TWA

antimony levels were as high as 0.351 mg/m^3 (Ludersdorf et al. 1987). Urine and blood antimony levels of exposed workers were enhanced. The median and maximum urine antimony levels in spot urine samples were 1.9 and 15.7 μg/L, respectively, compared with 0.4 and 0.7 μg/L for controls. Median and maximum blood levels for workers were 1.0 and 3.1 μg/L, respectively, versus 0.3 and 1.7 μg/L, respectively, for unexposed persons.

Nail samples from 71 Americans contained an average of 0.41 ppm of antimony. Averages for residents of four other countries ranged from 0.28 to 0.70 ppm (Takagi et al. 1988). In an analogous study, the mean concentration of antimony in hair samples from 55 men and women from Scranton, Pennsylvania, contained 0.096 ppm of antimony. The hair samples of populations from cities in four other countries contained mean antimony levels between 0.11 and 0.86 ppm (Takagi et al. 1986). These hair levels can also be compared to those in a Japanese national study in which the geometric mean concentration and standard deviation of antimony in washed hair samples from 234 healthy individuals were 0.078 and 2.5 ppm, respectively. No significant differences between different sexes or age groups were noted (Ohmori et al. 1981). In another Japanese study, hair and nail samples taken from workers at an antimony refinery, nearby residents, and a control group were analyzed before and after washing with a nonionic, surface-active agent in an ultrasonic cleaner (Katayama and Ishide 1987). The concentration of antimony in the nails of the three groups before and after washing was 730, 2.46, 0.19 ppm and 230, 0.63, and 0.09 ppm, respectively. The concentration of antimony in the hair of workers before and after washing was 222 and 196 ppm, compared with 0.21 and 0.15 ppm for controls. Exposure to antimony, therefore, greatly increases the antimony levels in nails and hair. The concentration in nails in exposed people is largely surficial.

A group of 21 workers from northern Sweden who were employed in nonferrous metal smelting and refining industries had median antimony concentrations‘ in their lungs of 0.30 ppm (wet weight). Controls from an unpolluted area had 0.029 ppm in their lungs (Hewitt 1988). Antimony concentrations in the lung tissues of eight British coal miners ranged from 0.19 to 0.59 ppm (dry weight); the levels in two controls were 0.47 and 0.62 ppm (Hewitt 1988).

5.6 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In discussing exposure to antimony, it is important to consider what form of antimony a person is exposed to and what is its availability. Such information is seldom available. Although high concentrations of antimony may be found in contaminated soil and sediment, the few studies that have been conducted indicate that much of the antimony may be embedded in a crystalline matrix or bound to hydrated iron, aluminum, and manganese oxides. In water, the pentavalent state is predominant, although significant levels of trivalent antimony and methylated antimony compounds exist. People who live or work near sources of antimony such as smelters, coal-fired power plants, and refuse

incinerators may be exposed to high levels of antimony in airborne dust, soil, and vegetation. People who live near or work at waste sites that receive slag from smelters or fly ash from power plants and refuse incinerators may also be exposed to higher than background levels. Exposure routes would include either inhalation of contaminated air or ingestion of contaminated soil or vegetation. Similarly, people who are exposed to soot and smoke in fires, such as firemen, may be exposed to high levels of antimony. Occupational exposure to antimony appears to be highest for those involved in the production and processing of antimony and antimony oxide. Workers in battery-forming areas of lead-storage battery plants may be exposed to high levels of stibine.

5.7 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA as amended directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of antimony is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of antimony.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.7.1 Data Needs

Physical and Chemical Properties. It is apparent from the physical and chemical properties of antimony and antimony trioxide shown in Table 3-2, that there are discrepancies in the literature values for the boiling points of antimony, antimony trichloride, and antimony trioxide (Freedman et al. 1978; Herbst et al. 1985; Weast 1988; Windholz 1983). This may be due to different levels of impurities in the samples tested. The fact that no numerical value exists for the water solubility of antimony trioxide, antimony pentoxide, and antimony pentasulfide is of no special significance. For inorganic salts, the solubility product coupled with stability constants for the ionic species in solution are the factors determining how much of the compound.goes into solution; the solubility in terms of the number of milligrams of the parent compound in solution, as used for organic compounds, is not meaningful. We do not know whether all the solubility products and stability constants for antimony and its compounds, required for determining the antimony species in natural water and their concentrations, are available. Other physical and

chemical properties in Table 3-2 for which there are no data are generally not well defined for antimony and its compounds or are not useful in determining their environmental fate.

Production, Import/Export, Use, and Disposal. Information on the production, import, and use of antimony and antimony trioxide is readily available (Carapella 1978; Llewellyn 1988; Plunkert 1982; U.S. Bureau of Mines 1989a). However, information on the production, import, and use patterns of other antimony compounds is not available, and is needed to assess human exposure to these compounds. Except for the recycling of batteries, little information is available concerning the disposal of antimony and its compounds.

Much of the antimony released to the environment is transferred to offsite locations for disposal (probably landfills) (TRI 1989). Most of the waste products from mining and smelting operations are discarded on land in large tailing piles; many of these are now abandoned (TRI 1989). Acid conditions are often created in these tailing piles by the oxidation of pyrites contained in the tailings that increase the potential for leaching (DeLaune and Smith 1985). Information concerning antimony leaching from slag heaps is important in assessing antimony releases to the environment. More detailed information regarding the form of antimony that is disposed of and the disposal methods is necessary to assess the potential exposure to these compounds.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxic Release Inventory (TRI), which contains this information for 1987, became available in May of 1989 (TRI 1989). This database will be updated yearly and should provide a list of industrial production facilities and emissions. Releases according to this database are shown in Table 5-l.

Environmental Fate. In assessing human exposure, the form (valence state, compound, adsorption, coprecipitation, particle size) of antimony and its availability must be considered. This information is apt to be sitespecific. Data concerning the forms of antimony in air, soil, water, and sediment are limited. Information regarding the transformations that may occur, the rates of transformation, and the conditions that facilitate the transformations is also lacking. For example, we do not know whether antimony is methylated in soil as is arsenic and as antimony itself may be methylated in the aquatic environment (Andreae et al. 1983; Austin and Millward 1988). Information relating to the adsorption of antimony and its compounds by soil and sediment is limited. This information should cover a range of soil types, soil components (e.g., clay), and conditions.

5. POTENTIAL FOR HUMAN EXPOSURE

Bioavailability from Environmental Media. Antimony is poorly absorbed following inhalation and oral exposure (Felicetti et al. 1979a, 1979b; Gerber et al. 1982; Thomas et al. 1973). Dermal exposure to high levels of antimony trioxide resulted in death in rabbits (Myers et al. 1978). The application area was occluded, suggesting that at least some forms of antimony can be absorbed through the skin. Although there is no information on the absorption efficiency of antimony from environmental media in humans, there is evidence in animals that it is absorbed. The vegetation and soils at sites near antimony smelters are heavily contaminated with antimony. Elevated levels of antimony in various tissues were observed in animals living near the smelter (Ainsworth 1988). An animal study designed to measure the rate of absorption of antimony from environmental media would be useful in assessing the toxicological significance of levels of antimony in the air and soil near hazardous waste sites.

Food Chain Bioaccumulation. Extensive studies at a smelter site indicate that the uptake of antimony from soil in grass and subsequent translocation in shoots is slight (Ainsworth 1988). At a polluted site, most of the antimony on plants resulted from atmospheric deposition. These studies additionally showed that there was no bioaccumulation of antimony in small mammals compared with their food. Other studies on fish and aquatic organisms indicate that the bioconcentration of antimony is low (Callahan et al. 1978; EPA 1980; Maher 1986). Accordingly, there is little indication that antimony would bioconcentrate in the food chain and in humans. It should be pointed out that data on the biconcentration of antimony in fish and biomagnification in higher trophic levels of animals is limited. Monitoring data on the levels of antimony in plants and animals is minimal. A larger database of information covering more sites and species is desirable. This would establish whether antimony might accumulate in some species or in the presence of some forms of antimony.

Exposure Levels in Environmental Media. Although some data on the levels of antimony in ambient air are available, these data are not representative and recent enough to estimate the current exposure levels to antimony by the U.S. population via inhalation (Austin and Millward 1988; Hopper and Barrie 1988; Johnson et al. 1984; Kowalczyk et al. 1982; Schroeder et al. 1987; Wiersema et al. 1984). While the levels of antimony in water are generally very low (Eckel and Jacob 1989), the data for ambient water are marginally adequate; data for drinking water and groundwater are virtually nonexistent. Similarly data regarding the levels of antimony in the various food classes and diet are fragmentary (Cunningham 1987; Syengar et al. 1987; Tanner and Freedman 1977). Reliable and recent monitoring data for antimony in air, water, and foods are essential for estimating the extent of exposure from each of these sources. While the levels of antimony in surface and groundwater at hazardous waste sites are elevated above ambient levels (CLPSD 1989), the elevation is not very great. Since it is not clear whether the levels reported at waste sites are for dissolved antimony as are the ambient

levels, the difference between antimony concentrations at waste sites and ambient sites may be lower. Antimony concentrations in soil at some hazardous waste sites are high (CLPSD 1989; Eckel and Langley 1988; View 1989), and there is a potential of exposure from ingesting soil at these sites. The leaching potential of these soils appears to be low (Ainsworth 1988; Foster 1989; King 1988; Trnovsky et al. 1988). The exposure potential from antimony at these sites from antimony reentrained by wind also appears to be low (Ainsworth 1988).

Exposure Levels in Humans. The levels of antimony in the hair, nails, and breast milk of a sample of the U.S. population are known (Iyengar et al. 1982; Takagi et al. 1986, 1988). While the tissue levels of antimony in Japanese people are available (Sumino et al. 1975), analogous levels for Americans were not found. In particular, no reliable data regarding the levels of this element in the blood and urine of unexposed U.S. residents are available. Such data may be helpful in establishing the background exposure levels of antimony. Levels of antimony in hair, nails, lung, blood, and urine of some exposed workers are available, but the amount of data is small (Hewitt 1988; Katyama and Ishidi 1987; Ludersdorf et al. 1987). None of these data refer to populations living around the hazardous waste sites containing elevated levels of antimony. Such data may be significant in assessing the exposure levels of this component of the population.

Exposure Registries. No exposure registries for antimony and its compounds were located. Antimony and its compounds do not currently have a subregistry established in the National Exposure Registry. They will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to the exposure to the compound.

5.7.2 On-going Studies

Remedial investigations and feasibility studies conducted at the 52 NPL sites known to be contaminated with antimony will add to the available database on exposure levels in environmental media, exposure levels in humans, and exposure registries, and will increase the current knowledge regarding the transport and transformation of antimony in the environment. NIOSH is updating its estimates of occupational exposure by including exposure to antimony and its compounds in trade name chemicals (NIOSH 1989). No other ongoing research studies pertaining to the environmental fate of antimony or to occupational or general population exposures to antimony were identified.

6. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting and/or measuring and monitoring antimony in environmental media and in biological samples. The intent is not to provide an exhaustive list of analytical methods that could be used to detect and quantify antimony. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used to detect antimony in environmental samples are the methods approved by federal agencies such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association ww - Additionally, analytical methods are included that refine previously used methods to obtain lower detection limits, and/or to improve accuracy and precision.

6.1 BIOLOGICAL MATERIALS

Methods for the analytical determination of antimony in biological materials are basically the same as those used for the environmental samples that are discussed below. The most commonly used methods determine the total antimony content of the sample, not the particular antimony compound or oxidation state that is present. Methodological differences are a function of the level of antimony in the sample, digestion procedures required to solubilize the sample, and the level of potentially interfering substances in the type of sample. Antimony occurs at very low levels in biological samples. The accurate determination of trace levels of antimony in these samples may require special methods (e.g., neutron activation) that are both sensitive and selective. Atomic absorption spectroscopy and inductively coupled plasmaatomic emission spectroscopy, with or without preconcentration or separation steps, are the most commonly employed methods. Atomic absorption has three variants: direct aspiration into a flame, atomization in an electrically heated carbon rod, or generation of stibine that is then passed into a heated silica tube.

Instrumental neutron activation analysis (INAA), with or without chemical separation, has very good sensitivity and selectivity for antimony, and it has the advantage of being able to measure many elements simultaneously. However, it is slow, costly, and requires special facilities. INAA is favored for surveys where trace levels of many elements are to be determined. It is often required for measuring antimony in tissues in which the antimony level is very low. The neutron activation analysis of antimony requires an exposure to neutron fluxes for 6 hours to 2 days. After the exposure period, the samples are kept for several days before counting, This allows the activity of short half-lived isotopes to decline, and thus improves accuracy of the analysis (Iyengar et al. 1978). Nondestructive INCA can be used to measure concentrations to levels somewhat below 1 ppm. Nondestructive methods are not only advantageous because of reduced sample handling, but also

because they are independent of the sample matrix and of the efficiency of the digestion or extraction procedure. While this is generally adequate for antimony determinations in hair and lung tissues, the antimony levels in blood serum and kidney tissues are usually too low to measure without preconcentration (Iyengar et al. 1978). Detection limits may be limited by interferences from matrix elements such as sodium, potassium, phosphorus, and bromine. Lower detection limits (approximately 0.006 ppm) can be obtained by digestion and solvent extraction to eliminate these interferences (Mok and Wai 1988).

Analytical methods and detection limits for antimony in biological materials are given in Table 6-1. Antimony contained in other biological materials such as hair and nails can be determined by using the same analytical techniques as for blood and tissue, but suitable procedures for dissolving the sample matrix must be used (Takagi et al. 1986, 1988).

6.2 ENVIRONMENTAL SAMPLES

Analytical methods for antimony in environmental samples generally determine the total antimony content of the sample; determining specific antimony compounds is difficult. Some methods can be used to determine antimony in different oxidation states, but these methods are only used in special circumstances.

The most common methods used for environmental samples are atomic absorption spectrometry (AAS) (either flame or graphite furnace) and inductively coupled plasma-atomic emission spectroscopy (ICP-AES). Before the widespread use of AAS, calorimetric methods were used for the determination of antimony; the best known of these methods is the rhodamine B method (APHA 1972). The basis for the method is the formation of a pink complex when pentavalent antimony reacts with rhodamine B in the presence of an excess of chloride ions. The complex is extracted into an organic solvent and the absorbance measured at 565 nm. Trivalent antimony must be oxidized to the pentavalent state with nitric, sulfuric, and perchloric acids.

Water and waste water samples can be analyzed for antimony by EPA Test Methods 220.1 (atomic absorption, direct aspiration), 220.2 (atomic absorption, furnace technique), or 200.7 (inductively coupled plasma-atomic emission spectroscopy) (EPA 1983b). These methods are suitable for groundwater, surface water, and domestic and industrial effluents. In open ocean water and in other water samples with a low antimony concentration, a preconcentration and/or separation procedure involving coprecipitation, chelation, selective adsorption, or hydride formation is required before analysis (Andresen and Salbu 1982; Apte and Howard 1986; Maher 1986; Sturgeon et al. 1985). The atomic absorption wavelength used for antimony is 217.6 nm. In the presence of lead concentrations of the order of 1 g/L, however, a spectral interference may occur at this resonance line, and the line at 231.1 nm should be used instead. When using direct aspiration, the spectral

TABLE 6-1. Analytical Methods for Determining Antimony in Biological Materials

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Blood, tissue, or hair[a]	Acid digestion	Method 8005, ICP-AES	No data	106% at 10 μg antimony	NIOSH 1985
Urine	Treat with EDTA and refrigerate; sample stable for 1 week; wet ash	Hydride generation-AAS	10 μg/L	No data	Anonymous 1977
Feces	Digest with concentration HCl/HNO_3, extract with hexane/hydrogen peroxide, nickel matrix modifier	Graphite furnace AAS	No data	96.9%, mean	Bio/dynamics 1990

[a]Method extended to hair (Takagi et al. 1986)

AAS = atomic absorption spectrometry
EDTA = ethylenediaminetetraacetic acid
ICP-AES = inductively coupled plasma-atomic emission spectroscopy

absorption of antimony is reduced when the concentration of acid increases. Therefore, it is important to match the concentration of acid in standards and samples (EPA 1983b). Background correction of nonspecific absorption is advisable for some samples, such as those containing sulfuric acid (Ainsworth 1988). Analytical methods and detection limits for antimony in environmental media are given in Table 6-2. If the determination of dissolved antimony is required, samples should be filtered using a 0.45 μm membrane filter.

Acid digestion to assure release of antimony from the sample matrix is a crucial step in the analysis of environmental samples. Unless the particular type of sample has been well studied, it is usually important to experiment with different digestion procedures. For the release of antimony from soil, hydrogen fluoride mixed with perchloric acid or another strong acid is generally required. Aqua regia, however, has been found to be suitable. For plant and animal tissue, a combination of sulfuric and nitric acids is most satisfactory (Ainsworth 1988).

Antimony forms a volatile hydride under reducing conditions, and hydride generation has been interfaced with different analytical procedures for enhanced sensitivity and selectivity. The most popular reagent used for this reduction is sodium borohydride (Andreae 1983). It is necessary to add KI to the reaction medium to completely reduce Sb(II1). In atomic absorption, increased sensitivity can be achieved by using hydride generation because the efficiency of atomization is greater for stibine than for antimony solutions introduced into the flame. Another advantage of hydride generation is that separati.on is achieved from nonhydride-forming elements, thereby eliminating interferences. Antimony reduction is pH dependent, possibly because neutral and cationic species (but not anionic ones) are subject to reduction by negatively charged borohydride ions. By exploiting the pH dependence of the reduction, it is possible to separately determine Sb(III) and Sb(V) in natural waters (Andreae 1983; Apte and Howard 1986). Other methods that distinguish Sb(II1) from Sb(V) rely on selective extraction techniques in which Sb(II1) is extracted into an organic solvent and analyzed. After analysis, Sb(V) is reduced and extracted (Abbasi 1989; Mok and Wai 1987). When very high sensitivity is required, such as that necessary for the analysis of antimony in food, neutron activation analysis is often employed. X-ray fluorescence (XRF) and anodic stripping voltammetry (ASV) are other analytical methods that are frequently used (Costantini et al. 1985; Gillain and Brihaye 1985; Ives et al. 1984; Johnson et al. 1984).

In the determination of trace metals, major concerns include contamination and loss. Contamination can be introduced from impurities in reagents and containers and from laboratory dust. Losses may also occur due to adsorption of the analyte onto container walls. In the case of antimony, a common source of loss is volatilization during acid digestion or ashing in an AAS furnace.

TABLE 6-2. Analytical Methods for Determining Antimony in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air	Filter collection and acid digestion	Method 301, colorimetric (Rhodamine B)	1.0 μg	95%-102% between 2 and 10 μg of antimony	APHA 1972
	Filter collection and acid digestion and reduction of Sb(V) with NaI	Hydride generation-AAS	4 ng	10% accuracy at 40 ng antimony	DeDoncker et al. 1983
Air (stilbine)	Filter collection (20 L sample) on $HgCl_2$-coated silica gel; desorption and treatment with concentrated HCl, ceric sulfate, and liquid extraction	NIOSH 6008, colorimetric (Rhodamine B)	No data	98.6% between 0.12 and 1.0 μg antimony	NIOSH 1987
Water, waste water	Acid digestion	Method 204.1, AAS/direct aspiration	0.2 mg/L	96% and 97% at 5 and 15 mg antimony/L	EPA 1983a
	Acid digestion, sample solutions should contain 2% HNO_3	Method 204.2, AAS/furnace technique	3 μg/L	Not applicable	EPA 1983a
	Filter and acidify sample	Method 200.7, ICP-AES	32 μg/L	Not applicable	EPA 1983a
Soil, sediment sludge, solid waste	Digestion with 4:1 HNO_3 and HCl[a]	Method 3050 (modified)[a] ICP-AES	No data	3% accuracy at 33 ppm antimony	EPA 1986
Food	Acid digestion and resin separation following irradiation	INAA	0.1-0.3 ppb	No data	Cunningham 1987

[a]The digestion procedure in Method 3050 is not suitable for antimony. A satisfactory digestion procedure has been proposed by Kimbrough and Wakakuwa (1989).

AAS = atomic absorption spectrometry; HCl = hydrochloric acid; $HgCl_2$ = mercuric chloride; HNO_3 = nitric acid; ICP-AES = inductively coupled plasma-atomic emission spectroscopy; INAA = instrumental neutron activation analysis; NaI = sodium iodide; NIOSH = National Institute for Occupational Safety and Health; Sb(V) = antimony (+5)

6. ANALYTICAL METHODS

6.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA as amended directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of antimony is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of antimony.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.3.1 Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Methods for determining antimony in biological materials are well developed, and there are methods available to most laboratories that are satisfactory for testing biological samples that naturally contain high concentrations of antimony or for occupational exposure testing (Anonymous 1977; NIOSH 1985). Since antimony occurs at very low levels in many biological materials, methods such as INAA that require special facilities must often be used to achieve adequate sensitivity (Iyengar et al. 1978). Standardized methods are available from NIOSH and other sources to measure antimony in blood, urine, and tissue (NIOSH 1985). Several authors have reported that antimony concentrations in hair, nails, blood, or urine are elevated in exposed individuals; therefore, antimony levels in these samples can be used as a biomarker for exposure to antimony (Katayama and Ishidi 1987). Available analytical methods are capable of determining the levels of antimony in these media in both normal and occupationally exposed persons (Bakagi et al. 1986, 1988; Iyenger et al. 1988). Methods with sufficient sensitivity (e.g., INAA), however, are not available in most laboratories.

No biomarkers that could be used to characterize effects of antimony have been identified, Should subtle biochemical or physiological changes unique to antimony be identified, methods to analyze for these changes could possibly be developed if they don't already exist.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Methods for determining antimony in environmental media are well developed and adequate. Standardized methods are available from EPA, NIOSH, and other sources (APHA 1972; Cunningham 1987; DeDonker et al. 1983;

EPA 1983a, 1986; NIOSH 1987). Since most analytical methods measure total antimony, the methods for analyzing for the parent compound and degradation product are identical.

6.3.2 On-going Studies

Analytical methods for antimony and antimony compounds are currently being developed at EPA's Environmental Monitoring Systems Laboratory in Cincinnati, Ohio (EPA 1989b). No on-going studies regarding new analytical methods for measuring antimony in biological materials were located In the available literature.

7. REGULATIONS AND ADVISORIES

National and international guidelines and state regulations pertinent to human exposure to antimony and compounds are summarized in Table 7-1.

Antimony compounds are regulated by the Clean Water Effluent Guidelines for the following industrial point sources: nonferrous metal manufacturing, steam electric, asbestos, timber products processing, mineral mining, paving and roofing, paint formulating, ink formulating, gum and wood, carbon black, and nonferrous metal forming (EPA 1988). Antimony compounds are also regulated by the Clean Air Act.

7. REGULATIONS AND ADVISORIES

TABLE 7-1. Regulations and Guidelines Applicable to Antimony and Compounds

Agency	Description	Information	References
NATIONAL			
Regulations:			
a. Air:			
OSHA	PEL TWA	0.5 mg antimony/m^3	OSHA 1989 (29 CFR 1910.1000)
b. Other:			
EPA OERR	Reportable quantity		
	Antimony pentafluoride	1 pound	EPA 1987 (40 CFR 300)
	Antimony pentachloride	1,000 pounds	EPA 1987
	Antimony potassium tartrate	100 pounds	(40 CFR 117.3)
	Antimony tribromide	1,000 pounds	
	Antimony trichloride	1,000 pounds	
	Antimony trifluoride	1,000 pounds	
	Antimony trioxide	1,000 pounds	
	Antimony	5,000 pounds	EPA 1986 (40 CFR 302.4)
Guidelines:			
a. Air:	TLV TWA		ACGIH 1989
	Antimony and compounds	0.5 mg antimony/m^3	
	Stibine	0.51 mg/m^3	
	REL TWA		NIOSH 1990
	Antimony and compounds	0.5 mg antimony/m^3	
	Stibine	0.5 mg/m^3	
b. Water:	Ambient Water Quality Criterion	145 µg/L	EPA 1980
c: Other:	RfD (oral)	0.0004 mg antimony/kg/day	EPA 1989
STATE			
Regulations and Guidelines:			NATICH 1988
a. Air:	Acceptable ambient air concentrations		
Antimony:			
Connecticut		10 µg/m^3 (8-hr avg)	
Florida (Tampa)		0.005 mg/m^3 (8-hr avg)	
Kansas		1.19 µg/m^3 (annual avg)	
Kansas (Kansas City)		1.19 µg/m^3 (1-yr avg)	
Nevada		0.012 mg/m^3 (8-hr avg)	
New York		0.67 µg/m^3 (1-yr avg)	
North Dakota		0.005 mg/m^3 (8-hr avg)	
Pennsylvania (Philadelphia)		1.2 µg/m^3 (1-yr avg)	
Rhode Island		40 µg/m^3 (annual avg)	
Virginia		8 µg/m^3 (24-hr avg)	
Antimony oxide:			
Connecticut		5.0 µg/m^3 (8-hr avg)	
Florida (Tampa)		0.005 mg/m^3 (8-hr avg)	
Nevada		0.012 µg/m^3 (8-hr avg)	
New York		0.67 mg/m^3 (1-yr avg)	
North Dakota		0.005 mg/m^3 (8-hr avg)	
Virginia		8.0 µg/m^3 (24-hr avg)	

7. REGULATIONS AND ADVISORIES

TABLE 7-1 (Continued)

Agency	Description	Information	References
STATE (Continued)			
b. Water: Antimony Kansas	Drinking water quality standards	143 µg/L	

avg = average; EPA = Environmental Protection Agency; OERR = Office of Emergency and Remedial Response; OSHA = Occupational Safety and Health Admistration; PEL = permissible exposure level; REL = recommended exposure level; RfD = reference dose; TLV = threshold limit value; TWA = time-weighted average

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9. GLOSSARY

Acute Exposure -- Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Adsorption Coefficient (K_{oc}) -- The ratio of the amount of a chemical adsorbed per unit weight of organic carbon in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (K_d) -- The amount of a chemical adsorbed by a sediment or soil (i.e., the solid phase) divided by the amount of chemical in the solution phase, which is in equilibrium with the solid phase, at a fixed solid/solution ratio. It is generally expressed in micrograms of chemical sorbed per gram of soil or sediment.

Bioconcentration Factor (BCF) -- The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Cancer Effect Level (CEL) -- The lowest dose of chemical in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Carcinogen -- A chemical capable of inducing cancer.

Ceiling Value -- A concentration of a substance that should not be exceeded, even instantaneously.

Chronic Exposure -- Exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Developmental Toxicity -- The occurrence of adverse effects on the developing organism that may result from exposure to a chemical prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Embryotoxicity and Fetotoxicity -- Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and in utero death.

EPA Health Advisory -- An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

9. GLOSSARY

Immediately Dangerous to Life or Health (IDLH) -- The maximum environmental concentration of a contaminant from which one could escape within 30 min without any escape-impairing symptoms or irreversible health effects.

Intermediate Exposure -- Exposure to a chemical for a duration of 15-364 days as specified in the Toxicological Profiles.

Immunologic Toxicity -- The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

In Vitro -- Isolated from the living organism and artificially maintained, as in a test tube.

In Vivo -- Occurring within the living organism.

Lethal Concentration $_{(Lo)}$ (LC_{Lo}) -- The lowest concentration of a chemical in air which has been reported to have caused death in humans or animals.

Lethal Concentration $_{(50)}$ (LC_{50}) -- A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

Lethal Dose $_{(Lo)}$ (LD_{Lo}) -- The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals.

Lethal Dose $_{(50)}$ (LD_{50}) -- The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time $_{(50)}$ (LT_{50}) -- A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Lowest-Observed-Adverse-Effect Level (LOAEL) -- The lowest dose of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Malformations -- Permanent structural changes that may adversely affect survival, development, or function.

Minimal Risk Level -- An estimate of daily human exposure to a chemical that is likely to be without an appreciable risk of deleterious effects (noncancerous) over a specified duration of exposure.

9. GLOSSARY

Mutagen -- A substance that causes mutations. A mutation is a change in the genetic material in a body cell. Mutations can lead to birth defects, miscarriages, or cancer.

Neurotoxicity -- The occurrence of adverse effects on the nervous system following exposure to chemical.

No-Observed-Adverse-Effect Level (NOAEL) -- The dose of chemical at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Octanol-Water Partition Coefficient (K_{ow}) -- The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Permissible Exposure Limit (PEL) -- An allowable exposure level in workplace air averaged over an 8-hour shift.

q1* -- The upper-bound estimate of the low-dose slope of the dose-response curve as determined by the multistage procedure. me ql* can be used to calculate an estimate of carcinogenic potency, the incremental excess cancer risk per unit of exposure (usually μg/L for water, mg/kg/day for food, and $\mu g/m^3$ for air).

Reference Dose (RfD) -- An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to nonthreshold effects such as cancer.

Reportable Quantity (RQ) -- The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 lb or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity -- The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

9. GLOSSARY

Short-Term Exposure Limit (STEL) -- The maximum concentration to which workers can be exposed for up to 15 min continually. No more than four excursions are allowed per day, and there must be at least 60 min between exposure periods. The daily TLV-TWA may not be exceeded.

Target Organ Toxicity -- This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Teratogen -- A chemical that causes structural defects that affect the development of an organism.

Threshold Limit Value (TLV) -- A concentration of a substance to which most workers can be exposed without adverse effect. The TLV may be expressed as a TWA, as a STEL, or as a CL.

Time-Weighted Average (TWA) -- An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Toxic Dose (TD_{50}) -- A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Uncertainty Factor (UF) -- A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LCAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

APPENDIX A

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in nontechnical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or substance release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the substance. The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Tables and Figures for Levels of Significant Exposure (LSE)

Tables (2-1, 2-2, and 2-3) and figures (2-l and 2-2) are used to summarize health effects by duration of exposure and endpoint and to illustrate graphically levels of exposure associated with those effects. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse-Effect Levels (NOAELs), Lowest-Observed-Adverse-Effect Levels (NOAELs) for Less Serious and Serious health effects, or Cancer Effect Levels (CELs). In addition, these tables and figures illustrate differences in response by species, Minimal Risk Levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upper-bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. The LSE tables and figures can be used for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text.

The legends presented below demonstrate the application of these tables and figures. A representative example of LSE Table 2-l and Figure 2-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See LSE Table 2-l

(1). Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exist,

three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 2-1, 2-2, and 2-3, respectively). LSE figures are limited to the inhalation (LSE Figure 2-l) and oral (LSE Figure 2-2) routes.

(2). Exposure Duration Three exposure periods: acute (14 days or less); intermediate (15 to 364 days); and chronic (365 days or more) are presented within each route of exposure. In this example, an inhalation study of intermediate duration exposure is reported.

(3). Health Effect The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table.

(4). Key to Figure Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to define a NOAEL and a Less Serious LOAEL (also see the two "18r" data points in Figure 2-l).

(5). Species The test species, whether animal or human, are identified in this column.

(6). Exposure Frequency/Duration The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to [substance x] via inhalation for 13 weeks, 5 days per week, for 6 hours per day.

(7). System This column further defines the systemic effects. These systems include: respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, one systemic effect (respiratory) was investigated in this study.

(8). NOAEL A No-Observed-Adverse-Effect Level (NOAEL) is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system which was used to derive an intermediate exposure, inhalation MR.L of 0.005 ppm (see footnote "cm).

(9). LOAEL A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest exposure level used in the study that caused a harmful health effect. LOAELS have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to

quantify the adverse effect accompanies the LOAEL. The "Less Serious" respiratory effect reported in key number 18 (hyperplasia) occurred at a LOAEL of 10 ppm.

(10). Reference The complete reference citation is given in Chapter 8 of the profile.

(11). CEL A Cancer Effect Level (CEL) is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiological studies. GELS are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses which did not cause a measurable increase in cancer.

(12). Footnotes Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "c" indicates the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 @pm.

LEGEND

See LSE Figure 2-1

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure levels for particular exposure duration.

(13). Exposure Duration The same exposure periods appear as in the LSE table. In this example, health effects observed within the intermediate and chronic exposure periods are illustrated.

(14). Health Effect These are the categories of health effects for which reliable quantitative data exist. The same health effects appear in the LSE table.

(15). Levels of Exposure Exposure levels for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure levels are reported on the log scale "y" axis. Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16). NOAEL In this example, 18r NOAEL is the critical end point for which an intermediate inhalation exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates a NOAEL for the test species (rat). The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17). CEL Key number 38r is one of three studies for which Cancer Effect Levels (CELs) were derived. The diamond symbol refers to a CEL for the test species (rat). The number 38 corresponds to the entry in the LSE table.

APPENDIX A

(18). Estimated Upper-Bound Human Cancer Risk Levels This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

(19). Key to LSE Figure The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 → TABLE 2-1. Levels of Significant Exposure to [Chemical x] - Inhalation

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 → INTERMEDIATE EXPOSURE							
3 → Systemic	5	6	7	8	9		10
4 → 18	Rat	13 wk 5d/wk 6hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
CHRONIC EXPOSURE							
Cancer					11		
38	Rat	18 mo 5d/wk 7hr/d			20 (CEL, multiple organs)		Wong et al. 1982
39	Rat	89-104 wk 5d/wk 6hr/d			10 (CEL, lung tumors, nasal tumors)		NTP 1982
40	Mouse	79-103 wk 5d/wk 6hr/d			10 (CEL, lung tumors, hemangiosarcomas)		NTP 1982

[a] The number corresponds to entries in Figure 2-1.

12 → [b] Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5×10^{-3} ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

CEL = cancer effect level; d = day(s); hr = hour(s); LOAEL = lowest-observed-adverse-effect level; mo = month(s); NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week(s)

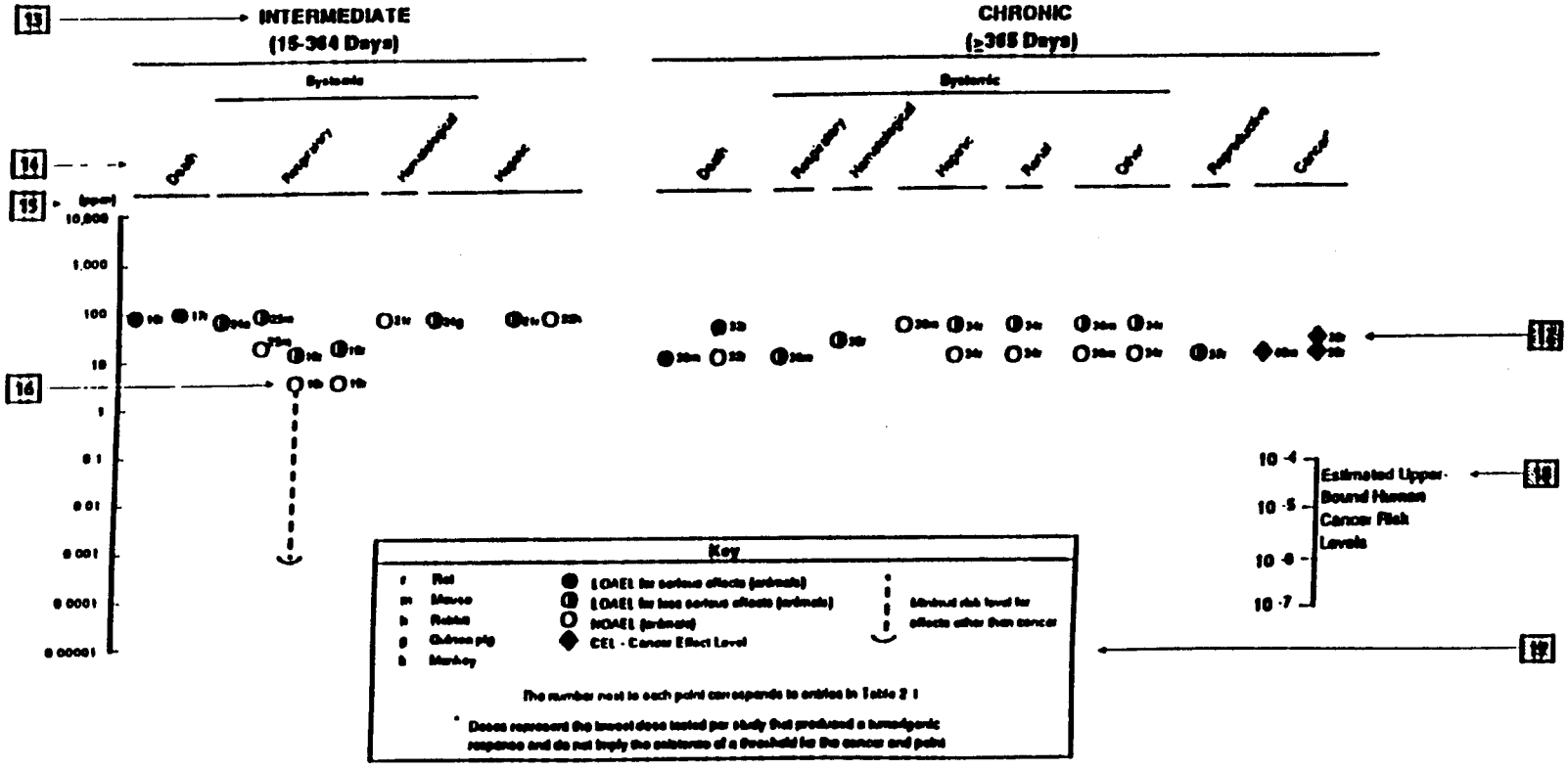

FIGURE 2-1. Levels of Significant Exposure to [Chemical X]-Inhalation

APPENDIX A

Chapter 2 (Section 2.4)

Relevance to Public Health

The Relevance to Public Health section provides a health effects summary based on evaluations of existing toxicological, epidemiological, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?

2. What effects observed in animals are likely to be of concern to humans?

3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The section discusses health effects by end point. Human data are presented first, then animal data. Both are organized by route of exposure (inhalation, oral, and dermal) and by duration (acute, intermediate, and chronic). In vitro data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this section. If data are located in the scientific literature, a table of genotoxicity information is included.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existingtoxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. MRLs for noncancer end points if derived, and the end points from which they were derived are indicated and discussed in the appropriate section(s).

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Identification of Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information was available, MRLs were derived. MRLs are specific for route (inhalation or oral) and duration (acute, intermediate, or chronic) of exposure. Ideally, MRLs can be derived from all six exposure scenarios (e.g., Inhalation - acute, -intermediate, -chronic; Oral - acute, -intermediate, - chronic). These MRLs are not meant to support regulatory action,but to aquainthealth professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a substance emission, given the concentration of a contaminant in air or the estimated daily dose received via food or water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

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MRL users should be familiar with the toxicological information on which the number is based. Section 2.4, "Relevance to Public Health," contains basic information known about the substance. Other sections such as 2.6, "Interactions with Other Chemicals" and 2.7, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology used by the Environmental Protection Agency (EPA) (Barnes and Dourson, 1988; EPA 1989a) to derive reference doses (RfDs) for lifetime exposure.

To derive an MRL, ATSDR generally selects the end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential effects (e.g., systemic, neurological, and developmental). In order to compare NOAELs and LOAELs for specific end points, all inhalation exposure levels are adjusted for 24hr exposures and all intermittent exposures for inhalation and oral routes of intermediate and chronic duration are adjusted for continuos exposure (i.e., 7 days/week). If the information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. The NOAEL is the most suitable end point for deriving an MRL. When a NOAEL is not available, a Less Serious LOAEL can be used to derive an MRL, and an uncertainty factor (UF) of 10 is employed. MRLs are not derived from Serious LOAELs. Additional uncertainty factors of 10 each are used for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the adjusted inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables.

APPENDIX B

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADME	Absorption, Distribution, Metabolism, and Excretion
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BSC	Board of Scientific Counselors
CDC	Centers for Disease Control
CEL	Cancer Effect Level
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
CLP	Contract Laboratory Program
cm	centimeter
CNS	central nervous system
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DGL	Department of Labor
ECG	electrocardiogram
EEG	electroencephalogram
EPA	Environmental Protection Agency
EKG	see ECG
FAO	Food and Agricultural Organization of the United Nations
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
f_1	first generation
fpm	feet per minute
ft	foot
FR	Federal Register
G	gram
GC	chromatography
Ha	hectare
HPLC	high performance liquid chromatography
hr	hour
IDLH	Immediately Dangerous to Life and Health
IARC	International Agency for Research on Cancer
ILO	International Labor Organization
in	inch
Kd	distribution ratio (soil/water)
Kg	kilogram
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{Lo}	lethal concentration low
LC_{50}	lethal concentration 50 percent kill
LD_{Lo}	lethal dose low

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LD_{50}	lethal dose 50 percent kill
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
m	meter
M	molar
mg	milligram
min	minute
mL	milliliter
mm	millimeters
mmo1	millimole
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
nm	nanometer
nM	nanomolar
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPL	National Priorities List
NRC	National Research Council
NTIS	National Technical Information Service
NTP	National Toxicology Program
OSHA	Occupational Safety and Health Administration
PEL	permissible exposure limit
pg	picogram
pmo1	picogram
PHS	Public Health Service
PMR	proportional mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
REL	recommended exposure limit
RfD	Reference Dose
RTECS	Registry of Toxic Effects of Chemical Substances
sec	seconds
SCE	sister chromatid exchange
SIC	Standard Industrial Classification
SMR	standard mortality ratio
STEL	short-term exposure limit
STORET	STORAGE and RETRIEVAL
TLV	threshold limit value
TSCA	Toxic Substance Control Act

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TRI	Toxics Release Inventory
TWA	time-weighted average
U.S.	United States
UF	uncertainty factor
WHO	World Health Organization
>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	less than or equal to
5	percent
α	alpha
β	beta
δ	delta
γ	gamma
μm	micron
μg	microgram

APPENDIX C

PEER REVIEW

A peer review panel was assembled for antimony. The panel consisted of the following members: Dr. William Buck, University of Illinois, Urbana, Illinois; Dr. George Cherian, University of Western Ontario, London, Ontario, Canada; Dr. Ernest Foulkes, University of Cincinnati, Cincinnati, Ohio; Dr. Derek Hodgson, University of Wyoming, Laramie, Wyoming; and Dr. Maryce Jacobs, American Institute for Cancer Research, Washington, DC. These experts collectively have knowledge of antimony's physical and chemical properties ,toxicokinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in the Comprehensive Environmental Response, Compensation, and Liability Act of 1986, Section 104.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers comments and determined which comments will be included in the profile. A listing of the peer reviewers comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.