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ECOLOGICAL AND BIOMEDICAL EFFECTS OF EFFLUENTS FROM NEAR-TERM ELECTRIC VEHICLE STORAGE BATTERY CYCLES

MASTER

ARGONNE NATIONAL LABORATORY

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by the
Division of Environmental Impact Studies
and the
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SUMMARY AND CONCLUSIONS

1. The ecological and biomedical effects due to commercialization of electric storage batteries as related to electric and hybrid vehicle (EV and EHV) applications were assessed. This assessment deals only with the near-term batteries, namely Pb/Acid, Ni/Zn, and Ni/Fe.

2. Storage battery technology has been considered in its totality in this assessment. The complete battery cycle comprises (1) mining and milling of the necessary raw materials; (2) manufacture of the batteries and their cases and covers; (3) use of the batteries in electric vehicles, including the charge-discharge cycles; (4) recycling of spent batteries; and (5) disposal of non-recyclable components.

3. The market penetration was assumed to grow at a rate of 25% for Pb/Acid battery and 30% for Ni/Zn or Ni/Fe batteries per year and to reach a total of three million Pb/Acid and eight million Ni/Zn or Ni/Fe powered electric vehicles on the road by the year 2000. Assessments were made both in terms of each megawatt-hour of installed capacity (assuming a one megawatt-hour requirement for each 40 electric vehicles), and for the total number of EVs expected for each type of battery.

4. An effort was made to identify the gaseous, liquid, and solid emissions from various phases of the battery cycle. The effluents dispersal in the environment was modeled and ecological effects were assessed in terms of biogeochemical cycles. The metabolic and toxic responses by humans and laboratory animals to constituents of the effluents were discussed. Pertinent environmental and health regulations related to the battery industry were summarized and regulatory implications for large-scale storage battery commercialization were discussed.

5. For the assumed scenario of three million Pb/Acid battery-powered electric vehicles on the road by the year 2000:

   a. The following impacts on the lead-acid battery industry are projected -

      - a 66% increase in lead mining and milling will be required (Table 2.7);
      - a 55% increase in primary lead smelting and refining will be required (Table 2.11);
      - a 94% increase in battery manufacturing facilities will be required (Table 2.20);
      - a 78% increase in battery breaking operations will be required (Table 2.27);
      - a 44% increase in secondary lead smelting and refining will be required (Table 2.28).

b. The following airborne emissions are projected -

- 3.3 metric tons (MT) per day lead in fugitive dust from mining and milling (Table 2.7);
- 1.3 MT/day lead in fugitive dust and controlled blast furnace emissions from primary smelting and refining (Table 2.11);
- 0.1 MT/day of lead in fumes and particles from battery manufacturing (Table 2.20);
- 4.7 MT/day of sulfur dioxide and 3.8 MT/day of particulates containing 0.9 MT of lead from secondary smelting and refining (Table 2.20).

c. The following liquid effluents are projected -

- 490 kg/day of flotation chemicals and 18 kg/day lead from lead mining and milling (Table 2.7);
- 0.43 kg/day of lead and of cadmium and 150 kg/day of sulfate from lead smelting and refining (Table 2.11);
- 22 kg/day of lead and 600 MT/day of sodium sulfate (if lye neutralization is used) from battery manufacturing using wet process; dry process emissions are expected to be larger by a factor of 2 to 3 (Table 2.20);
- 0.11 kg/day of lead and 0.53 kg/day of antimony and 4.2 MT/day of sulfates from the battery breaking industry (Table 2.27).

d. The following solid wastes are projected -

- 17,000 MT/day of mill tailings containing 16 MT of lead and 0.28 MT of cadmium (Table 2.7);
- 87 MT/day of sludge containing 12 MT of lead and 0.6 MT of cadmium from primary smelting and refining (Table 2.11);
- 1700 MT/day of $CaSO_4$ sludge (if lime neutralization is used) containing 0.4 MT of lead and 0.008 MT of antimony from battery manufacture (Table 2.20);
- 9.3 MT/day of sludge containing 23 kg of lead and 25 kg antimony from battery breaking (Table 2.27);
- 260 MT/day of sludge containing 0.14 MT of lead from secondary smelting and refining (Table 2.28).

e. Stibine and arsine gases can be generated during charging of the batteries which contain antimony and arsenic as additives. These gases are unstable and will decay to oxides in the form of dust. If EVs are charged in confined areas, such as home garages, measurable accumulations of antimony and arsenic oxide dusts on garage walls and floors are expected to occur over a period of time.

6. For the assumed scenario of eight million Ni/Zn battery-powered electric vehicles on the road by the year 2000:

a. The following impacts on the industries related to Ni/Zn battery are projected -

- a 48% increase in refined nickel production will be required as compared to current world output (Table 3.15);
- a 64% increase in cobalt production will be required as compared to current world output (Table 3.15);
- a 32% increase in KOH production will be required as compared to current U.S. output (Table 3.15);
- a Ni/Zn battery recycle industry will need to be developed because of the scarcity and cost of nickel and cobalt.

b. The following airborne emissions are projected -
- 19 MT/day of SO_2 from primary zinc production (Table 3.7);
- 20 MT/day of particulates from primary nickel production (Table 3.9);
- 610 MT/day of SO_2 from primary nickel production (Table 3.9);

c. The following liquid effluents are projected -
- 85,000 MT/day of water from primary zinc production (Table 3.7);
- 270,000 MT/day of water from primary nickel production (includes mining, milling, smelting and refining) (Table 3.9).

d. 16,000 MT/day of solid wastes from primary zinc production are expected to be produced.

7. For the assumed scenario of eight million Ni/Fe battery-powered electric vehicles on the road by the year 2000:

a. The following impacts on the industries related to Ni/Fe battery are projected -
- a 30% increase in refined nickel production will be required as compared to current world output (Table 4.14);
- a 35% increase in cobalt production will be required as compared to current world output (Table 4.14);
- a 26% increase in KOH production will be required, as compared to current U.S. output (Table 4.14);
- a 100% increase in LiOH production over current U.S. output will be required (Table 4.14);
- a Ni/Fe battery recycle industry will need to be developed because of the scarcity and cost of nickel and cobalt.

b. The following airborne emissions are projected -
- 380 MT/day of SO_2 from primary nickel production (Table 4.6);
- 7 MT/day of CO from iron and steel production (Table 4.4);
- 10 MT/day of particulates from primary nickel production (Table 4.6).

c. The following liquid effluents are projected -

- 18 MT/day of pickle liquor discharges from steel production (Table 4.4);
- 170,000 MT/day of wastewater discharges from nickel production (includes mining, milling, smelting, and refining) (Table 4.6);
- 1600 MT/day of wastewater discharges from LiOH industry (Table 4.10).

d. The following solid wastes are projected -

- 96 MT/day of slag from steel production (Table 4.4);
- 2800 MT per day of slag from LiOH production--chiefly from mining and milling (Table 4.10).

8. The emission levels were assessed using current control technology. Because no large-scale production facilities are under operation for the Ni/Zn and Ni/Fe batteries, information is not available for assembly-line operations. We have relied heavily on our own estimates which may or may not correspond closely to those from actual production facilities installed at some future date. The estimates of emissions are expected to be reasonable; however, there is a need for updating and/or revising the emission data as more information becomes available.

9. The generic dispersion analyses were prepared for emissions from the total battery cycles. The amounts and sources of emissons used in the dispersion analyses are as identified in Sections 2, 3, and 4 and summarized in items 5, 6, and 7 above. Analyses are presented for the total installed capacity in the year 2000 for these batteries, as well as for unit megawatt hour. Atmospheric concentrations, deposition rates, soil concentrations, and surface water runoff concentrations were calculated for conditions approaching the worst case. Wastewater dispersion analyses were made only for potentially hazardous emissions. Detailed dispersion analyses are not provided for solid wastes; it is assumed that potentially hazardous wastes will be contained and disposed of as required under the Resource Conservation and Recovery Act of 1976 (Sec. 5).

10. a. The elements emitted to the environment from the battery cycles are cycled among biota and their physical environments to some degree. Bioaccumulation of these elements above ambient levels is likely in aquatic ecosystems but not in terrestrial ecosystems. However, in terrestrial ecosystems higher-than-normal tissue concentrations do occur in polluted areas containing these elements. Biomagnification of elements along the food chain is not likely to be important for the major constituents of the emissions from the battery cycle.

b. The lead/acid battery-related aerial emissions and wastewater constituents could cause adverse human health and ecological impacts within several kilometers of the operations (Sec. 6.2.1). Lead, arsenic, and antimony appear to be hazardous emission constituents and exceed estimated permissible concentrations for the protection of health (EPC_H) and for the protection of ecology (EPC_E) in one or more of the battery-related industries (Tables 6.18, 5.1-5, and 5.18-5.22).

c. Zinc, nickel, lead, cadmium, cobalt, and sulfur dioxide appear to be the major hazardous emissions constituents for the nickel/zinc battery cycle exceeding EPC_H and EPC_E values within several kilometers of the facilities (Sec. 6.2.2 Table 6.18) in one or more of the battery-related industries (Tables 5.6-5.11 and 5.23-5.27).

d. Nickel, cobalt, lead, total particulates and sulfur dioxide appear to be major hazardous emissions constituents for the nickel/iron battery cycle exceeding EPC_H and EPC_E values within several kilometers of the facilities (Sec. 6.2.3 Table 6.18) in one or more of the battery-related industries (Tables 5.12-5.17 and 5.28-5.32).

11. Emissions from the three near-term battery cycles contain several potentially hazardous chemicals. The biomedical effects of these chemicals have been assessed in context of the scenarios described above. The anticipated effects of these chemicals are summarized below.

a. Antimony and the Gas Stibine (Sec. 7.1)

- Based on measurements made with a load-leveling lead/acid battery and assuming total release without decomposition of the stibine produced inside the battery, maximum possible stibine levels in home garages during battery charging are calculated to be 1 to 7 times the TLV (Threshold Limit Value) of 0.5 mg/m^3. The decomposition of stibine could result in maintenance of antimony trioxide levels in the garage close to the TLV of 0.5 mg/m^3. Antimony trioxide will be deposited in the garages and higher levels in air can be expected during garage cleaning operations. Stibine is a potent hemolytic agent. Information on toxic responses to low levels of stibine is not available. Analogy to arsine must be relied upon for estimating toxic responses to stibine. Inhalation exposure to high levels of antimony trioxide (45 to 125 mg/m^3) produced degenerative changes in the lungs, liver and spleen after 30 to 45 days; effects of low level exposures have not been reported.
- Past experience among antimony smelter workers has indicated a pneumoconiosis incidence rate of 10 to 15 percent. Exposure of smelter workers to antimony sulfide ore dust could result in degenerative changes of the heart muscle. Such effects have been observed in both animals and man following exposure for short periods (6 weeks in animals, $\leq$ 2 years in man) to antimony sulfide at 5 mg/m^3 (10 times the OSHA standard).

b. Arsenic and the Gas Arsine (Sec. 7.2)

- Occupational and public exposure to arsenic will increase in direct proportion to the increased use of the lead and zinc ores it contaminates.
- Arsine is generated along with stibine during charging of the lead/acid battery; maximum garage levels of arsine are estimated at less than one-tenth the TLV.
- Arsine is a potent hemolytic agent and low-level chronic exposures in humans have been known to cause slight decreases in hemoglobin and red blood cell levels.

c. Cadmium (Sec. 7.3)

- As a contaminant of lead and zinc ores, cadmium is of primary health concern for the smelter work force, the population living in the vicinity of the lead and zinc smelters, and the workers involved with nickel electrode impregnation during Ni/Zn battery manufacture.
- Cadmium oxide fumes at levels at or below the OSHA standard (0.1 mg/m^3) have been associated with emphysema. Occupational exposure data indicate that proteinuria develops with a few years' exposure to CdO fumes at 0.07 to 0.2 mg/m^3 or to dust at 0.02 to 0.7 mg/m^3. Dusts are generally considered less toxic than fumes. Studies linking cadmium exposure to specific kinds of cancer are inconclusive.

d. Cobalt (Sec. 7.4)

- Cobalt is a component of the nickel electrode in the Ni/Zn and Ni/Fe batteries. Cobalt is toxic only at very high levels of exposure.
- There is no epidemiological evidence associating occupational exposure to cobalt with increased risk of cancer.

e. Lead (Sec. 7.5)

- Significant increase in the occupational exposure and environmental release of lead can be expected with expansion of lead/acid battery industry.
- Toxic responses to lead of concern to exposed populations include effects on the hematopoietic, the renal and the nervous systems. Young children are particularly susceptible to toxic effects of lead exposure.

f. Nickel (Sec. 7.6)

- Epidemiological studies have shown that exposure to nickel in various types of nickel refineries has led to an increased incidence of tumors of the lung and paranasal sinuses. Chronic rhinitis, nasal sinusitis, and precancerous nasal lesions have also been observed in the occupational setting.
- Nickel subsulfide, a major component of nickel refinery flue dust, has been demonstrated to produce a 14% incidence of lung tumors in rats at the OSHA standard for nickel compounds (0.1 mg/m^3). Nickel carbonyl, a compound involved in the Mond process for nickel refining, is lethal to man following acute exposure to 30 ppm by volume.
- Nickel oxide and nickel chloride at 0.1 mg/m^3 produce detrimental lung changes in rats following several weeks of exposure. Inhalation exposure of workers to nickel compounds during nickel electrode manufacture for Ni/Zn and Ni/Fe batteries should be kept low enough to prevent such adverse impacts.

12. a. The State Implementation Plans for the limitations on atmospheric emissions of sulfur dioxide may pose an impediment to the development of an expanded EV fleet. (Sec. 8.1.1).

b. New sources which may be needed to meet the demands of an expanded EV industry will require compliance with minimum Federal standards as promulgated under the Clean Air

Act and its Amendments and local limitations. This generally entails permits prior to construction and operation of the new source (Sec. 8.1.1).

c. Although the Clean Air Act Amendments of 1977 provides a means for special relief for nonferrous smelters in meeting air pollution standards, Congress has mandated the U.S. EPA to proceed with a vigorous enforcement program against smelters. These air pollution limitations and associated costs for pollution control equipment will provide incentives for hydrometallurgical metal separation rather than smelting operations for copper, cobalt, nickel and zinc.

d. The U.S. EPA has proposed standards for atmospheric emissions of lead from new, modified, and reconstructed lead/acid battery plants which have a production capacity equal to or greater than 500 batteries per day. The U.S. EPA considers the economic impact of these proposed emissions limits to be reasonable and does not expect that they will prevent or hinder the expansion of the lead/acid battery manufacturing industry.

e. Sulfuric acid mist is generally not regulated. The control of hazardous mists may be required for near-term battery manufacturing plants (Sec. 8.1.1).

f. The U.S. is currently considering NESHAP (National Emission Standards for Hazardous Air Pollutants) for arsenic and lead (Sec. 8.1.1).

g. The requirements for best available technology for toxic substances, as well as pretreatment standards for discharges of pollutants to publicly owned treatment works, will necessitate water-pollution control compliance for the manufacturing and recycling phases of all three near-term battery systems (Sec. 8.1.2).

h. There will be liquid emissions associated with battery breaking facilities which will require treatment prior to disposal (Sec. 8.1.2).

i. The following components in the three near term battery cycles are classified as hazardous waste generators under Section 2001 of the Resource Conservation and Recovery Act:

- primary lead blast furnace dust
- primary lead lagoon dredging from smelter
- zinc anode sludge
- primary antimony-electrolytic sludge
- primary lead sinter dust scrubbing sludge
- secondary lead scrubber sludge from sulfur dioxide emission control, soft lead production
- secondary lead - white metal production furnace dust
- lead/acid storage battery production wastewater treatment sludges
- lead/acid storage battery production clean-up wastes from cathode and anode paste production (Sec. 8.1.3).

Any generator of a hazardous waste so classified must follow special handling and disposal practices as established by the USEPA and/or the state.

j. The implication to industries in the near-term battery cycles of Resource Conservation and Recovery Act regulations, when they are finalized, is that additional costs will be incurred by such industries because those wastes classified as hazardous will require special treatment. In general, the regulations do not pose technical barriers for compliance on the part of various segments of the near-term battery industries. Rather, they will result in an economic impact which is currently a matter of debate (Sec. 8.1.3).

k. In general, it appears that the regulatory limitations will result in a reduction in the number of smaller plants and firms in the battery industry in favor of larger-scale operations (Sec. 8.1.3).

13. This document does not represent a final and definitive assessment of emissions from near-term storage battery cycles and their impacts on ecosystems and human health. The information information. Suggestions for revision should be sent to:

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ECOLOGICAL AND BIOMEDICAL EFFECTS OF EFFLUENTS FROM NEAR-TERM ELECTRIC VEHICLE STORAGE BATTERY CYCLES

ABSTRACT

An assessment of the ecological and biomedical effects due to commercialization of storage batteries for electric and hybrid vehicles is given. It deals only with the near-term batteries, namely Pb/Acid, Ni/Zn, and Ni/Fe, but the complete battery cycle is considered, i.e., (1) mining and milling of raw materials; (2) manufacture of the batteries, cases and covers; (3) use of the batteries in electric vehicles, including the charge-discharge cycles; (4) recycling of spent batteries; and (5) disposal of nonrecyclable components.

It is assumed the market will grow at a rate of 25% per year for Pb/Acid batteries and 30% for Ni/Zn and Ni/Fe batteries and will reach a total of three million Pb/acid and eight million Ni/Zn or Ni/Fe powered electric vehicles on the road by the year 2000. Assessments are made both in terms of each megawatt-hour of installed capacity (assuming a 1-MWh requirement for each 40 electric vehicles), and of the total number of EVs expected for each type of battery.

The gaseous, liquid, and solid emissions from various phases of the battery cycle are identified. The effluent dispersal in the environment is modeled and ecological effects are assessed in terms of biogeochemical cycles. The metabolic and toxic responses by humans and laboratory animals to constituents of the effluents are discussed. Pertinent environmental and health regulations related to the battery industry are summarized and regulatory implications for large-scale storage battery commercialization are discussed.

1. INTRODUCTION

ABSTRACT

The purpose and scope of the document is described. The procedures followed in assessing the impacts of the near-term batteries for electric vehicle applications on human health and the environment are outlined. Factors limiting a more thorough assessment are discussed.

By virtue of the Electric and Hybrid Vehicle Research, Development, and Demonstration Act of 1976 (PL 94-413), the Department of Energy has been charged with the responsibility for research, development and demonstration of electric storage batteries for electric and hybrid vehicle applications.* Because of this responsibility, the Department of Energy, as mandated by the National Environmental Policy Act of 1969, also has the obligation to assess the impacts of this technology on the environment. To carry out this obligation, the ecological, biomedical and health effects of commercialization of electric storage batteries as related to electric and hybrid vehicle applications have been assessed, and a summary and analysis are presented in this document. This document is intended to provide information that may be needed to prepare an environmental assessment or an environmental impact statement for specific projects.

The batteries being considered or developed for electric vehicle application fall within two general categories: (1) *near-term batteries*, including lead/acid, nickel/zinc (Ni/Zn), and nickel/iron (Ni/Fe) batteries, and (2) *advanced batteries*, including Li-Al/metal sulfide, Na/S, and Zn/Cl_2 batteries. The near-term batteries currently are being used in DOE/DOT demonstration vehicles. Research on advanced batteries continues for their application to electric vehicles in the 1985-2000 time frame. Other batteries, such as Al/air and Zn/Br_2, are considered too speculative and long-range for this time frame. This assessment deals only with the near-term batteries.

Each of the three near-term battery systems under consideration has its debits and its merits. At this time, no clear overall superiority is indicated for any one system. The characteristics of these batteries and their respective developmental problem areas have been discussed by Yao, Ludwig, and Hornstra[1] and are summarized in Table 1.1. The following descriptions are based upon an assessment of the technology of these battery systems by the Office for Electrochemical Project Management, Argonne National Laboratory, which manages, directs and coordinates industrial cost-shared contracts for improving these technologies as part of DOE's Electric and Hybrid Vehicle R&D Program.

**Potential applications of such batteries also include wind and solar energy conversion and utility load leveling whereby electrical energy can be stored during off-peak hours for use during peak demand periods.*

Table 1.1. Promising Near-Term Batteries for Transportation End Uses[a]

Battery System	*Present Specific Energy*	*Developmental Problem Areas*
Lead-Acid	40 Wh/kg	Improve cycle life, increase specific energy
Ni/Fe	50 Wh/kg	Reduce cost, increase efficiency, improve low-temperature performance
Ni/Zn	70 Wh/kg	Increase cycle life, reduce cost

[a] *From N.P. Yao, F.A. Ludwig, and F. Hornstra, "Overview of Near-Term Battery Development," Paper No. 783101, in Proc. 5th International Electric Vehicle Symp., Philadelphia, October 2-5, 1978.*

Lead-acid batteries consist of improved state-of-the-art (ISOA) and advanced batteries. The ISOA lead-acid battery has an energy density of 40 watt-hours per kilogram (Wh/kg) and should be capable of a lifetime of 500 to 800 cycles in the near future. The performance characteristics of ISOA lead-acid batteries indicate that these batteries are suitable for use in limited-performance electric vehicles. However, the cost advantage of these batteries, resulting from relatively low cost materials and an established manufacturing industry, should persist; and even with the emergence of other higher performance systems, lead-acid batteries are expected to retain a significant share of the EV market.

The advanced lead-acid battery, which features significant departures from the existing technology, has technical goals of an energy density of 60 Wh/kg and a cycle life of 1000 deep cycles.* Achievement of either goal separately is not difficult to attain; however, the combination of both goals, while at the same time maintaining low cost, poses a very challenging objective. Improvement in the utilization of active materials (PbO_2 in the positive electrode and Pb in the negative) is recognized as one of the important ways to achieve higher energy densities.

The nickel/iron system has a demonstrated long life capability over deep discharge cycles and an ability to withstand rugged use. The battery has a specific energy of 45-55 Wh/kg; increase of the specific energy to about 60 Wh/kg appears attainable. Although the initial cost of the battery is the highest of the three near-term systems because of its longevity, the life-cycle costs make the system very attractive, especially for commercial fleet applications. Unfavorable factors include the evolution of large quantities of hydrogen during charging (creating reduced energy efficiency and hydrogen safety concerns) and reduced performance as battery temperatures drop below 10°C.

**A deep discharge cycle is a discharge to 80% of capacity. A battery is considered to have failed if its storage capacity has fallen to 80% or lower of its rated value. [Gould, Inc. "Nickel-Zinc Battery Development for Electric Vehicle Propulsion" in Second Annual Battery and Electrochemical Technology Conference, June 5-7, 1978, Arlington, VA, Agenda and Technical Presentations, U.S. Department of Energy, Report No. Conf. 780603, UC-94C, May 1978.]*

The nickel/zinc battery offers the highest specific energy of the near-term systems, presently about 70 Wh/kg, with an additional 25% improvement expected in the future. The peak-power and sustained-power characteristics are excellent. However, this battery suffers from low cycle life at deep discharges. The present cycle life of about 100 to 200 deep cycles in full-size cells may be extended to 400 to 600 deep cycles by 1984.

Like the nickel/iron battery, the initial cost of this battery is also high. Commercialization will be very sensitive to the availability and world market price of nickel, because imports presently are the source of 76% of the nickel consumed in the U.S. annually.

It thus seems reasonable to conclude that on balance, each near-term battery system has more or less equal probabilities for large-scale commercial market penetration. In fact, it is likely that each may be commercialized for specific applications appropriate to the battery's characteristics.

Storage battery technology has been considered in its totality in this document. The complete battery cycle comprises (1) mining and milling of the necessary raw materials; (2) manufacture of the batteries and their cases and covers; (3) use of the batteries in electric vehicles, including the charge-discharge cycles; (4) recycling of spent batteries; and (5) disposal of non-recyclable components. An effort has been made to identify the gaseous, liquid and solid emissions from various phases of the battery cycle. These effluents are dispersed in the environment either as controlled emissions, for which emission standards or criteria are mandated under a legal framework, or as uncontrolled emissions either for which no technology has been developed or for which technology has not been applied for curtailing emissions (and for which no legal requirements must be met). Ecological effects of these dispersed effluents are considered in terms of biogeochemical cycles.

Metabolic and toxic responses by humans and laboratory animals to battery constituents are discussed, along with toxicity in workplace and public exposure scenarios. Section 8 of the report contains a summary of pertinent environmental and health regulations related to the battery industry, and includes a discussion of how these regulations may become limiting factors in commercialization of the storage batteries.

The magnitudes of environmental impacts resulting from storage battery commercialization depend on the extent of market penetration of each battery type. Because a clear choice of the most feasible battery system is not possible, it is difficult to project the market penetration scenarios for total number of electric vehicles and for the mix of various batteries for a given year. Considerable study on electric vehicle market penetration scenarios has been conducted by the Energy and Environmental Systems Division of Argonne National Laboratory. Market penetration depends not only on the availability of a suitable storage battery, but also on such factors as relative costs as compared to internal combustion engine (ICE) vehicles, development of an infrastructure for sales and service, consumer acceptance, and the load and range characteristics of electric vehicles. Many of these factors are interrelated; thus, forecasts are sensitive to changes in any of the variables. Of necessity then, there is a need for a continued updating and refinement of market penetration scenarios as the technology and demonstration program advances.

It is beyond the scope of the present study to assess the impacts of a multitude of market-penetration scenarios. After careful scrutiny of the information currently available, we have

adopted the following procedure to estimate effluents from the total battery cycles: (1) The effluents are estimated on the per-megawatt-installed-capacity of the battery. This approach on a per-unit basis makes it convenient to determine the total effluents for any scenario by simple extrapolation. (2) The market penetration is assumed to be 3×10^6 lead/acid battery-powered vehicles and 8×10^6 each of the Ni/Zn and Ni/Fe battery-powered vehicles on the road by the year 2000. These numbers correspond to the low scenario for the lead/acid battery and medium scenario for the Ni/Zn and Ni/Fe batteries, as given by Singh et al.[2-7] As more definitive scenarios emerge, the amount of effluents can be adjusted accordingly in revisions of this document.

Because no large-scale production facilities have been designed or are under operation for the Ni/Zn and Ni/Fe batteries, information is not available for assembly-line operations and the resulting emissions in the workplace and the environment. Our approach has been to describe a representative battery assembly in a logical manner with all needed ingredients in order to characterize potential environmental impacts. Actual assembly-line, large-scale operations may not follow a similar sequence or techniques. The authors have relied heavily on their own calculations and estimates, which may or may not correspond closely to those from actual production facilities installed at some future date. Despite these uncertainties, the estimates of emissions are expected to be reasonable; however, the need for updating and/or revising the emission data as more information becomes available is obvious.

Descriptions of the front-end impacts of mining operations in this document have been derived largely from case histories. It has not been possible to assess more completely the emissions from nickel mining and milling operations, because most of the nickel used in the United States is imported. The primary exporter to the United States is Canada. Efforts to procure information concerning Canadian operations were not successful. Cobalt also is imported, principally from Zaire, and it has not been possible to ascertain emissions from cobalt mining and milling.

The concentration of certain additives in the battery manufacturing processes remains proprietary information and we have used the best available estimates.

Regardless of all the precautions and care that can be taken in predicting emissions, there remains the problem of cycle loss. The lead/acid battery industry reports greater than 10% cycle loss. Specifically where this loss occurs in the production and recycling is not known. Thus, it can be safely stated that a material-balance approach is neither feasible nor practical for the battery industry, at least for the current situation. It is likely that the cycle loss may be reduced with more strict controls.

This document does not represent a final and definitive assessment of emissions from storage battery cycles and their impacts on ecosystems and human health. The information presented here should be revised to accommodate more accurate or additional information.

References

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2. EFFLUENTS FROM THE STATE-OF-THE-ART LEAD/ACID BATTERY CYCLE

ABSTRACT

The lead/acid battery cycle--from mining and milling of raw materials through recycling of scrap batteries and disposal of wastes--is described. Material flows and emissions for various phases of the total cycle are estimated for per megawatt hour of installed capacity and for a scenario of three million lead/acid electric vehicles on the road by the year 2000.

The main steps in the state-of-the-art (SOA) lead/acid battery cycle consist of battery manufacture, use, battery breaking, and secondary recovery. Another important step is the primary generation of new materials which, together with the output from secondary recovery plants, make up the total material input to the manufacturing step. Battery breaking consists, in essence, of preparation of the battery for input to the secondary processes for recovery of some of the component materials. Besides these main steps in the cycle there are smaller ones such as transportion of component materials and finished batteries, and installation and removal of the batteries.

For each stage in the above cycle the effluents to the environment are characterized and quantified. That is, for each stage air emissions (as particulates, gases, and fumes), liquid emissions (as suspended or dissolved in water), and solid emissions have been considered. For each emission class, the chemical compounds (if known) and the amounts of each emitted have been determined. In most cases the amounts of different chemical compounds of an element which are emitted are not available. For these cases, the total amounts of the element in the emission class are given (e.g., total air emissions of lead in battery manufacturing).

The cycle represents a dynamic system. Thus, amounts of materials are given in terms of rates of material flow per unit installed capacity in EVs as kilograms per day per megawatt hour of installed capacity (kg/day per MWh). These units are convenient for scaling to any size industry. That is, to obtain the total emission rate of an effluent at any stage in the cycle, for an assumed size of the industry, one multiplies the amounts given by the total assumed capacity in MWh installed in EVs (i.e., total emission rate in kg/day = amount in kg/day per MWh × total MWh installed). These units also allow one to take account of EVs with different sized batteries, such as trucks and buses. For an EV battery capacity of 25 kWh, 1 MWh installed capacity is equivalent to 40 EVs.

The SOA lead/acid cycle represents the present-day pollution control technologies and industrial processes in use. The following assumptions have been made for calculating material flow rates and emissions:

- The percentage of materials recycled is increased from present-day figures to reflect increased resource conservation.

- All materials flowing in the cycle are produced in plants using modern pollution control technologies, even though at present only some of the industrial plants for a given process may be equipped with pollution control devices.
- The analysis will be given for state-of-the-art lead/acid batteries, even though by the time the EV fleet size is projected to be appreciable, in the 1990s, a more advanced lead/acid battery may be commercially available.

It is possible that, due to these assumptions, the industrial activity and emissions projected here for the 1990s may be too conservative. That is, the projected industrial activity and emission levels are greater than those which will actually occur. However, because of all the uncertainties present in making such projections, it seems prudent to consider batteries, industrial processes, and pollution control technologies which are commercially available now or are expected to become so in the immediate future. As a result any emission which is considered acceptable by today's standards will, barring future tightening up of standards, be acceptable in the 1990s.

2.1 INPUT PARAMETERS

It is assumed that the SOA lead/acid EV battery industry will grow at a rate of 25% per year,[1] and that by the year 2000, 3×10^6 EVs which use lead/acid batteries will exist.[2] The growth rate is that used by TRW[1] in a medium scenario and is close to the 27.5% value used by Bernard[2] in the low scenario. This scenario provides a fleet size of 250,000 and 870,000 EVs in the years 1990 and 1995, respectively. The EV numbers for the years 1990, 1995, and 2000 are used to illustrate both the impact of the growth on the existing industry and the growth in effluent amounts during the 1990's.

The typical EV using SOA lead/acid batteries is assumed here to have a 25 kWh battery with an energy density of 35 Wh/kg (at a 3-hour discharge rate) and a life of 500 deep discharge cycles.[3] The range of a typical EV using SOA lead/acid batteries is taken here to be 105 km, based on the assumption that for vehicles with a given battery weight fraction the range is proportional to the battery energy density.[1] Use of the values given by Hamilton[4] for EVs using advanced lead/acid batteries [battery weight fraction = 0.38, energy density = 50 Wh/kg, vehicle range = 150 km (SAE J-227a(D) driving cycle)] with the above assumption gives a range of 105 km for a battery weight fraction of 0.38 for SOA lead/acid batteries.

It is assumed that the average distance an electric vehicle is driven is 16,000 km/yr, or 44 km/day.[1,4] This is the distance an average car travels in a year.[4] Combining this with the range and cycle life parameters given above yields an average SOA lead/acid battery life of 3.3 years before it must be replaced.

In order to estimate the effluents resulting from large-scale manufacturing, use, and recycling of SOA lead/acid batteries in EVs, one also needs to know the composition of the batteries. To this end Table 2.1 gives the composition of present day lead/acid batteries in terms of weight percent and metric tons per megawatt hour (MT/MWh).*[5-8] These values are assumed to hold for all SOA lead/acid batteries.

**This breakdown refers to an average analysis of golf cart batteries made by four different manufacturers.[5] Golf cart batteries are probably more representative of future EV batteries than are standard auto batteries because they are designed for use in charge-discharge cycles.*

Table 2.1. Composition of SOA Lead/Acid Battery for EV Use

Material	Composition	
	Wt %	MT/MWh
Pb[a]	63	18.0
Sb[b]	1.3	0.37
As[b]	0.022	0.0063
Cu[c]	0.44	0.13
Electrolyte[a]	25	7.1
H_2SO_4 (pure)	9	2.6
H_2O	16	4.5
Case and Cover[c]	6.6	1.9
Separator[d]	0.34	0.097
Expander[e]	0.14	0.040
Other	3.2	0.91
TOTALS		
Dry Weight	75	21.5
Battery	100	28.6

[a] *Sources: G.S. Hartman, "Lead Acid Development," in Agenda and Technical Presentations, Second Annual Battery and Electrochemical Technology Conference, June 5-7, 1978, U.S. Department of Energy, CONF-780603, 1978.*

J.O. Ledbetter, "Battery Energy Storage," in Environmental Control Technology, R&D Requirements for Energy System, E.L. Kaufman, Los Alamos, N.M., Final Report No. LA-6979-Ms, September, 1977.

Review on Lead Acid Battery Science and Technology, J. Power Sources 2:3-120, 1977-78.

Environmental Development Plan, Electric Vehicle Systems FY 1978, U.S. Energy Research and Development Administration, Report No. EDP/C-01(77), August 1977.

[b] *Sources: G.S. Hartman, op. cit.*

J.O. Ledbetter, op. cit.

R. Varma and N.P. Yao, "Stibine and Arsine Generation for a Lead Acid Cell During Charging Mode Under a Utility-Load Levelling Duty Cycle," Argonne National Laboratory, JB. Report No. ANL/OEPM-77-5, March 1978. The positive and negative active materials contain essentially no antimony or arsenic.

[c] *Source: J.O. Ledbetter, op. cit.*

[d] *Source: "Assessment of Industrial Hazardous Waste Practices Storage and Primary Batteries Industries," Versar Inc., report prepared for the U.S. Environmental Protection Agency, January 1975, U.S. Dept. of Commerce, Report No. PB-241,204, Section 5.2, 1975.*

[e] *Modified from "Lead-Acid Battery Manufacture, Background Information," Proposed Standards, Draft, USEPA, North Carolina, May 1979, Section 3.*

The lead weight percent (antimony-free) is taken to be 63%, 58% of which is assumed to be in the grids and active materials,[5] and 5% in the posts and connectors.[6] (Other percentages reported in the literature for total lead have been 61%,[6] 62%,[7,8] 66%,[9] and 70%.[7]) This 58% may be further broken down into 18% (antimony-free) lead in the positive active material (PbO_2), 17% in the negative active material (Pb), and 23% in the grids (4.5% antimonial lead).[5]

The weight percents of antimony (Sb) and arsenic (As) given in Table 2.1--1.3% and 0.02%, respectively--are taken from a recent analysis of an industrial cell.[10] These figures are obtained under the assumption that the active materials are free of antimony and arsenic and that the total lead in the grid, post, and connector contains 4.5% Sb and 0.08% As. (The positive grid contained 4.6% Sb and 0.08% As and the negative grid contained 4.3% Sb and 0.08% As.) The antimony value of 4.5% falls within the range of the 2.75% to 6.25% that various manufacturers specify for grid material.[7]

Because of self-discharge effects and arsine and stibine generation during charging of the batteries, work is going on to reduce the Sb and As content of the grid metal. However, as Sb, and to some extent As, have beneficial effects on the grid properties, it is not clear how much of a reduction can be achieved.[7,10-12]

In addition to Sb and As, the grid metal and active materials contain other elements as minor contaminants.[7] The grid metal may contain small amounts of tin, silver, iron, manganese, copper, nickel, cobalt, zinc, bismuth and cadmium. There is ongoing research in which some of these metals, as well as selenium and tellurium, are used to replace antimony. Antimony, arsenic, and tin in the lead-containing oxide from which the active materials are made total about 0.005%. Other minor contaminants are bismuth, copper, iron, silica, nickel, cobalt, silver, zinc, cadmium and calcium.

Other than antimony and arsenic, the above contaminants will be ignored in characterizing and quantifying effluents from the lead-acid battery cycle. The main reason is that as contaminants, their concentrations (barring unforeseen beneficial effects) can be reduced below the present values.[7] Similarly, the use of calcium and tin in the maintenance-free batteries will not be considered here (such batteries do not stand up well to repeated charge-deep discharge cycling).[10-12]

The small amount of copper listed in Table 2.1 is used to improve the electrical conductivity in the battery posts.[6,13]

The weight percent of electrolyte, a 36% H_2SO_4 solution, has values ranging from 18.6% to 28.4%.[5-8] Somewhat arbitrarily, the percentage of electrolyte was taken here to be 25%. This gives respective weight percents of 9% H_2SO_4 and 16% water.

The case and cover, which comprises 6.6% of the battery,[6] traditionally has been made of hard rubber. However, rubber cases are heavy and hard to dispose of, as they cannot be burned. Now battery cases and covers are being made of plastics such as polyethylene, polypropylene, polyvinyl chloride and polycarbonate.[14,15] Polypropylene is recyclable,[16] as are some of the other thermoplastics. Polyvinyl chloride, a self-extinguishing flammable plastic, can reduce the fire hazard when used as an EV battery case.[14]

Separators have several important functions in a battery, such as preventing contact between electrodes of opposite polarity, preserving the electrode shape, and allowing free electrolyte flow. Traditionally they have been made of cellulose fibers. However, other materials such as microporous polyethylene, sintered polyvinyl chloride, and fiberglass mats are being used.[14,16] The separator weight percent given here is 0.34%.[17]

The expander, which comprises about 0.7% of the lead oxide (PbO) in the negative battery paste,[18] or 0.8% of the negative active material, is composed of carbon black, organics and barium sulfate.[18] Because the negative active material takes up 17% of the battery,[5] the expander takes up about 0.14% of the battery weight.

The remaining constituents have been grouped together in the table under the heading of "Other". These include the oxygen (2.8% of the total battery weight) which is present in the positive active material as well as other miscellaneous hardware, a small amount of dynel fibers, etc.[18]

It is assumed that 90% of the lead in the battery will be recycled and that 10% will be lost to the environment.[6]* This is appreciably more than the 80% recycling rate for battery lead which was given in a 1971 article[19] and in a more recent publication.[20] Because EV batteries are much larger than present-day auto batteries, and regulations governing discharge of lead into the environment can be expected to become more stringent in the future; the 90% figure may be somewhat low for the 1990s.

For ease in constructing the scenario it will be assumed that the 10% lead lost to the environment will be lost at one point in the cycle and as whole batteries. (Clearly this is an unrealistic assumption. Losses will occur at various points in the cycle--such as transportation of the lead to and from various facilities, and loss during loading and unloading--and from various parts of the battery during different manufacturing and recycling steps. It is beyond the scope of this report to attempt a quantitative estimate of the amounts and distribution of such losses; therefore, this simplifying assumption is made.) Because the percent loss is only 10%, removing or changing it will have, at worst, a small effect on the calculated effluents and material flows in the cycle.

During recycling, the battery cases and covers can be disposed of in landfills, burned as fuel, or returned to the cycle either as cases and covers or, if plastic, as plastic materials for re-use.[16] Most cases are made today of various plastics (such as polypropylene or polethylene)[14,16] but rubber is also used. At present, at least one manufacturer burns the polypropylene cases as fuel in the secondary smelting operation.[15] Here it will be assumed that the used cases and covers will be recycled either as fuel or as plastic feed material, and essentially none (other than some of the above 10% loss), will end in landfill. How one apportions the amounts of cases and covers recycled as plastic feed material or as fuel affects only a small part--the plastic case manufacturing part--of the cycle, and has no effect on the rest of the cycle.

The remaining components shown in Table 2.1 will be considered as nonrecyclable,[6,13,15,16] although even this could change as the result of a changing economic and regulatory environment.

Since the total amount of air, liquid, and solid emissions of lead to the environment summed over the manufacturing, use, battery breaking, and recycling operations is much smaller than 10% the figure of 10% can be regarded to represent other unknown types of losses.

2.2 MATERIAL FLOW RATES

An overall view of the materials flow within the lead-acid cycle, which is given in Figure 2.1, shows cycle activities and components, the emissions to the environment (arrows without terminal boxes), and the flow directions (other arrows). The numbers along the main cycle arrows give the flow of lead (not batteries) in kg/day per installed MWh (28,600 kg of batteries) in electric vehicles. As noted before, it is assumed for simplicity that all the cycle losses occur as new batteries before they are used.

After being manufactured, the completed batteries are transported and installed in electric vehicles. After some time of use, the batteries must be removed and replaced with new ones. The spent batteries are sent to a battery breaking plant where the tops are removed, the electrolyte dumped, and the lead plates are removed.[19] The lead plates are recycled to a secondary smelter which produces lead in a form suitable for battery manufacture. The battery breaking, secondary smelting and battery manufacture are sometimes carried on at one location.[19] As indicated in Figure 2.1, antimony and arsenic can be added to the lead during the smelting and refining operations or as part of the manufacturing step.

To estimate the emissions resulting from various activities in this cycle, it is first necessary to obtain the flow rates of the various battery components and materials through the cycle. As discussed in Section 2.1, a 25% annual growth rate for the numbers of electric vehicles[1] and a 3.3-year SOA lead/acid battery lifetime are used. These parameters, together with the battery component percents and weights as given in Table 2.1, yield the various component and material flow rates, in kg/day per MWh installed, which are given in Table 2.2. The column headed "Total" gives the output flow rate of materials and components in new batteries from the manufacturing step. The "Loss" column gives the loss rate of materials and components as new batteries under the assumptions previously discussed (10% of the flow rate of new batteries installed in EVs is taken as the loss rate and the loss is assumed to occur as new batteries immediately after the manufacturing step). The flow rates of materials in the cycle are not much affected by the loss rate. For example, if one wished to eliminate the loss rate entirely and assume that essentially all the lead is recycled, (the amount lost as controlled emissions is very small), then the values in the "Total" column would be reduced to be equal to those in the "Installation" column.

The "Installation" column gives the flow rates of components and materials as parts of new batteries, into new or replacement batteries for EV use. Thus, 34.8 kg of new batteries must be installed every day for each MWh of existing installed capacity. The "Removal" column gives the rate at which the spent batteries are removed, in kg/day per MWh, after 3.3 years of use.

The calculations of the overall flow rates are not trivial, and details are given in Appendices A and B. In rough outline one needs to first calculate the battery flow rate into (new) use and the spent battery flow rate out of use. First, one has the requirement that the net flow rate into use must yield a 25% growth rate. Next, from the requirement that the battery use lifetime is 3.3 years, one has the result that at time t, the (unnormalized) removal rate of spent batteries must equal the (unnormalized) installation rate at time t - 3.3 years. This gives a pair of equations which can be solved iteratively to yield (unnormalized) installation and removal flow rates of whole batteries. When divided by the total installed capacity at a given point in time the whole battery installation and removal rates (34.8 kg/day·MWh, and 15.2 kg/day·MWh) are given (see App. A).

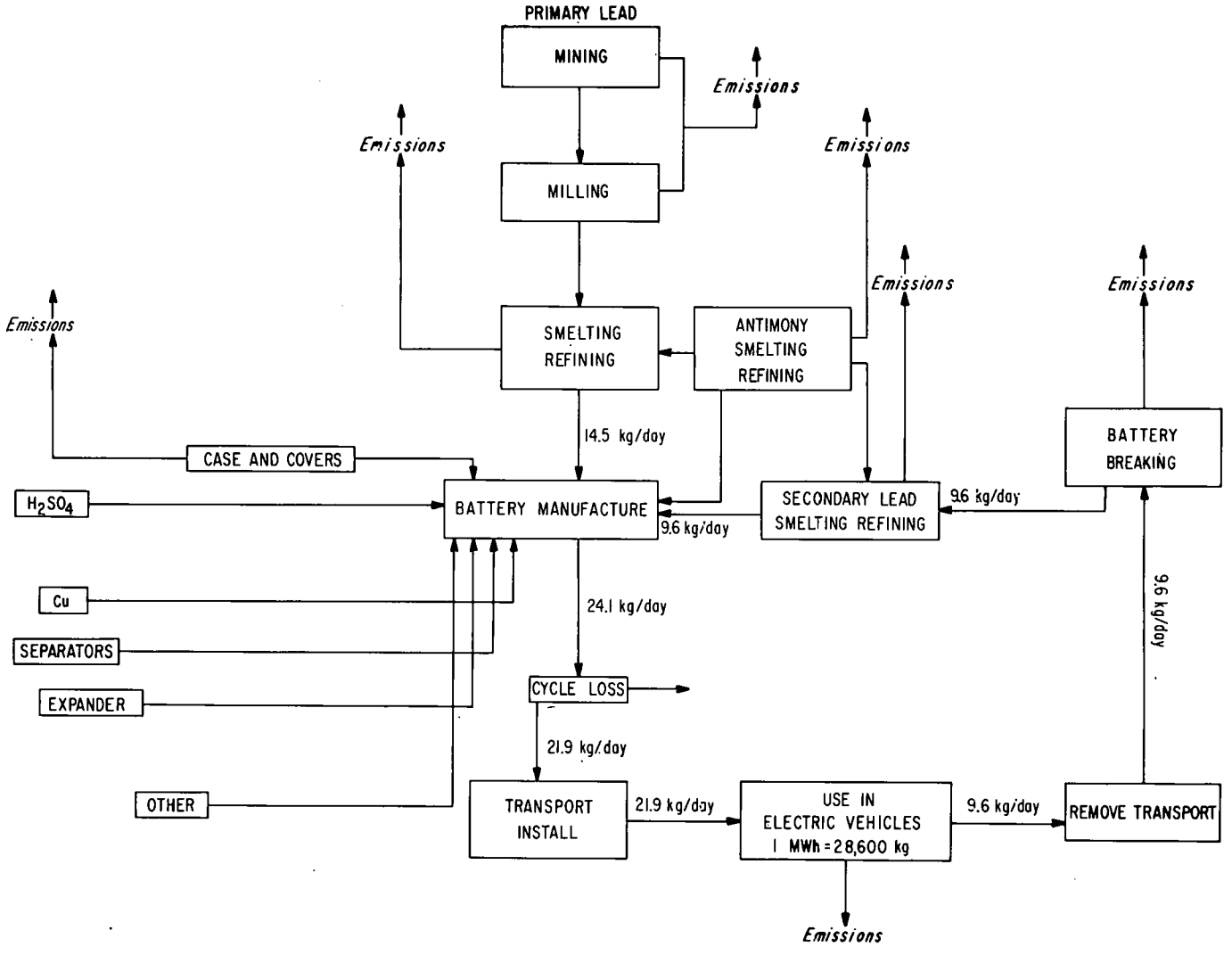

Fig. 2.1. The State-of-the-Art Lead/Acid Battery Cycle for Electric Vehicles.

Table 2.2. Battery Component Flow Rates through Cycle with a 3.3-yr Battery Lifetime and 25% Growth Rate Assumed (kg/day)/MWh[a]

Battery Component	New Batteries[b]			Removal of Spent Batteries[b,c]	
	Total	Loss	Installation		
Pb	24.1	2.2	21.9	9.6	
Sb	0.50	0.045	0.45	<0.20[d]	
As	0.0085	0.00077	0.0077	<0.0034[d]	
Cu	0.16	0.015	0.15	0.067	
Electrolyte	9.6	0.87	8.7	4.8	
H_2SO_4 (pure)	3.4	0.31	3.1	0.94[e]	0.43[f]
H_2O	6.2	0.56	5.6	<2.43[d]	
Case and Cover	2.5	0.23	2.3	1.00	
Separator	0.13	0.012	0.12	0.052	
Expander	0.053	0.0048	0.048	0.021	
Other	1.22	0.11	1.11	0.48[g]	
TOTALS					
Dry Weight	28.7	2.6	26.1	<11.4	
Battery	38.3	3.5	34.8	<15.2	

[a] *Based on new battery composition given in Table 2.1.*

[b] *Details of the calculations are given in Appendix A.*

[c] *Details of these calculations are given in Appendix B. It is assumed that spent batteries are removed from the EV in the fully discharged state.*

[d] *During charge-discharge cycling some of the As, Sb and H_2O is emitted as gaseous products. The values given are upper limit estimates, as they are calculated assuming no emission.*

[e] *The amount of H_2SO_4 bound as $PbSO_4$ in the discharged battery.*

[f] *The amount of H_2SO_4 remaining in the electrolyte in the discharged battery.*

[g] *During discharge, some of the oxygen in the positive active material is converted to water.*

The less-than symbol (<) before the Sb, As, H_2O and Total entries denotes the fact that during charge-discharge cycles, small amounts of Sb, As, and H_2O are lost in gaseous emissions. As a result these entries, which are calculated assuming no such losses, are upper limits and the actual values are slightly smaller. This will be discussed in more detail later.

To calculate the removal rates of materials and components in spent batteries one must account for the fact that the chemical composition of various components has changed. During discharge of a lead/acid battery, some of the active lead (and lead dioxide) and sulfuric acid are converted to lead sulfate and water. This is relevant to the cycle because lead and lead sulfate are smelted, which results in SO_2 emissions, whereas the remaining acid is neutralized to give $CaSO_4$. The two H_2SO_4 entries in the "Removal" column of Table 2.2 take this change into account. The left-hand entry, 0.94 (kg/day)/MWh, is the removal rate of that part of the original sulfuric acid that is bound to the lead as lead sulfate. The right-hand entry, 0.43 (kg/day)/MWh, is the part of the original sulfuric acid that remains in the electrolyte. Similarly the amount of water must be increased and the amount of "other" components must be decreased owing to conversion of the O_2 in PbO_2 into water. Details are given in Appendix B.

The flow rates in Figure 2.1 are given in terms of lead rather than whole batteries. Battery flow rates for much of Figure 2.1 would have to be given as upper limits, i.e., <15.2 (kg/day)/MWh. Definite numbers are preferable to upper limits, and lead is a principal battery component both in terms of the weight percent and environmental concern. Thus, lead flow rates are used in the figure.

It is important to clearly differentiate between the cycle loss and the difference between lead flow rates into and out of EV use as shown in Figure 2.1. The value of 24.1 - 21.9 = 2.2 kg/day lead represents the overall cycle loss of lead other than that accounted for by emissions. It includes such things as theft, nonrecoverable losses in accidents, and other unaccounted-for losses. The value of 21.9 - 9.6 = 12.3 kg lead/day net flow into the EV use box of Figure 2.1 represents the growth in the fleet size (25%/year) and the resultant increase in the rolling reserve. It does not represent a loss.

2.3 PRIMARY PRODUCTION OF INPUT MATERIALS

2.3.1 Lead

Mining and Milling

Lead ores are recovered by underground mining techniques. The ore-containing formations are blasted, with dynamite, ammonium nitrate-fuel oil, or slurry gels. The energy for drilling and transporting equipment is typically provided by diesel power. The exhausts from these and other various phases of mining are vented to the surface to maintain suitable air quality in the underground mining atmosphere.

An estimated 35,200 m^3 of mine air are exhausted from a "typical" Missouri lead mine per metric ton of lead mined. The particulate and lead concentrations of the exhaust air are 0.6 to 0.9 mg/m^3 and 10 to 15 $\mu g/m^3$, respectively. The gaseous components include[6] $\leq 0.1\%_V$ carbon dioxide; ~ 5 ppm_V carbon monoxide; $\leq$ few ppm_V oxides of nitrogen; ≤ 1 ppm_V oxides of sulfur; ~ 5 ppm hydrocarbons; ≤ 0.2 ppm_V aldehydes; ≤ 0.1 ppm_V organic acids; and ≤ 10 nCi/m^3 radon. The vented exhausts, although potentially hazardous, are not viewed as a major source of aboveground air contamination.[21]

Large quantities of waste rock are generated from the mining operations. The waste rock is used for mine backfilling, tailings dams and other construction, or deposited as waste in open dumps. This rock usually contains low levels (approaching the naturally occurring background levels) of potentially hazardous materials that are generally considered nonhazardous.[21] However, the potential for leaching, with subsequent mobilization of these materials, is increased with the fracturing of the waste rock, especially in acidic environments.

The milling of lead ores, which usually occurs at the mining site, involves crushing and grinding the ores and then separating out the lead and zinc minerals by flotation. The froth flotation procedures and reagents utilized are determined by the minerals to be isolated. If more than one mineral is to be isolated, differential flotation, achieved through different reagent utilization, is required. The floated lead and zinc mineral concentrates and other byproducts are thickened and then shipped to smelters. The mill tailings, gangue, excess mill reagents and water which contains suspended and dissolved solids that remain after isolation of

the target minerals, are transported to a tailings pond. Occasionally, the larger gangue is used for mine backfilling. Most of the tailings are considered potentially hazardous because the concentrations of potentially hazardous materials that they contain are above the naturally occurring background levels.[21] The water may contain fuel, oil, hydraulic fluid, blasting agents, and dissolved and suspended solids from the mining operation. It can be alkaline or acidic, depending on the nature of the ore mineralization and the local and regional geology of the mining area. Generally, alkaline waters have a low potential for solubilizing potentially hazardous materials, while acidic waters have a high potential.

Besides the wastewater from the flotation circuit, mine-mill water emissions include excess water from the mine, housecleaning water, overflow from the concentrate thickeners, and filtrate from the dewatered concentrate. Water separated from the concentrate is often recycled in the mill, but sometimes is pumped with the tailings to the tailings pond. Surface drainage from the mill area usually is diverted to the tailings pond treatment system.[21,22]

Air Emissions. Table 2.3 shows the air emissions expected from the mining and milling activity that would support an EV fleet using SOA lead/acid batteries at a growth rate of 25%/year. The table entries are derived from the entries of Table 2.2, which give the result that 24.1 - 9.6 = 14.5 kg/day per MWh of primary lead must be supplied as input to the manufacturing step. The metal concentrations in the mine exhaust particulates and fugitive dust particulates are assumed to be the same as from the crude ore.[21] The fugitive dust emissions arise during ore transportation and storage (2.0 kg/MT of ore handled) and ore grinding and crushing (1.0 kg/MT of ore handled).[23]

It can be seen that essentially all the air particulate emissions occur as fugitive dust emissions, and are quite large. However, most of these particles are relatively heavy and would be expected to settle out in the immediate neighborhood of the mine and mill.[23] A small fraction of the emissions which are smaller particles would settle out over a larger area.

Gaseous emissions[6] consist mainly of diesel exhaust gases and gases resulting from the use of explosives. The SO_x, hydrocarbon, aldehyde, and organic acid emissions have been calculated as equivalent amounts of SO_2, methane (CH_4), acetaldehyde (CH_3CHO), and acetic acid (CH_3COOH), respectively.

The principal methods of controlling fugitive emissions are good housekeeping and equipment maintenance. Truck loads are kept covered or wet; roads are paved, and the unloading area is wetted down. Storage piles are covered or wetted down, and crushing and grinding enclosures are maintained to prevent leaking.[23]

Liquid Emissions. Aqueous emissions (as flotation chemicals) expected from the primary lead mining and milling support activity for an EV fleet at a growth rate of 25%/yr and using SOA lead-acid batteries are shown in Table 2.4. The flotation chemicals--which are a complex mixture of activators, suppressors, frothers, etc.--are used to separate (by froth floating) the lead (and zinc) from the gangue. The water containing the residual flotation chemicals which flows out of the flotation circuit may or may not be recycled. The net outflow of these chemicals originates from non-recycled mill water and water adhering to the mill tailings.[24]

Table 2.3. Air Emissions from Lead Mining and Milling Activities for an SOA Lead/Acid Battery Powered EV Fleet[a]

Species	Concentration[b]	Emissions (kg/day/MWh)[c]		
		Mine Exhaust[d]	Fugitive[e]	Total
Particulates		3.1×10^{-4}	0.99	0.99
Pb	4.4%	1.3×10^{-5}	0.044	0.044
Cu	0.2%	6.2×10^{-7}	0.002	0.002
Zn	2.6%	8.1×10^{-6}	0.026	0.026
Fe	1.6%	5.0×10^{-6}	0.016	0.016
Co	0.012%	3.7×10^{-8}	0.00012	0.00012
Ni	0.017%	5.2×10^{-8}	0.00017	0.00017
Cd	0.012%	3.7×10^{-8}	0.00012	0.00012
Mn	0.16%	5.0×10^{-7}	0.0016	0.0016
Gases[f]				
CO	5	0.0032	---	0.0032
NO_x	$\leq$ few	---	---	---
SO_x	< 1	< 0.0014	---	< 0.0014
Hydrocarbons	5	0.0018	---	0.0018
Aldehydes	$\leq$ 0.2	$\leq 2.0 \times 10^{-4}$	---	$\leq 2.0 \times 10^{-4}$
Organic Acids	$\leq$ 0.1	$\leq 1.4 \times 10^{-4}$	---	$\leq 1.4 \times 10^{-4}$
Flotation Chemicals		---	---	Trace

[a] *Sources: Table 2.2 and the following references: J. O. Ledbetter, "Battery Energy Storage," in "Environmental Control Technology, R&D Requirements for Energy System," E. L. Kaufman, Los Alamos, N.M., Final Report No. LA-6979-Ms, September 1977. D. Bendersky, et al., "A Study of Waste Generation, Treatment and Disposal in the Metals Mining Industry," Midwest Research Institute, prepared under Contract No. 68-01-2665 for the U.S. Environmental Protection Agency, 1976. "Control Techniques for Lead Air Emissions." Vol. II, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, December 1977. Report No. EPA-450/2-77-012.*

[b] *The particulate concentrations represent the percent concentration in crude ore in a Missouri mine. (Bendersky et al., 1976.) The mine exhaust concentrations are given as ppm (volume) (Ledbetter, 1977).*

[c] *The metal values are calculated assuming that the metal concentrations in mine exhaust particulates and fugitive emissions from ore crushing and grinding and transportation are same as in the crude ore (Column 2).*

[d] *About 34,200 m^3 of air containing 0.6 mg/m^3 of particulates is exhausted per metric ton of lead mined (Ledbetter, 1977).*

[e] *Fugitive dust emissions from transport, storage, ore grinding, and ore crushing operations are 3.0 kg/MT of ore hauled. These particulates are heavy and do not travel far. "Control Techniques for Lead Air Emissions." Vol. II, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, December 1977. Report No. EPA-450/2-77-012.*

[f] *The mine exhaust gases listed are calculated as equivalent weights of NO_2, SO_2, CH_4, CH_3CHO, and CH_3COOH.*

Control technology consists of allowing the wastewater and mill tailings to settle in tailings ponds. Water from the mines may be added to the pond. The overflow from the ponds may go through other ponds before final discharge, and may have residence times of one to two months in the ponds.[24]

Table 2.4. Flotation Chemical Emissions from Primary Lead Mining and Milling[a]

Flotation Chemicals	*Emissions (kg/day)/xMWh*	
	Buick Mine[c]	*Fletcher Mine*[d]
Organics[b]	6.4×10^{-4}	3.2×10^{-4}
Long-chain Alphatic Alcohols	5.3×10^{-4}	2.3×10^{-4}
Propylene Glycol Methyl Ether	2.9×10^{-5}	2.3×10^{-5}
Isopropyl Ethyl Thionocarbonate	8.8×10^{-5}	6.8×10^{-5}
Other[e]	5.8×10^{-3}	4.7×10^{-3}
Sodium Diethyl Dithiophosphate	2.6×10^{-4}	--
Sodium Ethyl Xanthate	4.7×10^{-5}	3.2×10^{-4}
$ZnSO_4$	2.4×10^{-3}	2.7×10^{-3}
NaCN	2.9×10^{-5}	6.8×10^{-6}
$CuSO_4$	2.9×10^{-4}	4.6×10^{-4}
Sodium Sulfosuccinate	4.1×10^{-4}	4.7×10^{-4}
Starch	--	3.2×10^{-4}
SO_2	--	3.2×10^{-4}
Sodium Dichromate	--	9.5×10^{-5}
NaOH	--	1.1×10^{-4}
$Ca(OH)_2$	2.4×10^{-3}	--

[a] *Source: Ref. 24 and Table 2.2.*

[b] *Organics are those chemicals extractable by benzene or chloroform.*

[c] *The Buick mine and mill outputs from the final sedimentation pond into a creek total 17.4 kg organics/day. In 1973 the mill produced 204,000 MT of lead concentrate averaging 80% lead.*

[d] *The Fletcher mine and mill outputs from the final pond into a creek total 8.9 kg organics/day. It produces 4500 MT of ore daily. It is assumed here that lead content of the ore is the same as that (~9.5%) of the Buick mine.*

[e] *The entries assume (1) no fraction between the organic and non-organic emissions and (2) the same fractional composition in the effluent stream as exists in the chemical dosage.*

The numbers given in the table represent emissions calculated from the primary lead flow of 14.5 kg/day per MWh and from the data of Jennet and Callier[24] for two Missouri lead mines. These authors measured the net amount of organic materials discharged by the mines to the creeks. These data, plus a knowledge of the dosage of each flotation chemical[24] (kg/MT of ore), together with the assumption that the fraction of each chemical emitted in the final effluent was the same for each, made it possible to calculate the emission rates given in Table 2.4 for the two mines.

In addition to the flotation chemicals, the mine and mill wastewaters contain metals and suspended solids. Table 2.5 shows the concentrations before and after treatment and calculated emission rates for a large Missouri lead mine-mill complex.[22] The latter were obtained using a conversion factor of 14.5 kg/day per MWh of primary lead. The raw waste effluent from the mill consists of a 25% slurry of tailings. The treatment consists of passing the slurry through a tailings pond, a stilling pond, stream meanders, a polishing pond, and more meanders before final discharge.[22] The data indicate that the treatment removes most of the suspended solids,

Table 2.5. Water Emissions from Lead Mining and Milling Activities for a Representative Missouri Mine/Mill Complex

Parameters	Concentration (mg/L)[a] Raw	Treated[b]	Emissions (kg/day·MWh)[c,d,e]
TSS	2.5×10^5	8	0.018
Cyanide	0.03	< 0.01	$< 2.3 \times 10^{-5}$
Pb	1.9	< 0.1	$< 2.3 \times 10^{-4}$
Cd	0.005	0.005	1.1×10^{-5}
Cu	< 0.02	< 0.02	$< 4.6 \times 10^{-5}$
Hg	$<10^{-4}$	$<10^{-4}$	$< 2.3 \times 10^{-7}$
Zn	0.46	0.04	9.0×10^{-5}
Mn	0.08	0.16	3.6×10^{-4}
Cr	< 0.02	< 0.02	$< 4.6 \times 10^{-5}$
Fe	0.53	0.13	2.9×10^{-4}

[a] *Modified from Table VII-17 of EPA 440/1-75/061. In 1973 the mill processed 1.45×10^4 MT of ore containing 5.5% Pb; 34,100 m^3/day of wastewater were discharged (including rain runoff).*

[b] *Treatment consists of passing the mine/mill discharge through a tailings pond, a stilling pond, and stream meanders, a polishing pond, and more stream meanders (see text).*

[c] *Calculations use the fact that in 1974, 8.2×10^6 MT of Missouri ore produced 6.4×10^5 MT of lead concentrate (Bendersky, 1976) containing 70% lead (Jennet and Callier, 1977).*

[d] *Conversion factor:* $\frac{34{,}100 \times 365}{1{,}450{,}000 \times 0.055} = 156 \frac{kg\ H_2O\ discharge}{kg\ Pb\ in\ ore}$, *and Table 2.2.*

[e] *These are emissions after treatment.*

Pb, and Zn, but is not successful in removing Cd, Cu, Hg and Mn. However, these metals are initially present in low concentrations in the raw wastewater.

The recommended 30-day average effluent limitations for TSS, cyanide, Pb, Cd, Cu, Hg and Zn in mg/L, using best practicable control technology currently available are 20 mg/L TSS, 0.01 mg/L cyanide, 0.2 mg/L Pb, 0.05 mg/L Cu, 0.001 mg/L Hg and 0.2 mg/L Zn. In general, these values are higher than the values given in Column 3 of Table 2.5. One reason is that Idaho mine effluents are included as well as smaller mine mills with simpler treatment systems. On the other hand, the mill effluent limitations recommended for the best available technology economically achievablable are zero, because of an assumed zero water discharge.[22] This seems difficult to achieve in a geographical area of heavy rainfall such as Missouri, where most U.S. lead is mined.

Solid Emissions. The solid emissions from primary lead mining and milling consist of waste rock from the mining and tailings sludge from the milling operation. In 1974, Missouri produced 639,000 MT of mill lead concentrate, 1.3×10^6 MT of waste rock and 7.3×10^6 MT (dry weight) of mill tailings.[21] Assuming the concentrate averages 70% lead, this works out to 3.0 MT of waste rock and 16 MT (dry weight) of mill tailings per metric ton of lead.

Table 2.6 shows the solid emissions to be expected from lead mining and milling activity to support the model EV fleet. The tailings composition entries (column two) are for a Missouri mine.[21] As before the flow rate of 14.5 kg/day per MWh of primary lead (Table 2.2) is needed as a conversion factor. The entries show that a large quantity, 232 kg dry weight of tailings, are produced each day for each MWh of electric capacity in EV batteries. The metals content of the material, which is produced as a sludge, is seen to be appreciable. The tailings sludge that settles out in ponds must ultimately be disposed of in landfills or put back in old mines.

Table 2.6. Solid Emissions from Lead Mining and Milling in Support of an EV Fleet

Material[a]	*Composition*[b] %	*Emissions (kg/day)MWh*[c]
Waste Rock	--	44.
Mill Tailings, dry wt	--	232.
Pb	0.093	0.22
Cu	0.018	0.042
Zn	0.083	0.19
Fe	1.3	3.0
Co	0.0045	0.010
Ni	0.0063	0.015
Cd	0.0016	0.0037
Mn	0.18	0.42

[a] *Assumes 3 MT of waste rock and 16 MT of tailings produced per ton of lead (see text).*

[b] *Source: D. Bendersky et al., "A Study of Waste Generation, Treatment and Disposal in the Metals Mining Industry," Midwest Research Institute, prepared under Contract No. 68-01-2665 for the U.S. Environmental Protection Agency, 1976.*

[c] *Scaling factor (Table 2.2): 14.5(kg/day)/MWh primary lead required.*

Impact on Existing Industry. Table 2.7 gives the impact that a large fleet of SOA lead/acid EVs, growing at 25%/year, would have on the existing primary lead mining and milling industry. Columns 2 and 3 give the EV fleet size, in thousands of vehicles and MWh battery capacity for the years 1990, 1995, and 2000 for a scenario which projects 3×10^6 lead/acid EVs in the year 2000.[1]

The next three columns give the primary lead output, in terms of metric tons per day, percent of capacity existing in 1974, and the number of large new mine-mill complexes which are needed to support such an EV fleet.

Table 2.7. Impact of EV Fleet on Existing Primary Lead Mining and Milling[a]

Year	Fleet Size: Vehicles, thousands	Fleet Size: MWh	Lead Output[b]: MT/day	Lead Output[b]: % of Existing Capacity (1974)	Lead Output[b]: No. of New Mine-Mill Complexes Required	Air Emissions, kg/day: Particulates: Total	Pb	Cu	Zn	Fe	Co	Ni	Cd	Mn	Gases: CO	SO_x	Hydrocarbons	Aldehydes	Organic Acids
1990	250	6,200	90	5.4	0.5	6100	260	12	160	99	0.7	1.0	0.7	9.9	20	<8.7	11	<1.2	<1.1
1995	870	22,000	320	19.	1.8	2.2×10^4	970	44	570	350	2.6	3.7	2.6	35.	70	<30.	40	<4.8	<3.1
2000	3000	75,000	1100	66.	6.1	7.4×10^4	3300	150	1900	1200	9.	13.	9.	120.	240	<100.	140	<15.	<10.

Year	Fleet Size: Vehicles, thousands	Fleet Size: MWh	Water Emissions, kg/day: Flotation Chemicals[c]: Organic	Flotation Chemicals[c]: Other	TSS	Cyanide	Pb	Cd	Cu	Hg	Zn	Mn	Cr	Fe
1990	250	6,200	4.0	35.	112	<0.14	<1.4	0.068	<0.28	<0.0014	0.56	2.2	<0.28	1.8
1995	870	22,000	14.	130.	400	<0.51	<5.1	0.24	<1.0	<0.0051	2.0	7.9	<1.0	6.4
2000	3000	75,000	48.	440.	1300	<1.8	<18.	0.82	<3.4	<0.018	6.7	27.	<3.4	22

Year	Fleet Size: Vehicles, thousands	Fleet Size: MWh	Solid Emissions, MT/day: Waste Rock	Mill Tailings: Total	Pb	Cu	Zn	Fe	Co	Ni	Cd	Mn
1990	250	6,200	270	1,400	1.4	0.26	1.2	19	0.062	0.09	0.023	2.6
1995	870	22,000	960	5,100	4.8	0.92	4.2	73	0.22	0.33	0.081	9.2
2000	3000	75,000	3300	17,000	16.	3.1	14.	220	0.8	1.1	0.28	32.

[a] *Sources: Tables 2.2, 2.3, 2.4, 2.5, 2.6 and included footnotes, Section 2.2.*

[b] *Total U.S. primary production of lead in lead ore in 1974 was 1660 MT/day. The production rate of a large mine-mill complex is equivalent to 180 MT of lead produced/day, Minerals Yearbook, 1974, Vol. I, Metals Minerals and Fuels, Bureau of Mines, U.S. Dept. of Interior, 1976.*

[c] *These are values for a large Missouri mine. For a further breakdown see entries of Table 2.4.*

The next 36 columns give the air emissions in kg/day, the water emissions in kg/day, and solid emissions in MT/day resulting from the above primary lead industrial support. For example, a fleet of 3×10^6 SOA lead/acid EVs, growing at 25%/year and which is projected to exist in the year 2000, will require an increased primary lead input to battery manufacturing of 1100 MT/day. This corresponds to a 66% increase of present day (1974) capacity, or 6.1 large new mine-mill complexes would be needed. This primary mining and milling activity would result in 3300 kg/day lead in particles discharged to the atmosphere. Seventeen thousand metric tons of mill tailings containing 16 MT of lead, 3.1 MT of copper, etc., would also be produced each day.

The table entries show that particularly by the year 2000 the impact on the existing mining and milling industry is considerable and there are large amounts of heavy metals emitted as atmospheric particulates. Most of these originate as fugitive dusts which are not expected to travel far from the mine-mill complex. Even larger amounts of heavy metals are present in the mill tailings sludge. As noted above this material is potentially hazardous and, thus, must be disposed of in secured landfills.

Although the amounts of heavy metals emitted as effluents are considerable, they must be kept in perspective. Even by the year 2000 the amounts are about 2/3 of the amounts presently being emitted. Also one would expect pollution control technology to advance so that the pollutant emission levels could be reduced below the amounts given in Table 2.7.

Smelting and Refining

The lead ore concentrate produced by the mining and milling operations is smelted and refined to obtain pure lead and to separate out in pure form, other metallic elements associated with the lead ore. In the smelting operation, ore concentrate is fed to a sintering furnace, together with limestone and silica fluxes and residues, to remove most of the sulfur (as SO_2 and SO_3) and the more volatile impurities such as arsenic, antimony and cadmium.[25]

The sinter produced in the sintering furnace operation is fed, with coke, into the blast furnace, to produce lead bullion and large amounts of slag. The slag is partially recycled to the sintering furnace and partly sent to the slag-granulation operation and then to the slag dump. The lead bullion is passed through drossing kettles to remove most of the copper and is then sent to the lead refinery where the various metal impurities such as antimony, arsenic, zinc, silver, tin, bismuth, cadmium and other metals are removed[23,25,26] and often purified. The dross, which contains from 9% to 30% of the original lead, is further treated in a dross reverberatory furnace. Flue dusts from the blast furnace are sent to a cadmium plant.[23,26]

The flow diagram of the various waste streams in a primary lead smelter is fairly complex and can include much recycling. The SO_2 flue gases from the front of the sinter machine, which contain 6% to 7% SO_2, are sent through wet scrubbers and baghouses, and, in three of the seven smelters in operation the cleaned SO_2 gas stream is fed into an onsite sulfuric acid plant. The gas from the back of the sinter, which contains about 0.5% SO_2, is either vented or recycled through the sinter machine.[23,25] Gases from the blast and dross reverberatory furnaces are passed through baghouse filters before being vented to the atmosphere. The dry dusts removed from the baghouse filters are recycled to the sintering furnace. Dust slurries obtained from the scrubbers and the acid plant are allowed to settle in pits or lagoons, and are then dredged,

and deposited in piles on land or in a slag dump. In some plants, after the sludge has dried out over a period of several months, it is recycled to the sintering furnace.

These dust sludges (about 89 kg sludge/MT of lead) contain large amounts of lead (14.3%), zinc (8.0%), cadmium (0.7%), copper (0.6%), antimony (0.09%), mercury (0.02%), manganese (0.09%), and chromium (0.003%) and are potentially hazardous.[26] Leaching of the materials into the ground can occur either during settling (if the settling ponds are not lined or drying (during the months the dredged piles dry), or, if the material is discarded in the slag dump. One plant immediately recycles the sludges from the acid plant and sintering furnace, to avoid land storage or disposal problems. Control and disposal methods currently in use need improvement.[26]

The blast furnace slag not recycled to the sinter (about 30%) is, in some plants, passed through a slag fuming furnace to remove zinc and some residual lead. The slag (whether fumed or not) is then granulated by quenching in water, and piled on a slag dump. The water from this operation passes in succession into two settling ponds, which are periodically dredged. The dredgings are added to the slag dump.

Process wastewater from the wet scrubbers and slag granulation contains small amounts of lead, cadmium, and other metals. In some plants, very little or none of this water is discharged into the environment; in others it is being discharged (as of 1974-1975). Some of the water flow circuits are being revised in order to meet the guidelines for the best practicable control technology currently available, especially in areas of heavy rainfall, which is where most of the lead is mined and smelted.[25]

Air Emissions. It will be assumed for this discussion of air emissions, that by the 1990s all primary lead smelters will incorporate sulfuric acid plants. As a result, the only air emissions will be from the blast furnace, the dross reverberatory furnace and fugitive dusts. The air emissions from the sintering furnace, which are fed into the sulfuric acid plant, will be discussed separately under the primary production of sulfuric acid.

Table 2.8 gives the air emission flow rates, in kg/day per MWh, from the primary lead smelting and refining operations under these assumptions. For the blast and reverberatory furnaces, the respective uncontrolled dust and lead emission factors are taken to be 125 kg/MT containing 40% lead, and 10 kg/MT containing 35% lead.[23] Analyses of fugitive dust emissions from different plants are extremely variable both as to amounts of dust and lead content; values for total dust emission range from 0.72 kg/MT to 22.4 kg/MT,[23] with lead concentrations ranging from 3% to 58%. A fugitive dust emission factor of 2.5 kg/MT and a rough weighted average of 25% lead concentration are used for this discussion.[3]

The SO_2 emission flow rates in Table 2.8 have been obtained by converting the published volume concentrations of SO_2 in the furnace flue gases by using the published flow rates and temperatures of the furnace flue gases.[23]

Uncontrolled emissions originate principally from the blast furnace, and much of the controlled particulate and lead emissions arise from fugitive dusts (Table 2.8). The reason is that fugitive dusts are, in essence, not controlled. As a result, control of furnace emissions reduces them to levels which are lower than those for the fugitive dusts. The amount of fugitive dust

Table 2.8. Air Emissions from Primary Lead Smelting and Refining

	Emissions (kg/day)/MWh[a]	
	Uncontrolled	*Controlled*
Blast Furnace[b]		
Particulates	1.8	0.018
Pb	0.72	0.0072
SO_2[c]	0.0012	0.0012
CO	appreciable	
Reverberatory Furnace[d]		
Particulates	0.14	0.0014
Pb	0.051	0.00051
SO_2[e]	8.9×10^{-5}	8.9×10^{-5}
CO		
Fugitive Dust[f]		
Particulates	0.036	0.036
Pb	0.009	0.009
TOTAL		
Particulates	1.98	0.055
Pb	0.78	0.017
SO_2	0.0013	0.0013
CO	appreciable	

[a] *Modified from "Control Techniques for Lead Air Emissions," Vol. II, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, Report No. EPA-450/2-77-012, December 1977. Also Ref. 3.*

[b] *Assumes 125 kg particulates/MT containing 40% Pb with 99% control efficiency.*

[c] *Conversion factor (EPA-450/2-77-012):*

$$\frac{0.63\ m^3/min}{MT/hr} \times \frac{60\ min}{hr} \times \frac{1000\ L}{m^3} \times \frac{.273^\circ\ moles}{22.4 \times 873^\circ L} \times$$

$$0.0025\ Vol.\ Fraction\ SO_2 = \frac{1.31\ moles\ SO_2}{MT\ product}$$

[d] *Assumes 10 kg particulates/MT containing 35% Pb with 99% control efficiency.*

[e] *Conversion factor (EPA-450/2-77-012):*

$$\frac{0.31\ m^3/min}{MT/hr} \times \frac{60\ min}{hr} \times \frac{1000\ L}{m^3} \times \frac{273^\circ\ moles}{22.4 \times 873^\circ L} \times$$

$$5 \times 10^{-4} = \frac{0.0985\ moles\ SO_2}{MT\ product}$$

[f] *Assumes 2.5 kg/MT containing 25% Pb with no control (see text).*

released will depend on good housekeeping practices such as wetting the ore, ore-handling areas and waste piles, and covering trucks during transport of materials. Appreciable fugitive emissions also originate from the sintering and blast furnace operations.[23]

The SO_2 emissions from blast furnaces are at present uncontrolled as they constitute a fairly small amount (< 0.05% by volume) of the flue gas. The values given in Table 2.8 are calculated assuming no control. However, it may be that with increasing concern for sulfur oxide emissions, the emission regulations will require this source to be controlled.

Liquid Emissions. The process wastewater from primary smelters, even after liming and/or settling, contains appreciable amounts of lead and cadmium. As a result, some smelter operators are rearranging (as of 1974-75) their water circuits to recycle wastewater, with the object of reducing or eliminating wastewater emissions. (In areas of high net rainfall where many of the smelters are located, it will probably be difficult to completely eliminate all process wastewater emissions.) These changes make it difficult to predict wastewater emissions from smelters operative in the 1990s. Consequently, it seems appropriate to calculate the emission flow rates using as inputs the recommended effluent limitations for primary lead smelters to be achieved by 1 July 1977, using the best practicable control technology currently available.[25]

The values for total suspended solids, lead, cadmium, and zinc given in Table 2.9 are calculated from the above considerations. No effluent limitations are given for the other entries (arsenic, nickel or sulfates) because the data are either insufficient (As), the concentration limit (Ni) is controlled by limits set forth for other parameters, or the species ($SO_4^=$) is not considered toxic. The arsenic emissions have been calculated from measured effluent concentrations in the wastewater from the non-contact cooling and treated acid-plant blowdown operations of one plant. The nickel and sulfate ion emissions are calculated from the values given as "effluent from projected treatment plant with revised water circuits" for one plant.[25]

The water emissions flow rates (Table 2.9) for total suspended solids Pb, Cd, Zn, As and Ni are quite small. The sulfate flow rate is somewhat larger. However, sulfate is not considered to be a very toxic material, and as a result, effluent limitations or regulations either do not exist or are weak.

Solid Emissions. Table 2.10 gives the solid waste emissions flow rates for primary lead smelting and refining. Column 2 indicates that portion of the total solid waste which is temporarily or permanently stored in piles or in lagoons (dry weight); Column 3 gives that part of the solid waste which is permanently placed in lagoons or in dumps. Those portions of the waste (such as dry baghouse dust, and some of the slag and slurry) that are immediately recycled are not included in the table.

To estimate the amount of sludge and metal contaminants that are not finally recycled is difficult, as recycling practices vary among the plants. Also, sludge materials may be subject to significant leaching, as they are rather easily solubilized; and it is not known how much dry sludge is lost during temporary storage or how much of the metal content is lost through leaching of the piles or percolation into the ground by lagoon waters. Therefore, the sludge entries in the third column of the table have been left blank. The metal contaminants in the slag are not considered hazardous, as they are not easily solubilized.[26,27]

Table 2.9. Water Emissions from Primary Lead Smelters[a]

Species	Emissions	
	Limitations (kg/MT)[b]	Flow Rate (kg/day)/MWh
TSS	0.021	3.0×10^{-4}
Pb	0.0004	5.8×10^{-6}
Cd	0.0004	5.8×10^{-6}
Zn	0.004	5.8×10^{-5}
As[c,d]	--	1.8×10^{-7}
Ni[c,e]	--	2.0×10^{-6}
Sulfates (As $SO_4^=$)[e]	--	0.0060

[a] *Source: "Development Document for Interim Final Effluent Limitations Guidelines and Proposed New Source Performance Standards for the Lead Segment of the Nonferrous Metals Manufacturing Point Source Category," U.S. Environmental Protection Agency, Effluent Guidelines Division, Report No. EPA-440/1-75/032, February 1975.*

[b] *These are the proposed emission limitations to be achieved by 7/1/77 under the best practicable control technology currently available for smelters located in areas of net rainfall. The values given are the 30-day averages.*

[c] *No effluent limits (best practicable control technology currently available) are proposed, either because the data are insufficient (As) or the concentrations are controlled (Ni) by limits set for other parameters.*

[d] *The arsenic flow rate was calculated from the data available for one plant which outputs 1.2×10^{-5} kg As/MT lead product in the non-contact cooling waters and treated acid plant blowdown. Source: EPA-440/1-75/032, p. 43.*

[e] *The respective Ni and sulfate ion flow rates of 1.4×10^{-4} kg Ni/MT lead product and 0.42 kg $SO_4^=$/MT lead product, which were used to compute the entries in the table, are values given for one plant as "Effluent from projected treatment plant with revised water circuits." Source: EPA-440/1-75/032, p. 42.*

In view of the possible solubilizing of the sludge contaminants, and the increasing acidity of rainfall, it is likely that increased amounts of metals may be leached from the solid wastes. This may well necessitate revision of the storage and dumping operations at the primary lead smelters.

Impact on Existing Industry. The impact on primary lead smelting of a large fleet of EVs growing at 25% per year will be considerable. Table 2.11 shows the relevant amounts projected to the year 2000 for the scenario given in Section 2.1. In the 1990s, the impact of an EV fleet on the existing primary smelting industry becomes appreciable; by the year 2000, 55% of the existing capacity must be added, which is equivalent to 4 new smelters with a capacity of 100,000 MT/yr each.

Considerable amounts of some airborne effluents will be produced. Missouri, where most of the primary lead is mined, will be the state most affected. For example, in the year 1995, an increase of 1200 kg of particulates containing 370 kg of lead will be emitted into the air each day as controlled emissions. In the same year, 28 kg/day of SO_2 will be emitted. By the year 2000, these increases will rise to 4100 kg particulates containing 1300 kg lead, and to 98 kg/day of SO_2.

Table 2.10. Solid Emissions from Primary Lead Smelting and Refining[a]

Material	Solid Waste Flow Rates, (kg/day)/MWh	
	Temporary Plus Permanent[b] Land or Lagoon Storage	To Environment[c]
Slag[d]	6.4	6.4
Pb	0.20	0.20
Cd	1.1×10^{-4}	1.1×10^{-4}
Cr	1.1×10^{-4}	1.1×10^{-4}
Cu	1.1×10^{-3}	1.1×10^{-3}
Mn	6.9×10^{-4}	6.9×10^{-4}
Sb	3.8×10^{-5}	3.8×10^{-5}
Zn	0.46	0.46
Sludge[e]	1.2	--
Pb	0.16	--
Cd	0.0079	--
Cr	3.1×10^{-5}	--
Cu	0.0067	--
Mn	0.0011	--
Sb	0.0011	--
Zn	0.092	--
Hg	2.1×10^{-4}	--

[a] *Source: "Assessment of Industrial Hazardous Waste Practices in the Metal Smelting and Refining Industry. Volume II, Primary and Secondary Nonferrous Smelting and Refining," Calspan Corporation, Buffalo, New York, U.S. Department of Commerce Report No. PB-276-170, April 1977. "Assessment of Industrial Hazardous Waste Practices in the Metal Smelting and Refining Industry," Volume IV, Appendices, Calspan Corporation, Buffalo, N.Y., U.S. Department of Commerce Report No. PB 276-172, April 1977.*

[b] *This material is either permanently disposed of in lagoons or in dumps, or is stored in a disposal area for some time (months) before recycling. An unknown fraction is dispersed to the environment by leaching, percolation or blowing.*

[c] *This material is permanently disposed of in lagoons or in dumps.*

[d] *Relevant slag production rate = 440 kg/MT lead.*

[e] *Relevant sludge (dry weight) production rate = 79 kg/MT lead.*

Table 2.11. Impact of EV Fleet on Existing Primary Lead Smelting amd Refining[a]

	Fleet Size		Lead Output			Air and Water Effluents Generated									
						Controlled Air Emissions, kg/day			Water Emissions, kg/day						
Year	Vehicles (Thousands)	MWh	MT/day	% of Existing Capacity	Number of New Smelters Required[b]	Particulates	Pb	SO_2	TSS[c]	Pb[c]	Cd[c]	Zn[c]	As[d]	Ni[d]	$SO_4^{=}$[d]
1990	250	5,200	90	4.5	0.33	350	110	8.1	1.8	0.037	0.037	0.37	1.1×10^{-3}	0.013	36
1995	870	22,000	320	16.	1.2	1200	370	28.	6.5	0.12	0.12	1.2	0.0038	0.045	130
2000	3000	75,000	1100	55	4.0	4100	1300	98	23.	0.43	0.43	4.3	0.014	0.16	450

	Fleet Size		Solid Wastes Generated							
				Metals in Slag, kg/day[e]						
Year	Vehicles (Thousands)	MWh	Slag[e], MT/day	Pb	Cd	Cr	Cu	Mn	Sb	Zn
1990	250	6,200	40	1,200	0.68	0.68	6.8	4.3	0.23	2,800
1995	870	22,000	130	4,300	2.3	2.3	23.	15.	0.81	10,000
2000	3000	75,000	480	15,000	8.1	8.1	81.	51.	2.8	35,000

	Fleet Size									
				Metals in Sludge, kg/day[f]						
Year	Vehicles (Thousands	MWh	Sludge[f] MT/day	Pb	Cd	Cr	Cu	Mn	Sb	Zn	Ag
1990	250	6,200	7.2	1,000	48	0.20	42	6.8	6.8	580	1.3
1995	870	22,000	25.	3,500	170	0.66	140	23.	23.	2000	4.5
2000	3000	75,000	87.	12,000	600	2.3	500	75.	75.	6800	16

[a] *Sources, Tables 2.2, 2.8, 2.9, 2.10 and Section 2.2. The values are given to two significant figures.*

[b] *At an average output of 100,000 MT of lead/yr (see text).*

[c] *Calculated from emission limits for best practicable control technology available to be achieved by 7/1/77 (see footnote b in Table 2.9).*

[d] *Calculated from data available for one plant (see footnotes c through e in Table 2.9).*

[e] *The slag is disposed of in dumps. The metals solubilize to the order of 1 ppm in leaching studies (see text).*

[f] *The sludge is temporarily stored in lagoons or dumps before recycling, and is recycled immediately, or not recycled, depending on the smelter. The metals solubilize appreciably so the sludge is potentially hazardous. (See text.)*

The water emissions for total suspended solids, lead, cadmium and zinc correspond to the emission limits to be achieved by 1 July 1977 using the best practicable control technology currently available for smelters located in areas of high net rainfall.[25] The values for arsenic are obtained from effluent flow rates given for one plant; the values for nickel and $SO_4^=$ are obtained from effluent flow rates which are projected to be valid after revision of the plant's water circuits.[25]

Large amounts of solid wastes would also be generated. In 1995, for example, 130 MT of slag containing 4300 kg of lead and 2.3 kg of cadmium will be generated and dumped each day. By the year 2000, these numbers will have risen to 480 MT of slag containing 15,000 kg lead, and 8.1 kg cadmium. Because the metals in the slag do not solubilize appreciably during leaching ($\sim$ 1 ppm)[26,27] these materials are probably not potentially hazardous.

The situation is rather different for the sludge. In 1995, 25 MT of sludge containing 3500 kg lead, 170 kg cadmium, 23 kg antimony, and 4.5 kg silver would be produced each day. By the year 2000, these values will rise to 87 MT, 12,000 kg, 600 kg, 75 kg, and 16 kg, respectively. This sludge is treated differently in different plants. In some, it is recycled immediately; in others, some or all of it is not recycled at all. This sludge must be considered potentially hazardous as the metals content is significantly solubilized by leaching.[26,27]

The values given above, although large, must be placed in perspective. Even by the year 2000, the total waste emissions will be only about one-half larger than those emitted at the present time, as the total smelting increase will be 55% (Column 5 of Table 2.11). Furthermore, the values given in the table assume there will be no changes in the waste control and treatment technology, although many changes in this technology are likely to occur.

2.3.2 Antimony and Arsenic

At present, both antimony and arsenic are used as alloying elements in the grid metal of the lead/acid battery. For the SOA lead/acid battery, the grid metal concentrations are taken to be 4.3% Sb and 0.08% As in the negative grid, and 4.6% Sb and 0.08% As in the positive grid.[10] These values give overall percent ratios of antimony to lead and arsenic to lead of 2.06% and 0.035%. (The active materials are free of antimony and arsenic.)

It will not be necessary to consider arsenic production for EV use in a separate section in this report. The amount of arsenic required is very small (0.035% of the lead). Some, and possibly all, of the arsenic needed is or can be recovered as a byproduct of lead smelting. Thus, most of the emissions associated with arsenic production (other than the alloying operation) are already included in the lead production values. At present, however, most arsenic is produced as a byproduct of copper smelting.[28]

Of the total amount of antimony required to make battery lead, only a small fraction arises as a byproduct of primary lead refining. In 1974, out of the total U.S. primary production of 604,000 MT of lead, only 11,400 MT was produced as antimonial lead containing 8.8% Sb.[29] This corresponds to 48,500 MT containing 2.06% Sb, which is only 8% of the total primary lead production. The lead in the SOA lead/acid battery considered here contains 2.06% antimony on the average (Table 2.1); thus, 92% of the primary lead used in battery manufacture (this assumes

that the fraction of the antimonial lead discussed above, and primary lead that go into battery manufacture are the same) must be alloyed to 2.06% Sb by use of antimony from sources other than from lead mining.

In 1973, 91% of total input to antimony smelters in this country was from foreign ores and only 9% was from domestic ores. Thus, the mining and milling of antimony will not be discussed, as the environmental impact is outside the U.S. Only the smelting of antimony will be considered.

About 80% of the total antimony smelted in the U.S. is produced by blast-furnace smelting at a plant in Texas; most of the remainder is produced electrolytically by plants in Idaho. The charge to the blast furnace consists of mixed oxides and sulfides, mattes, slags, flue dusts and residues from lead/zinc operations, as well as charcoal and fluxes. The impure antimony so obtained is further refined in a reverberatory furnace. Dust and gas emissions from the furnaces are passed through baghouses before being vented to the atmosphere. Large amounts of slag are produced (2.8 MT/MT Sb produced) and piled in open dumps.[26]

The electrolytic process begins by leaching the antimony ore with sodium sulfide (Na_2S) to dissolve the antimony as sodium thioantimoniate (Na_3SbS_4). The solution is clarified by settling and filtration and is electrolyzed to produce pure antimony. About 13 m^3 of spent solution containing 210 kg of solids is produced per metric ton of antimony. This is disposed of into a tailings pond that also receives wastes from mining and milling operations occurring at the site.[26]

Emissions

No data seem to be available on air or water emissions from antimony smelting. However, solids emission data are available[9] and have been used to generate the values given in Table 2.12. The total amount of antimony required, in kg/day per MWh, is computed as 92% of 2.06% of the amount of primary lead (14.5 kg/day/MWh) required (see Table 2.12). It is assumed that 80% of the antimony is produced pyrometallurgically and the remainder is produced electrolytically.[26] Any amounts of antimony which are needed to replenish the antimony in the battery lead recycled through the secondary smelting operation were ignored in the generation of the values in Table 2.12. The reason is that such amounts are expected to be quite small.

It is clear from the table that most of the waste emissions are quite small except for the slag and its antimony content, and the sludge. A leaching study on the slag has shown that significant amounts of antimony as well as some arsenic and copper are leached out; so, unlike the case for lead, the antimony slag must be considered potentially hazardous.[26,27] The electrolytic sludge also showed some solubilization in leaching studies; as a result, it should be considered potentially hazardous also.[26,27]

Impact on Existing Industry

Table 2.13 is a projection of the impact that a fleet of EVs, growing at 25% per year to give 3×10^6 SOA lead/acid Evs in the year 2000, has on the existing antimony smelting and refining industry. Only some of the metals given in Table 2.12 are included.

Table 2.12. Solid Emissions from Antimony Smelting and Refining[a]

Material	*Output, (kg/day)/MWh*
PRODUCT	
Sb[b]	0.27
WASTES	
Slag[c]	0.60
Sb	0.011
Pb	4.0×10^{-5}
Cu	3.0×10^{-5}
Zn	3.0×10^{-4}
Anolyte Sludge[c]	0.011
Sb	3.1×10^{-4}
Pb	5.6×10^{-8}
Cu	5.6×10^{-7}
Zn	2.2×10^{-8}
Ni	5.6×10^{-8}
Mn	2.3×10^{-7}
Cr	3.6×10^{-7}
As	1.8×10^{-7}
Cd	1.1×10^{-8}

[a] *Source: "Assessment of Industrial Hazardous Waste Practices in the Metal Smelting and Refining Industry. Volume II, Primary and Secondary NonFerrous Smelting and Refining," Calspan Corporation, Buffalo, New York, U.S. Department of Commerce Report No. PB-276-170, April 1977.*

[b] *Calculation: 92% of the 2.06% Sb in the primary lead input (Table 2.2) comes from antimony smelting. See text for details.*

[c] *80% of the antimony is produced pyrometallurgically and 20% is produced electrolytically.*

Table 2.13. Impact of EV Fleet on Existing Antimony Smelting[a]

Year	Fleet Size: Vehicles (thousands)	Fleet Size: MWh	Antimony Output[b]: MT/day	Antimony Output[b]: % of Smelter 1974 Output	Antimony Output[b]: % of U.S. Domestic Mine Output	Waste Effluents, kg/day: Slag	Metals in Slag: Sb	Metals in Slag: Pb	Waste Effluents, kg/day: Sludge	Metals in Sludge: Sb	Metals in Sludge: As
1990	250	6,200	1.7	4.0	50	3,700	68	0.25	68	1.9	1.1×10^{-3}
1995	870	22,000	5.8	14.	180	13,000	240	0.88	240	6.8	4.0×10^{-3}
2000	3000	75,000	20.	48.	610	45,000	830	3.0	830	23	13.5×10^{-3}

[a] *Source: Tables 2.2, 2.12, and Section 2.2, Minerals Yearbook, 1974 (U.S. Bureau of Mines), and "Assessment of Industrial Hazardous Waste Practices in the Metal Smelting and Refining Industry." Volume II, "Primary and Secondary Nonferrous Smelting and Refining," Calspan Corporation, Buffalo, New York, U.S. Department of Commerce Report No. PB-276-170, April 1977. Values are rounded to two significant figures.*

[b] *In 1974, the total U.S. primary smelter output of Sb was 15,000 MT, only 8% of which came from domestic mining. Half the domestic output came from antimony mines and the remainder as a byproduct of lead smelting (in excess of the amount used to produce antimonal lead at the primary lead smelters) (Minerals Yearbook, 1974).*

As shown in Table 2.13, the impact is considerable. For example, in 1995, 14% of the 1974 U.S. smelter output will be required to make antimonial battery lead; in the year 2000, 48% will be required. Because these increases are more than the 1974 domestic U.S. mine production of antimony, they would probably have to be met by increasing the amount imported. These increases are over and above the increased antimonial lead output resulting from the expansion of the primary lead smelting as detailed in Table 2.11. The values in Table 2.13 also show that in the year 2000, 47 MT of slag containing 830 kg of antimony and 3 kg of lead will be produced each day. Leaching studies show that, in contrast to the slag produced at lead smelters, the metals in this slag are soluble. Thus the slag, as well as the sludge, should be considered potentially hazardous.[26]

As with other operations in the cycle, these numbers must be kept in perspective, Even by the year 2000, the total smelter output increase required is 48% of the 1974 output. Also, the waste outputs assume no changes in control technology or smelting operations. Thus the values are more appropriately regarded as giving the scenario if SOA lead/acid batteries were manufactured in the 1990s using antimony produced by present-day smelting operations and control technology, with no changes.

2.3.3 Sulfuric Acid

Sulfuric acid (H_2SO_4) is used in the electrolyte of the battery and in battery manufacturing. It was noted in previous sections that primary (but not secondary) lead smelters have sulfuric acid plants associated with them. The first question to answer is whether the sulfuric acid manufactured by the acid plant at a primary smelter is sufficient for SOA lead/acid battery manufacturing.

As shown in Table 2.12, 14.5 (kg/day)/MWh new lead plus 9.6 (kg/day)/MWh recycled lead is used in fabrication of batteries. This requires 3.4 (kg/day)/MWh H_2SO_4 as battery electrolyte, plus an additional 5.6 (kg/day)/MWh to 18.1 (kg/day)/MWh of acid used in the manufacturing step.[17] The total amount of acid available from the primary smelting of galena (PbS), assuming that all the sulfur goes into the manufacture of sulfuric acid, is 6.8 (kg/day)/MWh [14.5 kg/day MWh × (98/208)]. Thus, more than enough acid is produced to satisfy the battery electrolyte requirements, but less than the total amount required for processing. The amounts that must be made up

from sources other than the acid plant at the smelter range from 2.2 (kg/day)/MWh (9.0 - 6.8) (Table 2.12) to 14.7 (kg/day)/MWh (21.5 - 6.8), depending on which type of manufacturing process is used.[17]

Sulfuric acid emissions will not be considered further here because the increase in production of sulfuric acid required to meet the demands of even a large fleet of EVs is very small compared to the present manufacturing level. For example, for a fleet of three million EVs growing at 25%/yr, from 2.5×10^5 MT/yr to 5.9×10^5 MT/yr of H_2SO_4 would be required for battery electrolyte plus manufacture. The total amount of H_2SO_4 manufactured in 1973 was 2.9×10^7 MT.[30] Thus, for the above scenario up to 2% of the total U.S. production of H_2SO_4 in 1973 might be required for the SOA lead/acid EV battery industry in the year 2000, which corresponds to the output of three average sized acid plants.[30] This is incrementally quite small and, for the purposes of this report, can be neglected.

2.4 MANUFACTURING PROCESS

2.4.1 Case and Cover

Lead/acid battery cases and covers are now made out of plastics instead of hard rubber, which was once the preferred material. The plastic produces a lighter weight case and has appreciable recycling potential, either as fuel or as plastic material. Rubber cases are heavy and are not recyclable.

Those plastics that have been used for battery cases include high-density polyethylene, polypropylene, polycarbonate and polyvinyl chloride.[14,16] Rather than treat all of the possibilities, the discussion here will be limited to polypropylene as a representative example. Polypropylene is widely used and is considered as an especially good material for battery cases and covers.[15,16]

The only part of case and cover manufacturing to be considered here is the polymerization of propylene into the polymer. The monomer propylene is produced as part of the petroleum refining operations. Thus, it is difficult to separate out emissions due only to propylene. The molding of cases and covers from the polymer will not be considered, as little information seems to be available about this manufacturing step.

In essence, the polymerization process consists of mixing the monomer with an appropriate catalyst and solvent in a reaction vessel at appropriate temperatures and pressures. The precipitated polymer is washed in alcohol to remove the catalyst and then steam treated to remove the solvent. Finally it is dried. The catalyst is separated from the alcohol and both are recycled.[31] Waste products include particulate and monomer air emissions,[31,32] and aqueous waste containing some catalyst, alcohol, polymer, and an inhibitor added to the monomer to prevent polymerization during storage. A newer (as of 1973) version of the polymerization process (described in Ref. 31) which produces less waste, has a growing commercial acceptance.

Emissions

Table 2.14 gives the emissions, in (kg/day)/MWh, to be expected from the polymerization of propylene into polypropylene, with the assumptions given earlier. The air emissions are uncontrolled

Table 2.14. Emissions from the Production of Polypropylene from Propylene for Lead/Acid Battery Cases and Covers[a]

Emission	*Emissions (kg/day/MWh)*
AIR[b,c]	
Particulates	0.0038
Propylene	8.7×10^{-4}
WATER[d]	
Total Suspended Solids	0.0014[e]
Vanadium[f]	--
Titanium[f]	--
Aluminum[f]	--
Solvents[f]	--
Inhibitor	--
SOLIDS	
Sludge	0.0025 - 0.025[g]

[a] *It is assumed that all of the used cases and covers are burned as fuel in the secondary smelters and none are recycled either as plastic feed material or as reusable cases and covers.*

[b] *Source: "Compilation of Air Pollutant Emission Factors." 3rd Edition, U.S. Environmental Protection Agency, Research Triangle Park, NC, Report No. AP-42, August 1977.*

[c] *These are uncontrolled emissions. Control methods are often considered as a part of the process that leads to recovery of a reactant or product. Emissions from the polymerization of vinyl chloride are larger by a factor of 12 to 25.*

[d] *Source: D. L. Becker, "Development Document for Effluent Limitations Guidelines and New Source Performance Standards for the Synthetic Resins Segment of the Plastics and Synthetic Materials Manufacturing Point Source Category," U.S. Environmental Protection Agency, Washington, D.C., U.S. Department of Commerce Report No. PB-239-241, March 1974, pp. 81, 96, 110, 111, 143-147.*

[e] *This is a controlled emission after extensive treatment of the raw wastewater from exemplary plants. The BPCTCA and BATEA average 30-day values are respectively 2.0 and 0.40 times the value given in the table.*

[f] *No data are given for amounts. The metals are included in the catalysts.*

[g] *The numbers give the estimated range of emissions and refer to the equivalent dry weight of the sludge.*

emissions. Much of the control equipment used is considered as part of the reactant or product recovery and not part of the controls.[32] No data are given for metals, solvents or inhibitor in the aqueous emissions.[31] These materials are expected to be present as they are used in the processing. The metals are used in the catalyst.[31]

Impact on Existing Industry

The impact on the existing polypropylene industry of an EV fleet growing at 25% per year and powered by SOA lead acid batteries, is indicated in Table 2.15. The entries in rows 4 and 5 of the table were generated by use of the facts that in 1972, 767,000 MT of polypropylene were consumed and there were nine producing plants.[31] Also it was assumed that U.S. production equals U.S. consumption.

The amount of polypropylene required for battery cases and covers is a very small percent of the large existing industry. Even by the year 2000, the amount of polypropylene required is only 8.7% of the 1972 consumption.

Table 2.15. Impact of EV Fleet on Existing Polypropylene Industry[a]

	Year		
	1990	*1995*	*2000*
Electric Vehicle Fleet			
Total EVs (thousands)	250	870	3,000
MWh Installed Batteries	6,200	22,000	75,000
Polypropylene Mfg.			
MT/day Required	16	53	180
% of Existing Capacity[b]	0.73	2.5	8.7
Number of New Plants[b] Required	0.067	0.23	0.078
Environmental Impact			
		AIR	
Particulates, kg/day	23	82	280
Propylene, kg/day	5.5	18	65
		WATER	
Total Suspended Solids, MT/day[c]	8.6	30	100
		SOLID	
Sludge (dry weight), MT/day[d]	82	280	980

[a] *From Tables 2.2 and 2.14, and beginning of Section 2.2.*

[b] *In 1972, 767,000 MT of polypropylene were consumed. There were nine producing units. The table entries assume U.S. production to be equal to U.S. consumption.*

[c] *The metals, solvents and inhibitor listed in Table 2.14 are excluded here as no emission data are given.*

[d] *The values given are computed using the midpoint of the range given in Table 2.14.*

The effluent emission rates are appreciable, especially from 1995 to 2000; however, they represent only a small fraction of the 1972 total emissions. For example, in the year 2000, the projected emissions from the polypropylene manufacturing support of the SOA lead/acid EV battery industry are only 5.2% of the total emissions in 1972.*

2.4.2 Battery

Manufacturing Process

A flow diagram of the lead/acid battery manufacturing process is given in Figure 2.2.[17,18,23] Lead alloy ingots are melted in a grid casting furnace, the antimony content is adjusted if necessary, and then the molten lead is molded into grids in a grid casting machine. Sodium silicate, and sometimes acetylene soot, are used as mold release agents.[18] After ejection from the mold, grids are trimmed and stacked. Air emissions from this operation, which consist mainly of lead alloy fumes and particulates, are generally low (about 0.4 kg lead/1000 batteries** produced).[18,23] Some manufacturers vent the emissions from the furnace to the outside atmosphere to protect the workers. The casting machine area is generally unvented. Solid emissions from this operation consist mainly of dusts, dross, lead scrap, and rejects of the lead alloys, and total about 22.5 kg/1000 kg batteries.[16,17**] Waste material is reclaimed† by sending it to a lead smelter or lead reclamation furnace.

Lead monoxide, which is used to make the battery paste, is made from refined lead by using either the ball mill or the Barton process. In the former, lead ingots are tumbled in a mill to which air is admitted. The tumbling action yields small particles of lead and generates heat, which initiates the oxidation of the lead particles.[23] In the Barton process, air is drawn through molten lead that is being rapidly stirred in a kettle; the atomized lead particles are oxidized by the air. In both these processes, the larger particles are collected in a cyclone or settling chamber and are further pulverized in a hammer mill. The finer particles are collected in baghouse filters.[18,23] Air (controlled) emissions include 0.05 kg lead/1000 batteries.[23] The filters are considered to be part of the manufacturing processes and not part of the emission control process.

In the paste mixing operation the lead oxide is mixed with sulfuric acid, water, and a very small amount of dynel fiber (about 0.03%) in a batch operation in a dough-type mixer. About 1% expander (a mixture of carbon black, barium sulfate and organics) is added if the paste is to be used for the negative plates.[18,23] The process must be temperature controlled to produce lead sulfate paste of the desired consistency. Most air emissions occur during addition of the dry ingredients to the mixer and consist of lead oxide and a small amount of the other paste constituents emitted at the rate of 5.1 kg/1000 batteries produced.[18] Solid emissions consist of contaminated paste, about 1.5 kg paste/1000 kg batteries,[16,17] which is sent to lead smelters

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This assumes that the emission rates used to obtain the entries in Table 2.14 represent fairly the actual emission rates for 1972.

**In this section all emission and waste amounts given in kg/1000 batteries refer to a starting, lighting and ignition battery (SLI), not an EV battery. Also, all waste amounts given in kg/1000 kg batteries (from Ref. 17) are obtained by multiplying the reference values (given in kg/1000 kg batteries dry weight) by 3/4 as 25% of the battery is electrolyte (Table 2.1).*

†*Solid emissions which are directly reclaimed during manufacturing are not indicated in Figure 2.2.*

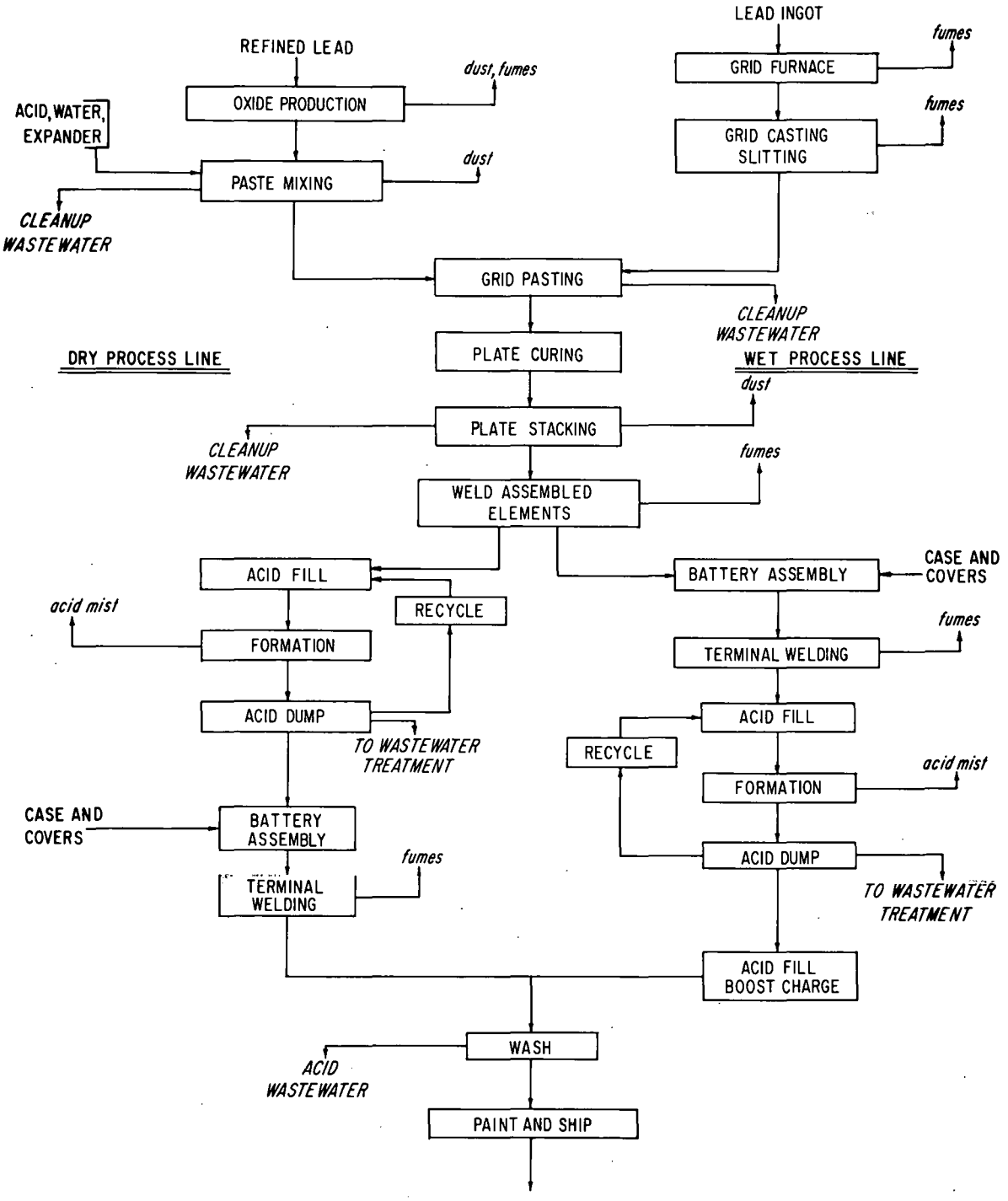

Fig. 2.2. Flow Diagram of the Lead/Acid Battery Manufacturing Process. Modified from Fig. 8, Ref. 17, and Fig. 4-4, Ref. 23.

for reclamation. After the grids are slit to form spaces, the paste is forced into the interstices in the grid-pasting operation to form plates (grids are called plates after the paste is applied). The plates are then cured for up to 72 hours. There are essentially no air emissions from this operation; however, the pasting area floor must be kept clean of paste as it is a potential source of fugitive dust.[23] Cleanup of the pasting area produces about 3.7 kg of waste product/1000 kg of batteries suspended in 560 kg of water.[17]

After curing, the plates are stacked with positive and negative plates alternating and with separators placed in between the positive and negative plates. Then the positive plates in the stack are connected together by either welding or burning a lead to each of the plates or by pouring molten lead around and between the plate tabs to form the connection. The negative plates are treated similarly, and then positive and negative posts are welded on. The complete units are then assembled into battery cases either before formation (wet process) or after formation (dry process). These two processes are explained later.

Much of the manufacturing air emissions occur during this three-process operation--stacking, burning and assembling--as much of the work is done by hand. Typically, operators straighten stacks by striking them edge-on against a hard surface,[18] as a result of which, paste particles become airborne. Methods of protecting the workers include wearing masks or venting the work area. Lead emissions in air for this three-process operation are 6.6 kg Pb/1000 batteries.[18,23] Solid emissions are mainly rejected plates which are recycled to the smelter.

At this point the positive and negative plates consist of lead grids with lead sulfate in the interstices of the grids. The lead sulfate in the plates is converted to active material in the formation process. Formation consists of immersing the plates in a sulfuric acid solution, connecting the positive and negative plates to the respective positive and negative sides of a D.C. source, and passing current through the system. This converts the lead sulfate to lead dioxide and lead which are the active materials on the positive and negative plates respectively.

Batteries made by the wet process are shipped filled with electrolyte and are ready to use. Batteries made by the dry process are shipped empty and must be filled with electrolyte before use. Dry process batteries do not deteriorate with standing and storage before use; however, they are somewhat more difficult to make and yield more effluents than is the case for the wet process batteries.

In the wet process line the elements are assembled into the case, the cover is placed on, sulfuric acid is placed into the case, and formation takes place in the case. After formation, which takes one to four days,[18] the acid is dumped, fresh acid is added, and the batteries are given a boost charge. Then they are tested, washed and readied for shipping.

In the dry process line, the plate assemblies are placed in tanks of sulfuric acid and formed for about 16 hours. The assemblies are then rinsed, dried and placed in battery cases; the lid is sealed on and the batteries are readied for shipping.[16-18] Completed batteries found to be faulty in testing are reclaimed by sending them to the battery recycling operation.

Air emissions from the formation process consist mainly of sulfuric acid mist released by hydrogen and oxygen bubbles that form during the process. These bubbles rise to the surface and

break, which throws a small amount of sulfuric acid into the air. Acid mist emission is worse for the dry process than for the wet process as formation for the dry process is carried out in open tanks and for a relatively short time.[18] Liquid and solid emissions from the formation process consist mainly of sulfuric acid, containing a small amount of lead.

The larger battery plants, which are presumed to be more typical of those manufacturing EV batteries than are the smaller plants, typically operate a low-temperature pot-type lead-reclamation furnace. This furnace reclaims the lead scrap and dross produced as waste or rejects during the various stages in the process prior to assembly.[23] The total lead air emissions from this lead reclaiming process are about 0.3 kg/1000 batteries.[23]

Control Technology

Air emissions from the overall manufacturing process are often controlled by scrubbers and fabric filters. In particular, emissions from the grid furnace and casting machines, paste mixing and plate drying operations, parts casting and formation can be controlled by scrubbers.[18,23] Grid casting, paste mixing, the three-process operation (stacking, burning and assembling), and parts casting can be controlled by fabric filters. Often, grid and parts casting and plate drying are uncontrolled, as these are minor sources of emissions. (The fabric filters and cyclones used in the lead oxide process are considered to be part of the process and not part of the control technology.) All the dusts and materials obtained from the filters are recycled to reclaim the lead.[23] Solid effluents such as lead oxides and sulfates and lead obtained from contaminated paste, rejected plates, dross and scrap, and precipitants settling out in the acid waste sump are recycled to the lead smelter.[16,17] The wastewater coming from the pasting area cleanup, acid dump during formation, and washing operations is neutralized with lye or with lime. The water remaining after filtration, which contains a small amount of lead (0.0075 kg lead/1000 kg batteries),[16,17] is discharged to the environment. Depending on the method of neutralization, the solids precipitated out are either recycled or disposed of in landfill.

Input

Table 2.16 gives the input flow, in kg/day per MWh installed, of various components or materials needed to manufacture the SOA lead/acid EV batteries; the total flows into batteries were taken from Table 2.2. Columns 2 through 4 give the total flow into batteries, showing the flow components coming from recycling and from new materials separately. For antimony and arsenic the "less than" and "greater than" symbols indicate that the recycle and new entries are upper and lower limits which must sum to a fixed total. As noted before, the recycle flow value assumes that no antimony or arsenic is lost to gaseous emissions during use of the battery. Since this is not the case, the value given is an upper limit. The new flow value, which must equal the total flow minus the recycle flow, must be increased by whatever amount the recycle flow should be decreased.

As noted in Section 2.1 it is assumed here that all of the cases and covers are recycled either as fuel or as plastic feed material, and none are reused directly as cases and covers. This could well change if it becomes economical to reuse EV battery cases and covers directly instead of using them as fuel or as plastic feed material. None of the remaining components are recycled, including the small amount of copper in the posts.[15]

Table 2.16. Input Flow into Lead/Acid Battery Manufacturing Process, (kg/day)/MWh

Component or Material	Into Batteries			Into Process[c]		Total	
	Recycle	New	Total[a]	Wet	Dry	Wet	Dry
Pb	9.6	14.5	24.1	--	--	24.1	
Sb	<0.20[b]	>0.30[b]	0.50	--	--	0.50	
As	<0.0034[b]	>0.0051[b]	0.0085	--	--	0.0085	
Cu	0	0.16	0.16	--	--	0.16	
Case and Cover	0	2.5	2.5	--	--	2.5	
Separator	0	0.13	0.13	--	--	0.13	
Expander	0	0.053	0.053	--	--	0.053	
Other	0	1.22	1.22	--	--	1.22	
H_2SO_4	0	3.4	3.4	5.6	18.1	9.0	21.5
H_2O	0	6.2	6.2	126	442	132	448
NaOH[d]	--	--	--	4.6	14.8	4.6	14.8
$Ca(OH)_2$[d]	--	--	--	4.2	13.6	4.2	13.6

[a] *From Table 2.2.*

[b] *Some of the Sb and As is lost during use of the batteries. The recycle amounts are upper limits which assume no such loss. The new amounts are lower limits, as whatever is lost during use must be made up by new material.*

[c] *Source: "Assessment of Industrial Hazardous Waste Practices Storage and Primary Batteries Industries," Versar Inc., report prepared for the U.S. Environmental Protection Agency, January 1975, U.S. Department of Commerce, Report No. PB-241, 204, Section 5.2, 1975. (Conversion factor = 28.7/1000; see text.)*

[d] *NaOH and $Ca(OH)_2$ are neutralization alternatives, so only one of the two applies to any given plant.*

As noted before, sulfuric acid and water are used both as electrolyte in the batteries and as part of processing to make batteries. Table 2.16 gives the flow components into batteries and into processing, with the latter flows given for both wet and dry processing methods. NaOH (lye) and $Ca(OH)_2$ (lime) are used only in the processing to neutralize the wastewater and to precipitate out most of the lead in the wastewater. Note that either one or the other but not both is used in any given plant.

The last column gives the total amounts of materials used both in the batteries and in the processing. Only the acid and water are used in both operations. No NaOH or $Ca(OH)_2$ goes into the batteries, and none of the remaining components is used in the processing.

Output

The output flow from the manufacturing process is 38.3 (kg/day)/MWh [28.7 (kg/day)/MWh dry weight] of SOA lead/acid EV batteries (Table 2.2). In addition to this are various waste product flows (air, liquid, and solid emissions).

Air Emissions. Table 2.17 gives typical air emission parameters for the battery manufacturing industry. In constructing the table, it was assumed that for each operation lead constitutes 50% of the total particulates emission.[23] This number appears, from a study of the

Table 2.17. Typical Air Emission Parameters for Lead/Acid Battery Manufacturing

	Uncontrolled Emissions,[a,b] (kg/1000 batteries)		Control[b]		Controlled Emissions, (kg/1000 batteries)	
	Particulates	Lead	Method	Efficiency	Particulates	Lead
Grid Casting	0.8	0.41	Scrubber (Rotoclone)	90%	0.08	0.04
Lead Oxide[c]	--	--	Baghouse filter	--	0.10	0.05
Paste Mixing	10.2	5.1	Scrubber	90%	1.02	0.51
Three-process Operation[d]	13.2	6.6	Baghouse filter	99%	0.13	0.07
Lead Reclamation Furnace	0.70	0.35	Scrubber	98%	0.014	0.0070
Formation	1.24(H_2SO_4)		Scrubber mist/ eliminator	97%	0.037(H_2SO_4)[e]	

[a] *Source: "Control Techniques for Lead Air Emissions," Vol. II, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, Report No. EPA-450/2-77-012, December 1977.*

[b] *Source: Lead/Acid Battery Manufacture Background Information, Proposed Standards. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, May 1979, Sections 3, 4, 6, 7.2 and Appendix C.*

[c] *Baghouse filters are considered part of the process, so only the controlled emissions are given.*

[d] *Stacking, burning and assembling before formation.*

[e] *In units of kg H_2SO_4/1000 batteries.*

reference source, to be an estimate rather than an actual measurement. Furthermore it is not likely that this percentage is the same for all the operations considered. For one reason, the chemical form in which the lead is emitted depends on the operation considered. For example, the lead grid casting emissions will be mainly in the form of lead fumes and lead oxide particles, whereas the three-process lead emissions are mainly in the form of lead paste consisting of a mixture of lead oxide and lead sulfate.[7] The values for the uncontrolled lead emissions are obtained from actual test results run at several plants.[18,23] The control methods given are those typically used for the various steps; however, they are not used by all plants. Fabric filters are never used because manufacturers believe that the sodium silicate used as a mold release agent will blind the fabric.[18] Paste mixing, at present, is controlled in most plants by a scrubber only; other plants use both baghouses and scrubbers to control the mixing. Even though baghouse filters are more efficient, about 98% to 99%, as compared to 90% for scrubbers, the scrubbers are preferred, occasionally in conjunction with filters, to control emissions during that part of the mixing process when the ingredients are moist with acid.[18,23] The industry typically uses baghouse filters for the three-process operation, although scrubbers are sometimes used. Scrubbers are typically used for the lead reclamation furnace, a low-temperature pot furnace.[18,23]

No lead emissions occur during the formation process, which gives off sulfuric acid mist along with some hydrogen and oxygen. More mist is given off during formation in the dry process, which is done in open tanks, than in the wet process; the value given in Table 2.17 is the result of tests made on a dry process line (Ref. 18, Section 4 and Appendix C). Essentially no data seem to be available on the wet process.[14] Typically no control methods are used for formation in the wet process line. As a result, it will be assumed here that the controlled dry process emission rate given in the table applies to both the wet process (no controls) and the dry process (controlled).

It should be noted that the H_2SO_4 mist emission rate given in Table 2.17 (1.24 kg/1000 batteries) is smaller by factors of 12 to 15 than other values in the literature[18] (14 kg/1000

batteries and 19 kg/1000 batteries). Clearly this is a large discrepancy, and indicates that further work is needed in this area.

The control efficiencies assigned to the various processes are associated with the methods in use for well-controlled processes at the present time. It is assumed that these or better methods will be used in all plants which manufacture SOA lead/acid batteries.

Finally, it should be noted that control efficiencies may not scale. For example, control efficiencies are higher by 0.2% for a 500 battery per day plant than for a 2000 to 6500 battery per day plant.[18] In a process controlled at 99% efficiency for 500 batteries per day, this can lead to a 20% increase in emission rates for the larger plants. This effect will be ignored here as it yields an uncertainty that is small compared to others.

The air emission flow rates from the manufacturing process are given in Table 2.18. Several considerations are used in calculating the air emissions from the data in Table 2.17. The amount of lead in an average battery is taken to be 11.8 kg.[18] It is assumed that the lead metal fume emissions are contributed by emissions from the grid casting and lead reclaiming operations only. The lead emissions as lead oxide come from the oxide mill plus 86.5% of the total emissions from paste mixing plus the three-process operations; the lead emissions in the form of lead sulfate comes from 13.5% of the paste mixing plus the three-process operations. These percentages are derived from the fact that in a typical paste formulation,[18] the amount of acid added is sufficient to convert 13.5% of the lead oxide to lead sulfate. The expander emission is 0.22% of the lead emissions from the paste mixing and three-process operations, and the separator emissions are contributed by the three-process operations only. The antimony and arsenic emissions are calculated assuming no fractionation and as arising from the grid casting and lead reclamation operations only (lead paste does not contain antimony or arsenic).

Liquid and Solid Emissions. Wastewater in a lead/acid battery manufacturing plant comes from many sources, including washdowns of the paste mixing area, the grid pasting area, the oxide production area, the formation room and the acid mixing area, water from wet scrubbers used for pollution control, rinsing and washing the battery during acid fill and dump, rinsing the dry charge plates, etc;[16] and sulfuric acid that is dumped and not recycled. Typically, 17.8 kg wastewater is generated per kg battery lead in the dry process manufacturing. For the wet process, 5.1 kg/kg battery lead is generated.[17] The median amount of wastewater for all lead/acid battery plants is 7.6 kg/kg battery lead.[16]

The wastewater contains about 2% to 4% sulfuric acid by weight and less than 25 ppm of lead (by weight).[17] In addition to lead and acid, the wastewater contains small amounts of iron (0.0045 to 0.063 kg/MT product), oil and grease (0.0094 to 0.092 kg/MT product), and total suspended solids (0.027-0.20 kg/MT product).

The wastewater is neutralized either with lye (NaOH) or lime ($Ca(OH)_2$). Lye neutralization is more expensive but it has the advantage of generating much less sludge, making disposal problems easier. In either case, the final treated water, which contains less than 3 ppm lead,[17] is discharged into an adjacent river, sanitary sewer, percolation pond, or the ground--depending on the location of the plant.[16] The final sludges, which contain appreciable concentrations of lead as lead hydroxides and lead sulfates, are at present disposed of in landfills.[16] Because these compounds are appreciably soluble in water (42.5 mg/liter for $PbSO_4$ and 150 mg/liter for

Table 2.18. Air Emissions Rates from Lead/Acid Battery Manufacturing

Material	Emissions, (kg/day)/MWh[a]	
	Uncontrolled	Controlled
Particulates	0.050	0.0028
Pb	0.025	0.0014
Pb Fumes[b]	0.0016	9.6×10^{-5}
Pb Oxides[c]	0.021[d]	0.0011[d]
Pb Sulfate	0.0032[d]	1.6×10^{-4}[d]
Sb[e]	3.2×10^{-5}	1.9×10^{-6}
As[e]	5.3×10^{-7}	3.4×10^{-8}
Expander[f]	5.1×10^{-5}	2.6×10^{-6}
Separator[g]	7.2×10^{-5}	7.2×10^{-7}
H_2SO_4	0.00252	7.6×10^{-5}

[a] *Conversion Factor:* $\frac{1000 \text{ batteries}}{11{,}800 \text{ kg Pb}} \times \frac{24.1 \text{ kg Pb}}{\text{day} \times \text{MWh}}$.

[b] *Assumes lead metal fumes come from grid casting and lead reclaiming only.*

[c] *Lead as lead oxide comes from lead oxide making, plus 86.5% of the total emissions from paste mixing and the three-process operations. Lead as lead sulfate comes from 13.5% of the total paste mixing and three-process operations. (See text.)*

[d] *The values given are the amount of lead emitted in the form shown.*

[e] *Assumes no fractionation.*

[f] *The expander emission is 0.22% of the total lead emissions from the paste mixing and the three-process operations.*

[g] *The separator emissions are part of those from the three-process operations.*

$Pb(OH)_2$[16]) precautions must be taken that the landfills are secured and that these toxic materials do not leach out into the groundwater.

Table 2.19 shows liquid and solid emissions flow rates from a SOA lead/acid battery manufacturing plant as calculated from the data in Tables 2.1, 2.16 and the 1975 Versar, Inc., Report to the EPA.[17] The emissions are given separately for the wet process and the dry process methods of manufacturing and for the use of lye or lime as the neutralizing agent for the wastewater.

If lime neutralization is used the final wastewater flow to the environment for the wet process battery is 112 (kg/day)/MWh. The water contains 2 to 3 ppm of lead. If lye neutralization is used, this wastewater includes a large amount of sodium sulfate [8.1 (kg/day)/MWh]. If the dry process is used, the corresponding flow rates of water and sodium sulfate are increased by a factor of about 3.5, with the lead flow remaining constant.

The flow of solid sludge in the case of lye neutralization is small [26 (g/day)/MWh (wet process) to 38 (g/day)/MWh (dry process)]; however, it contains 35% lead salts and must be

Table 2.19. Liquid and Solid Emissions Rates from Lead/Acid Manufacturing (kg/day/MWh)[a]

	Wet Process		Dry Process	
Material	*NaOH*	*$Ca(OH)_2$*	*NaOH*	*$Ca(OH)_2$*
Liquid	134	112	468	395
Na_2SO_4	8.1	--	26.2	--
Pb	← 2.9×10^{-4} →			
Sb[b]	← 6.0×10^{-6} →			
As[b]	← 1.0×10^{-7} →			
H_2O	126	112	442	395
Solid (Sludge)[c]	0.026	22.3	0.038	72
Pb (total)[c]	← 0.0053 →		← 0.010 →	
$PbSO_4$[c,d]	← 0.0048 →		← 0.0093 →	
$Pb(OH)_2$[c,d]	← 0.0022 →		← 0.0044 →	
Sb[b,c]	← 1.1×10^{-4} →		← 0.00021 →	
As[b,c]	← 1.9×10^{-6} →		← 3.5×10^{-6} →	
$CaSO_4$	--	7.8	--	25.2
Expander[b]	← 1.2×10^{-5} →		← 2.2×10^{-5} →	
H_2O	0.016	14.5	0.025	46.8

[a] *Source: "Assessment of Industrial Hazardous Waste Practices Storage and Primary Batteries Industries," Versar, Inc., report prepared for the U.S. Environmental Protection Agency, January 1975, U.S. Department of Commerce, Report No. PB-241,204, Section 5.2, 1975. Note: In the Versar, Inc., report, the results are given in terms of kg effluent/MT dry weight of batteries. The SOA lead/acid batteries contain 830 kg Pb/1000 kg dry weight (Table 2.1). Thus (Table 2.2), the conversion factor is* $24.1 \frac{kg \cdot Pb}{day \times MWh} \times \frac{1000 \text{ kg dry wt}}{830 \text{ kg Pb}}$.

[b] *Assumes no fractionation.*

[c] *The Versar, Inc., data are given for a composite plant producing 80% dry process and 20% wet process batteries. The wastewater lead content is given for the composite plant; the difference in the wastewater lead output (0.174 kg/MT dry weight) between the two processes is also given (assuming both lines process wastewater through the sump). Thus, one can solve for the output of the two processes separately.*

[d] *The amounts of chemical products, not lead equivalents, are given.*

disposed of carefully in secured landfills. In the case of lime neutralization, the flow of sludge is much larger [22.3 (kg/day)/MWh and 72 (kg/day)/MWh for the respective wet and dry process lines]. This sludge contains 240 ppm (wet process) or 140 ppm lead (dry process) in forms that will leach, so care must be taken with its disposal.

Impact on Existing Industry

Table 2.20 gives the impact on the existing lead/acid manufacturing industry of a large fleet of SOA lead/acid EVs. Columns 3-6 give the battery output in terms of metric tons/day of batteries, % of existing capacity (1973) and number of large new plants required. Columns 7-29 give the air emissions, water emissions, and solid emissions. Since 80% of the manufacturing occurs by the wet process, the water and solid emissions were calculated for this process only. As can be seen from Table 2.19, the emission factors for the dry process are 1.5-3 times larger. Emission factors are given for both the lye and the lime neutralization processes.

Table 2.20. Impact of EV Fleet on Existing Lead/Acid Battery Manufacturing Industry[a]

Year	Fleet Size: Vehicles, thousands	Fleet Size: MWh	Battery Output: MT/day	Battery Output: % of Existing Capacity[b]	No. of Large New Plants Required[c]	Controlled Air Emissions, kg/day: Partic.	Pb	Sb	As	H_2SO_4 Mist
1990	250	6,200	190	7.8	1.9	18	8.7	0.012	0.00021	0.46
1995	870	22,000	830	27	6.8	62	30	0.042	0.00075	1.6
2000	3000	75,000	2800	94	23	210	100	0.14	0.0026	5.6

Year	Fleet Size: Vehicles, thousands	Fleet Size: MWh	Water Emissions: Lye Neutralization: Water[d,e]	Na_2SO_4[d,e]	Pb[f]	Sb[f]	As[f]	Lime Neutralization: Water[d,e]	Pb[f]	Sb[f]	As[f]
1990	250	6,200	830	50	1.8	0.042	6.2×10^{-4}	700	1.8	0.042	6.2×10^{-4}
1995	870	22,000	2,900	175	6.3	0.13	2.2×10^{-3}	2400	6.3	0.13	2.2×10^{-3}
2000	3000	75,000	10,000	600	22	0.45	7.5×10^{-3}	8400	22	0.45	7.5×10^{-3}

Year	Fleet Size: Vehicles, thousands	Fleet Size: MWh	Solid Emissions[d]: Lye Neutralization Sludge[g]: Total[e]	Pb[f]	Sb[f]	As[f]	Lime Neutralization Sludge[g]: Total[e]	$CaSO_4$[e]	Pb[f]	Sb[f]	As[f]
1990	250	6,200	0.16	33	0.68	0.012	138	48	33	0.68	0.012
1995	870	22,000	0.56	110	2.3	0.042	480	170	110	2.3	0.042
2000	3000	75,000	1.45	400	8.3	0.14	1700	580	400	8.3	0.14

[a] *Sources: Tables 2.2, 2.18, 2.19 and Section 2.2.*

[b] *1973 consumption of lead in battery manufacture was 7.0×10^5 MT, which is equivalent to a battery production rate of 3000 MT/day. (Source: Table 2.1 and Z. A. Munir, E. Fuss and L. Ivers, "An Analysis of the Recycling of Metals, Final Report," University of California, Davis, for U.S. Dept. of Energy, Report No. TID-28286, January 1978.)*

[c] *A large battery plant produces 6500 standard auto batteries per day, each containing 11.8 kg of lead.*

[d] *Calculated for the wet process only. Values for the dry process are 2 to 3 times larger.*

[e] *Values are given in MT/day.*

[f] *Values are given in kg/day.*

[g] *Sludge is taken to be 65% water and 35% solids.*

As is clear from the entries in Table 2.20 the impact of an EV fleet growing at 25%/year to 3×10^6 SOA lead/acid EVs in the year 2000 is considerable. By the year 2000 the industry capacity would have to add 23 large new manufacturing plants which corresponds to a 94% increase in capacity. This new capacity would emit 100 kg Pb/day, 2.2 kg Sb/day and 5.6 kg H_2SO_4 mist/day into the atmosphere. Water emissions include 22 kg Pb/day and 600 MT Na_2SO_4/day (if lye neutralization is used). Solid emissions include 1.45 MT sludge/day if lye neutralization is used and 1700 MT sludge/day if lime neutralization is used. In either case the sludges will contain 400 kg Pb/day and 8.3 kg Sb/day.

In particular the lead air emissions and sludge emissions are considerable. In case of the lime sludge it would have to be disposed of in an environmentally acceptable way, as in a secured landfill. The sludge obtained in the case of lye neutralization has a sufficient concentration of lead so that it may be economical to send it to a smelter for reclaiming lead.

In any case these amounts of emitted lead, although considerable, must be kept in perspective. The water and solid emissions of lead noted above are less than the total amount emitted at present by the battery manufacturing industry and the air emissions of lead are a small fraction of that emitted by the burning of leaded gasoline.[20] Furthermore, advances in control technology anticipated to be in use in the 1990s should reduce the emissions below the values given in the table.

Cycle Loss

A 1969 estimate quoted in Reference 20 states that 72% of the battery lead is recycled during battery manufacturing. Another estimate[19] by the lead industries association, which was reported in 1971, states that 80% of the battery lead is recycled. Recent reports anticipate that in the future 90% or more of the battery lead will be recycled.[6,16]

For the calculations made in this report, the cycle losses (except for those explicitly discussed) are assumed to occur as whole batteries and at one point in the cycle. In evaluating the effect of a 10% cycle loss there are two factors to keep in mind. The effect on the cycle parameters or on the emissions calculations of removing the 10% figure or distributing it as it occurs in the cycle is quite small and can be ignored. However, the direct effect of the loss, if it is lost as an emission to the environment, can have a very large effect and can completely swamp the calculations reported here. Clearly, if the 10% cycle loss were lost as air emissions of lead, this would swamp all the other lead air emissions.

At present, no data are available indicating what this cycle loss consists of, now and in what form the emissions from this loss are distributed, or even if there are emissions associated with this loss. In essence the loss consists of lead which is unaccounted for in the manufacture-use-recycle lead/acid battery cycle.

Because of these uncertainties and lack of knowledge about the loss/distribution in the cycle, the loss has been concentrated at one point in the cycle in an easily treatable form and any possible emissions which the loss might represent have been ignored.

2.5 BATTERY USE

The use scenario for the SOA lead/acid battery in the early 1980s typically considers a four-passenger, secondary urban EV car[4] with an average daily distance driven of 44 km.[1,4] This is roughly the distance driven each day at present in the large urban areas[1] and is well below the projected range of 105 km for such vehicles.

Other parts of the scenario considered most likely to evolve are that car owners will charge their vehicles in their home garages or apartment garage stalls when the cars are not being used and that charging stations will have to be provided both along highways and at hotels and motels for the occasional long trips and for the motorist who runs out of electricity.

During the battery lifetime, the owner of the vehicle will have to maintain the batteries by charging them at regular intervals and by adding water at occasional intervals (to replace the water lost by electrolysis during charging). At present, it is not clear whether the typical car owner will recharge his car at the end of each day's driving to keep the batteries fully charged or whether he will wait until the batteries are close to being fully discharged, which will happen on the average every (105/44) 2.4 days, before completely recharging the batteries. In any case, it is likely that most of the charging will be done at home in the garage (or in the apartment or condominium stall) in the nighttime when the car is not in use.

The most likely scenario, then, seems to be one in which most battery charging will take place at night. For a large fleet of electric vehicles, this can constitute a large power demand from the utility power stations. Because EV power demand will occur at a time when other demands for electric power are less, a large fleet of EVs may greatly assist in leveling the power load demand of a utility, and may even obviate another projected use of storage batteries, i.e., as load levelers for power stations. However, this aspect, as well as the trade-offs in emissions resulting when an internal combustion vehicle is replaced by an EV deriving its energy from the power station,[33] are outside the scope of this report.

Various watering devices have been developed to make the water-replacement operation simple and automatic.[34] Work is going on at present to develop maintenance-free batteries for EV application.[12] If developed, such batteries would require little or no added water during their lifetime. At present, maintenance-free starting, lighting and ignition (SLI) batteries do exist. In these batteries, calcium and tin replace antimony and arsenic in the grids. Thus far, these batteries do not stand up well to repeated deep charge-discharge cycles.[12,14] Other methods which are being developed to reduce gas emission include catalytic recombination devices to recombine the hydrogen and oxygen into water.[12] Because of the problems with calcium-tin grids, it is not known at present how the gassing problem will be solved for EV batteries. As a result calcium and tin emissions are ignored in this report (even though regular battery lead can have as much as 0.5% tin).[7]

2.5.1 Air Emissions

During normal use in driving, EVs are essentially emission free. The only emissions that may occur in driving are a small amount of ozone produced by sparking in the electric drive motors and possible emissions arising from regenerative braking. The former is outside the scope of this report and the latter will be discussed below.

The main emissions occur during charging of a battery, especially during overcharge. During the main part of the charge cycle, the lead sulfate on both the positive and negative plates is converted back to lead dioxide, and lead, respectively, with the release of sulfuric acid into the electrolyte. As the charging nears completion and overcharge begins, electrolysis of water occurs with the generation of hydrogen and oxygen gas at the negative and positive electrodes. At the same time, small amounts of arsine (AsH_3) and stibine (SbH_3) gases are produced. As overcharging continues, the production rate of AsH_3 and SbH_3 rises rapidly, persists over the first 1.5 to 2 hours of the overcharge period, and slowly decreases.[10] Production of these gases is very sensitive to the voltage; very little gas is produced at charging voltages below 2.40 volts per cell.[10] At present, not too much is known regarding arsine and stibine generation. A recent review of some of the literature on this subject[11] discusses some of the work done in this area.

The production rate profile for AsH_3 and SbH_3 is obtained by first carrying out a constant current charge until the cell is almost fully charged, followed by a constant voltage tapered charge for three hours. Occasionally an equalization charge which is a constant voltage tapered charge is carried out on the system. Overcharge regimes are necessary in order to ensure that in a large battery, all cells, including the weakest, are fully charged at the end of the charge period. This becomes especially important as the battery ages, as all cells do not age uniformly. If the weakest cell is not fully charged, the possibility arises during deep discharge, of failure and polarity reversal of weak cells, resulting in failure of the EV battery.

The charge regime of one equalization charge for every four regular charges is one that is projected for use of batteries in the load-leveling mode, which is the only mode for which actual detailed measurements of SbH_3 and AsH_3 evaluation have been carried out.[10] In the absence of further information, these results will be assumed to apply to EV batteries.

Table 2.21 gives the air emissions to be expected under such an assumption. Columns 2-4 give the amounts in grams (not kilograms) of the species emitted in one charge of a 26 kWh EV battery. The two equalization charge columns (3 and 4) give the emissions resulting from the equalization charges carried out at 2.45 V and 2.65 V, respectively, and with the time current profile given in Reference 10. The values given for hydrogen and oxygen are upper limits since they are calculated assuming that the entire overcharge and equalization charge is used to electrolyze water. The arsine and stibine values are calculated by scaling the measured values given in Reference 10 for a 4.2 kWh battery up to 25 kWh. The average flow rates, in kilograms per day per MWh, installed, were calculated assuming that on the average, one full charge is carried out every 2.4 days (44 km/day distance driven in a car with an 105 km range) and that one out of every five charges is an equalization charge (also 40 × 25 kWh = 1 MWh).

In addition to H_2, O_2, AsH_3 and SbH_3 emissions, traces of ozone and H_2SO_4 mist have been reported.[6] The latter arises from the action of H_2 and O_2 bubbles breaking on the surface of the electrolyte, just as during the formation operation in the manufacturing process. However, most of the acid falls back in and does not escape out of the battery.

It must be emphasized that the emissions given in Table 2.21 were calculated with a charging profile assumed to be representative of the load-leveling mode, and are very uncertain. It is not known if the emissions so calculated are representative for batteries used in electric vehicles. If it is possible to change the charge parameters for EV batteries without compromising

Table 2.21. Air Emissions from the Charging of EV Batteries[a]

Chemical Species	Emissions from One EV Battery, g: One Regular Charge	One Equalization Charge: 245 V	One Equalization Charge: 2.65 V	Average Flow Rate, (kg/day)/MWh[b]: 2.45 V	Average Flow Rate, (kg/day)/MWh[b]: 2.65 V
H_2	<45[c,d]	<8.3[d,e]	<55[d,e]	<0.63	<0.78
O_2	<360[c,d]	<66[d,e]	<440[d,e]	<5.0	<6.2
SbH_3[f]	0.072	0.043	0.38	0.0011	0.0022
AsH_3[f]	0.0020	0.00065	0.0020	2.8×10^{-5}	3.3×10^{-5}
Ozone		Trace			Trace
H_2SO_4 (mist)		Trace			Trace

[a] *Source: R. Varma and N. P. Yao, "Stibine and Arsine Generation for a Lead Acid Cell During Charging Mode Under a Utility-Load Levelling Duty Cycle," Argonne National Laboratory, Il. Report No. ANL/OEPM-77-5, March 1978. These are uncontrolled emissions and assume neither recombination of H_2 and SO_2 nor retention or decomposition of SbH_3 or AsH_3.*

[b] *Assumes one full charge every (105 km/44 km/day) = 2.4 days and one equalization charge after four regular charges.*

[c] *Assumes 1200 Ah overcharge (References 6 and 13) for a regular charge cycle on a 25 kWh battery (Faraday constant: 1 mole electrons = 26.8 Ah).*

[d] *Values are upper limits, as they are calculated assuming that all the overcharge and equalization charge electrolyzes water.*

[e] *Calculated from Tables of Varma and Yao, which give average current flow and duration of equalization charges and scaled by 25 kWh/4.2 kWh, as measurements by Varma and Yao were done on a 4.2 kWh battery.*

[f] *Conversion factor: 25 kWh/4.2 kWh (Varma and Yao).*

battery performance, then clearly the numbers given are too high. In this connection, one notes that SbH_3 and AsH_3 production rates are quite sensitive to the charging voltage and show a threshold of about 2.40 volts per cell.[10] The values in the table were calculated assuming no control measures--such as catalytic recombination of H_2 and O_2, or retention or decomposition of SbH_3 and AsH_3. Any such control methods, if feasible, would reduce the amounts of these gases and of the H_2 and O_2 that would be emitted.

In spite of these caveats, the main point to be made is that arsenic and stibine production during charge presents a potential environmental health problem[9] that is unique to the lead/acid battery. A scenario in which millions of cars, while being charged in a home garage, are each giving off 72 mg stibine and 2 mg arsine every 2.4 days (regular charge) into the restricted garage atmosphere cannot be ignored. At present, arsine and stibine are thought to be extremely toxic gases (Sec. 7). The present threshold limit values (TLVs) of 0.2 mg/m^3 for arsine and 0.5 mg/m^3 for stibine which are the maximum allowable concentrations for an eight-hour exposure have been unchanged since 1950.[11] However, the amount of research done on the effect of these gases on animals is quite small.[11] In particular, the effect of repeated low-level doses needs to be investigated.

Account must also be taken of the fact that SbH_3 and AsH_3 decompose in air to produce antimony and arsenic oxides, presumably in form of a dust. SbH_3 has a half life for such decomposition of 6 to 12 minutes.[11] Under uncontrolled conditions and after years of use, the amounts of antimony and arsenic oxide dust which have accumulated on the walls and floor of a

home garage could present a serious health problem. Also, venting of these gases during charging to outside the garage will produce an oxide dust fall. An estimate made[6] for batteries used in the load-leveling mode gave a surface-soil concentration (5 cm deep) of 32 ppm antimony outside the building.

There are many possible methods being considered by which to reduce the arsine and stibine emissions to environmentally acceptable levels. It is not necessary to evaluate them here. The main points to be made are that (1) the arsine-stibine problem is unique to lead/acid batteries used in deep discharge-charge cycle modes (batteries used in internal combustion cars are maintained in a state of more or less continuous charge without leading to overcharge); (2) a technological fix of this problem is required before large-scale production of these batteries for use in EVs is begun.

Besides the arsine and stibine problem, the emission of H_2 and O_2 presents a problem in that a potential for explosion is present. This problem, which is common to many battery systems and has been recognized for some time,[9] also must be solved before large-scale use of batteries in EVs occurs. A further possible source of air emissions during use of the lead/acid battery arises from the fact that EVs may have some form of regenerative braking, i.e., part of the braking action needed to slow down or stop a vehicle will come from using the energy of motion of the car to add charge to the batteries. This can increase the range of the vehicle by about 15%.[32] The charge rate during regenerative braking will necessarily be quite high, which may lead to the possible production of arsine, stibine, hydrogen and oxygen when regenerative braking is used.

However, the environmental and health effects of these emissions (Sec. 7) would be expected to be much smaller than those arising from recharging, for two reasons: (1) the batteries are being charged, not overcharged, and if the necessary high charge rate does not lead to gas evolution, the amounts of gas evolved should be small; (2) the gas emissions that do result will be given off into the open area (the streets) rather than into restricted areas such as a garage.

2.5.2 Liquid and Solid Emissions

The lead/acid battery generates essentially no liquid or solid emissions during driving or charging. As a result such emissions will not be considered in this report.

2.6 BATTERY RECYCLING

2.6.1 Process Description

Battery Breaking

At the end of its useful life in an electric vehicle the lead/acid battery will be removed and collected for recycling. Major recovery sources include battery sales distributors and automobile reclamation/salvaging sites. Generally, spent batteries are collected by scrap dealers, who transport the batteries to a dismantling and processing site.

The batteries may or may not be drained of electrolytes prior to transportation. In either case, there generally exist some residual electrolyte. A wash with water prior to transportation may be part of the operation; otherwise, acid leaking from the batteries during handling and transportation will cause safety and equipment corrosion problems. On arrival at the processing site the scrap batteries are transferred to a storage facility.

Battery breaking operations are widely distributed and are carried on either by scrap dealers, who sell the scrap to secondary smelters, or on the site of the secondary smelting operations. Often these operations are located in urban areas in order to be close to the supply of used batteries.[35]

A number of different dismantling operations are in use. The cases can be separated from the top by use of a hand axe, by a sawing operation, by the dropping of the battery top downwards, or by crushing. The plate material is then separated from the cases which may be reusable if they are not damaged by the dismantling operations. There are operations in Scandinavia[36] which drain the electrolyte and feed the whole battery into the secondary smelting furnace. Also, at one operation in Pennsylvania, batteries having polypropylene cases are crushed and then the entire battery is fed to the reverberatory furnace.[15]

At present it is uncertain which of the breaking processes will be used for the lead/acid EV batteries, or whether the batteries will be smelted whole. Rather than treat each process separately, one operation will be used here as representative of the processes: dismantling by sawing followed by separation of the used plates from the case.

The main waste product from battery breaking operations is wastewater containing the spent electrolyte with suspended solids. The wastewater is also generated by such operation as cooling and washing the saws, and washing the cases before recycling as plastic. The waste is discharged to sanitary sewers. At present, some plants treat the waste before discharge.[35]

As noted before, it is assumed in this report that the plastic cases and covers of the electric vehicle batteries will be either burned as fuel in the secondary smelting operation or ground up and reused as plastic feed materials. It may be possible that some of the plastic cases and covers be directly reused.[6,8]

Other battery components such as separator material and copper from the posts are considered waste. The separator material which is stuck in the plates will be burnt in the smelting operation. The remainder will either follow the cases or be disposed of in landfills. In one operation, any copper present ends up in the slag from the furnace in the secondary smelting operation.[15]

Secondary Smelting and Refining

After separation from the used battery, the lead plates and other lead battery scrap (posts, connectors, etc.) are reclaimed by being smelted and refined. In 1973, 56.5% of the total secondary lead production came from used battery plates.[20] The smelting process proceeds by feeding the battery scrap plus fluxes (which consist of limestone and iron scrap for the blast furnace)[23,26] into either a reverberatory furnace or a blast furnace. The output slag

from the reverberatory furnace is also used as feed for the blast furnace. About two-thirds of the secondary lead is processed in a blast furnace.[23]

The output lead from the reverberatory furnace is soft or antimony-free lead which is suitable for making lead oxide. The output of the blast furnace is hard or antimonial lead which is suitable for making battery grids. If necessary, the composition of the output lead is adjusted with alloy elements in a pot furnace.[23,26]

There are about 80 secondary lead smelters in the United States.[35] These smelters are small compared to primary smelters and are located mainly in urban areas in order to be close to sources of supply. An average smelter produces about 20,000 metric tons of lead/year.[35]

2.6.2 Air Emissions

Battery Breaking

There is little available information concerning atmospheric emissions related to the collection, storage and dismantling operations of spent lead/acid batteries. Furthermore, there are no known or reported air pollution abatement facilities associated with battery dismantling operations in the United States. However, an analysis of the anticipated atmospheric emissions generated in the various possible routes to dismantling and resource recovery has been made and is presented below.

There are a number of process steps which result in hazardous atmospheric emissions of aerosols or entrained liquid particles (mist), as well as other hazardous emissions including fugitive dust containing lead. Sulfuric acid mist is generated by the agitation of the electrolyte and residuals in the collection and transportation of scrap batteries. Also, fugitive lead dust is generated during collection and transportation of the lead scrap. Batteries are kept wet, to minimize the amount of lead dust and dirt emitted. However, sulfuric acid mist from battery breaking remains a potential environmental and health problem. For example, small droplets of acid mist emitted from storage battery reclamation sites have been detected.[37]

Storage sites, particularly open sites where battery scrap is allowed to dry, will be sources of lead dust emissions due to normal wind-blown activity as well as due to normal battery conveyance activities. A study of lead dispersion in the area adjacent to a smelter in Germany indicated that two-thirds of the lead found outside the smelter was attributed to fine materials carried from piles of materials stored in the stockyard. At this study site, even though attempts were made to keep the material damp, some material did dry in the sun and the dust became windborne.[36]

Particle emissions and acid mists can also be generated by various steps used to dismantle old batteries. If the batteries are dismantled by a sawing process, the particle emissions would be low as the saw is kept wet. However, acidic mists would be generated. Processes which crush batteries and then separate components would also be expected to generate dusts and acid mists. The dusts would include particles of case and separator material as well as some lead particles. Processes which feed the batteries directly into the smelter without prior component separation or crushing would be expected to generate minimal dust and mist.[36]

It should be noted that the spent electrolyte in old batteries contains appreciable amounts of antimony (24-62 mg/L), cadmium (0.5-5 mg/L), and arsenic (0.8-5 mg/L) besides lead (6-280 mg/L) and other metals.[35] These materials all have presumably leached out of the battery lead during use and would be present in the acid mist.

Secondary Smelting and Refining

The main sources of atmospheric pollutants from the secondary smelting and refining operations are emissions from the blast and reverberatory furnaces and fugitive dust emissions resulting from various operations in the plant. In particular, uncontrolled air emissions from the furnaces contain particles with a 23% lead content. Refining operations, which are often done in pot furnaces, also contribute a small amount of lead fumes.[23]

Air emission control technology includes the use of baghouse filters to control the reverberatory and blast furnace emissions. The output gases from the baghouse filters are fed into wet scrubbers to remove sulfur oxides, and the resultant wastewater is neutralized with lime. The dust collected by the baghouse filters is recycled to the reverberatory furnace.[26] It is assumed here that by the early 1980s all secondary smelters will use baghouse filters followed by wet scrubbers plus lime neutralization of the output to control air emissions.[26]

Air emission flow rates from secondary smelting of the battery plates are shown in Table 2.22. As is the case for previous tables, the flow rates are given in terms of kg/day per MWh of battery capacity installed in electric vehicles. It is assumed that a baghouse filter followed by a lime scrubber is used to control the emissions for the furnaces. An uncontrolled furnace emission factor of 113 kg particulates per metric ton of lead produced, and a particulate lead composition of 23%, were assumed. These figures are essentially the same for reverberatory and blast furnaces.[23] Other sources give somewhat lower uncontrolled particulate emissions of 74 kg/MT.[32]

The control factor of 99.7%[32] for the particulates and lead appears high. However, it would seem to be reasonable since these emissions are controlled by both the baghouse filter and the scrubber. The control factor for the SO_2 is taken to be 90%[38] as it is removed by the scrubber only. The SO_2 comes mainly from the lead sulfate in the spent battery plates. The emission flow is calculated from the H_2SO_4-as-$PbSO_4$ entry in Table 2.2 by use of the assumption that all the sulfur originally present as sulfate is emitted as SO_2.

Fugitive dusts arise from such operations as moving, piling, and furnace loading of lead scrap, and other operations associated with the smelting. They are kept to a low level by good housekeeping techniques, which are the main method of control. Since there do not seem to be any data on the lead content of fugitive emissions, the figure of 23%, which was used for the furnace emissions, was used here for the fugitive dust also.

The total emissions are given in Table 2.22 as the sum of the controlled furnace emissions and the fugitive emissions which are uncontrolled. It is evident that most of the total emissions arise from fugitive dusts. These results indicate that further reductions in lead air emissions from secondary smelters are dependent on improving housekeeping techniques.

Table 2.22. Air Emissions from Secondary Lead Smelting,[a] (kg/day)/MWh

Species	Furnaces		Fugitive[c]	Total[d]
	Uncontrolled[b]	Controlled[b]		
Particulates	1.08	0.0032	0.048	0.051
Pb[e]	0.25	7.5×10^{-4}	0.011	0.012
Sb[f]	0.0052	1.5×10^{-5}	2.2×10^{-4}	2.4×10^{-4}
As[f]	8.7×10^{-5}	2.6×10^{-7}	3.8×10^{-6}	4.1×10^{-6}
SO_2[g,h]	0.63	0.063	--	0.063

[a] *Sources: Refs. 15, 23, 32, 38 and Table 2.2.*

[b] *Assumes 113 kg particulates/MT lead product (uncontrolled) and a control factor of 99.7%. These figures apply to reverberatory furnace and are quite similar to the emission factors for the blast furnace (Refs. 23 and 32).*

[c] *Assumes 5.1 kg particulates/MT lead product and no control (Ref. 23).*

[d] *Entries in this column are the sum of the controlled furnace and the fugitive entries.*

[e] *Assumes lead is 23% of the particulates (Ref. 23).*

[f] *Assumes no fractionation of Sb and As from Pb.*

[g] *Assumes all the sulfur, as $PbSO_4$ in the spent battery plates is emitted as SO_2.*

[h] *Control factor = 90% for lime/limestone scrubber (Ref. 38).*

2.6.3 Liquid Emissions

Battery Breaking

As noted, the main liquid emissions from the battery breaking operations include the discarded electrolyte, washwater from the cases and saws, and rainfall runoff from buildings and stored piles of plates. One typical battery breaking plant produces from the saw cooling and electrolyte 690 to 1000 liters of acid wastewater per metric ton of lead. This wastewater is characterized by high acidity and sulfate content, high dissolved and suspended solids, and high metal content, particularly lead and antimony. Another typical plant, which includes water from case washings, produces 2400 liters of acid water per metric ton of lead.[35]

Applicable control technology consists of treating the wastewater with lime and flocculants (0.014 kg lime and 0.00023 kg flocculant per liter of wastewater), then settling and possibly vacuum filtration for plants discharging to streams or neutralization with ammonia (0.38 kg NH_3/liter wastewater) and settling before discharge to sewers. Also, pits are constructed to contain runoff from battery case and plate storage areas.[35] If the solid sludge is disposed of in an onsite landfill then the leachate from the landfill can be recycled for further treatment.[39]

At present, pollution control technology of water emissions by plants with battery breaking operations varies widely from careful treatment to none at all. The Environmental Protection Agency has stated that for implementation of the BCPTCA,* BATEA** and NSPS† effluent limitations,

**BCPTCA is acronym for Best Practicable Control Technology Currently Available.*

***BATEA is acronym for Best Available Technology Economically Achievable.*

†*NSPS is acronym for New Source Performance Standards.*

lime plus flocculant treatment followed by settling and possibly filtration is the required treatment.[35] For this reason it will be assumed in what follows that all plants which recycle EV batteries will use this type of wastewater treatment.

Table 2.23 shows influent and effluent concentrations and waste loads for a plant using an exemplary lime treatment of wastewater. Most of the influent waste load comes from the spent battery electrolyte and the battery breaking operations. The reason is that the wastewater for the secondary smelting part of the operation, which comes from the SO_2 scrubber, is treated and recycled, and is not discharged.[35] Analyses of the spent electrolyte or battery breaker sump at different plants all show appreciable amounts of antimony, arsenic, and cadmium as well as other heavy metals such as zinc, copper, and iron.[35,39] These metals are all present in the grid lead of new batteries[7] and are probably leached out of the grid during use of the battery. In particular, during battery charging and discharging, antimony diffuses from the grids into the electrolyte.[11] Also, more antimony than lead remains in solution in the spent electrolyte.

Table 2.23. Exemplary Lime Treatment of Wastewater from Battery Breaking Operations[a]

	Influent		*Effluent*		*Removal Efficiency*
Parameter	*Concentration, mg/L*	*Waste Load,[b] kg/MT*	*Concentration, mg/L*	*Waste Load,[b] kg/MT*	*% Based on Influent*
pH	1.3[c]	-	8.8[c]	-	-
TSS	134	0.11	24	0.020	82
TDS	10,200	8.5	5,120	4.3	50
Pb	76	0.063	0.41	3.4×10^{-4}	99.5
Sb	8.5	0.0071	0.70	5.8×10^{-4}	92
Zn	0.53	4.4×10^{-4}	0.03	2.5×10^{-5}	94
As	0.024	2×10^{-5}	0.03	2.5×10^{-5}	0
Cu	0.41	3.4×10^{-4}	0.03	2.5×10^{-5}	93
Cd	0.83	6.9×10^{-4}	0.005	4×10^{-6}	99
Fe	-	-	-	-	-
$SO_4^=$	6,600	5.5	2,500	2.1	38

[a] *Source: modified from Table VII-15 of Ref. 35.*

[b] *Units are kilograms per metric ton of lead sent to secondary smelting.*

[c] *In pH units.*

Appreciable fractionation of lead, antimony, and arsenic occurs (Table 2.23). Before lime neutralization the wastewater contains lead, antimony, and arsenic in the ratios 1:0.12:0.00032. After lime neutralization, the effluent ratios are 1:1.7:0.073. The relative concentrations of antimony and arsenic have increased by factors of 15 and 200, respectively. The data show that for the particular plant considered, lime neutralization removes 99.5% of the lead, but only 91.8% of the antimony and none of the arsenic. Also, one notes that for this plant 99.4% of the cadmium is removed.

A recent detailed study of another plant using a lime-plus-flocculant treatment of wastewater also supports this fractionation effect. The results of this study were that the removal percentages for lead, cadmium and copper were 95%, 73%, and 75%, respectively, and that essentially none of the antimony was removed.[39]

From results such as these, influents and effluents for typical exemplary battery breaking operations can be developed. Table 2.24 indicates typical influents and effluents for lime wastewater treatment in terms of both kg/MT of lead and kg/day per MWh.

It is to be emphasized that the effluent loadings shown in Table 2.24 represent those of typical exemplary battery breaking operations. As of 1975-1976, many of the operations do not have such low wastewater effluent discharges;[35] their discharges would be better represented by the influent loadings given in the table. However, it is assumed that by the 1990s, most or all battery breaking operations will comply with effluent limitation regulations, which give effluent loadings similar to those in the table. The guidelines proposed by the EPA[35] are based on the parameters for exemplary plants.

Table 2.24. Influent and Effluent Aqueous Emissions from Typical Exemplary Battery Breaking Operations[a]

	Influent Wastewater Load		*Effluent Wastewater Load, Lime Treatment*	
Parameter	*kg/MT*	*(kg/day)/MWh*	*kg/MT*	*(kg/day)/MWh*
TSS	0.231	2.2×10^{-3}	0.025	2.4×10^{-4}
Oil, Grease[b]	0.0736	7.0×10^{-4}	0.01	9.6×10^{-5}
Pb	0.0325	3.1×10^{-4}	1.5×10^{-4}	1.4×10^{-6}
Sb	0.0366	3.5×10^{-4}	8×10^{-4}	7.6×10^{-6}
As	0.00113	1.1×10^{-5}	1×10^{-5}	9.6×10^{-8}
Cu	0.00229	2.2×10^{-5}	3×10^{-5}	2.9×10^{-7}
Zn	9.8×10^{-4}	9.2×10^{-6}	1×10^{-4}	9.6×10^{-7}
Cd	9.6×10^{-4}	9.2×10^{-6}	1×10^{-5}	9.6×10^{-8}
Ni	3.0×10^{-4}	2.9×10^{-6}	5×10^{-5}	4.8×10^{-7}
$SO_4^=$	14.9[c]	0.14	5.7[d]	0.055

[a] *Modified from Table VIII-14 in Ref. 35.*

[b] *From Table V-19, Ref. 35.*

[c] *Average value for four plants (Tables V-15 through V-19, Ref. 35).*

[d] *Obtained as 14.9 × (2.1/4.4), see Table 2.23.*

Secondary Smelting and Refining

In many plants the SO_2 scrubber water and noncontact cooling water from the secondary smelting operation is treated and recycled with zero discharge. As a result the BPCTCA and BATEA proposed by the EPA is complete recycling of generated raw wastewater for the secondary smelting operation.[35] This may be too idealistic in that problems may arise, such as those associated with the treatment of leachate generated at onsite landfills (see discussion of the Resource Conservation and Recovery Act in Section 10). However, for the purposes of this document, such problems will be ignored. Thus it is assumed here that no water emissions result from the secondary smelting operations supporting a lead/acid EV fleet.

2.6.4 Solid Wastes

Battery Breaking

The solid emissions from the battery breaking operations consist mainly of solids which settle out of the waste battery acid and saw wash water before and after neutralization. As was discussed before it is assumed that by the year 2000 almost all the other solid battery components will be recycled or disposed of in the secondary smelting operation. The latter includes those cases and covers recycled directly for reuse or ground up for use as plastic or burned as fuel; posts and connectors; separator materials; and lead materials. If the cases and covers are burned as fuel, any copper in the attached posts appears in the furnace slag in the secondary smelting operation.[15]

It is likely that, due to rough treatment in the breaking operation, some fraction of the separator materials also appears in the battery wastewater and the total suspended solids. However, this will be ignored here.

Estimates of solid emissions resulting from the battery breaking operation are shown in Table 2.25. The table entries are obtained as influents minus effluents based on values in Table 2.24. The total suspended solids include particles of lead, lead sulfate and hydroxide, and other insoluble metal sulfates and hydroxides as well as separator particles and case and cover particles. In some plants, much of the lead sulfate and hydroxide particles are removed by clarification before neutralization. The sulfates and hydroxides so recovered are recycled to the secondary smelter.[15]

The lime treatment sludge entries in the table represent the equivalent dry weight of $CaSO_4$ produced by the lime added to neutralize the acid. From the entries in Table 2.25 one sees that the dry weight sludge contains 2500 ppm lead, 2700 ppm antimony, 86 ppm arsenic, 170 ppm copper, 67 ppm zinc, 73 ppm cadmium, and 19 ppm nickel. This sludge, which contains these metals as mixed sulfates and hydroxides, is potentially hazardous, especially with respect to acid rain leaching, and must be disposed of in secured landfills.

Secondary Smelting and Refining

The principal solid emissions from the secondary smelting and refining operations are blast furnace slag and SO_2 scrubber sludge. Dry baghouse dust can be returned to the furnaces for recycling. About 450 kg of blast furnace slag is produced per metric ton of lead.[26] Because of its high lead content, the reverberatory furnace slag is recycled to the blast furnace.[35]

The literature values for the sludge production rate and heavy metal content will not be used here. The literature values apply to input scrap which is only partly used battery scrap, and do not seem to be applicable to EV battery scrap. Also the energy density of standard auto batteries is lower than that assumed for EV batteries. The auto batteries are not delivered in a completely discharged state, whereas to avoid fire and shock hazard the EV batteries must be completely discharged. All these factors imply that there will be more SO_2 scrubber sludge per metric ton of lead generated from secondary smelting of used EV battery lead than is produced in present smelter operations.

Table 2.25. Solid Emissions from Typical Exemplary Battery Breaking Operations[a]

	Solid Waste Emissions, Lime Treatment	
Parameter	*kg/MT*	*(kg/day)/MWh*
TSS[b]	0.206	2.0×10^{-3}
Oil, Grease	0.0636	6.0×10^{-4}
Pb	0.0323	3.0×10^{-4}
Sb	0.0358	3.4×10^{-4}
As	0.00112	1.1×10^{-5}
Cu	0.00226	2.2×10^{-5}
Zn	8.8×10^{-4}	8.2×10^{-6}
Cd	9.5×10^{-4}	9.1×10^{-6}
Ni	2.5×10^{-4}	2.4×10^{-6}
$SO_4^=$	9.2	0.085
Sludge (dry weight)[c]	13.0	0.120

[a] *Entries in table are influents minus effluents given in the preceding table.*

[b] *The total suspended solids values probably include particles of lead sulfate and hydroxide and other insoluble metal sulfates and hydroxides as well as separator and case particles. In any case, they represent suspended solids before the addition of lime.*

[c] *Sludge weight is given as equivalent dry weight of $CaSO_4$ produced from the added lime.*

One can estimate the amount of SO_2 sludge produced as well as its lead, antimony, and arsenic content as follows: (1) assume that the EV batteries are scrapped in the completely discharged state and that essentially all the lead sulfate produced during discharge remains on the plates; (2) calculate the ratio of sulfur to lead delivered to the secondary smelter in the form of used battery plates (see Appendix B for details). It is also assumed that 90% of the SO_2 is captured in the lime/limestone scrubber[38] as $CaSO_3$ and that sludge is 30% solid. The metal content of the sludge is calculated assuming that the baghouse filters are 99% efficient at removing particulates[23] and that there is no fractionation among lead, antimony and arsenic.

The results (Table 2.26) indicate that 4.2 kg/day per MWh of slag is produced which contains 1.8% lead.[26] This slag is in the form of large, hard chunks of silicates. Since the lead is not easily solubilized, this material is not considered potentially hazardous.[26]

Also 3.5 kg/day per MWh of $CaSO_3$ sludge containing 30% solids is produced. On a dry weight basis this sludge contains 1800 ppm lead, 38 ppm antimony and 0.6 ppm arsenic. Another report[26] gives higher concentrations of lead and antimony as well as 0.03% Cd in present-day SO_2 scrubber sludge from secondary smelting operations.

Table 2.26. Solid Emission Flow Rates from Secondary Smelting[a]

Species	*Amount (kg/day)/MWh*
Slag[b]	4.3
Pb	0.077
SO_2 sludge[c]	3.5
$CaSO_3$[d]	1.1
Pb[e]	0.0018
Sb[f]	3.6×10^{-5}
As[f]	6.1×10^{-7}
H_2O	2.4

[a] *Calculated from the data in Table 2.22 and a baghouse filter efficiency for Pb, Sb, and As of 99%.*

[b] *Assumes 450 kilogram slag containing 1.8% Pb is produced per MT of lead (Ref. 26).*

[c] *Assumes sludge has 30% solids (Ref. 26).*

[d] *Amount = (120/64) × (0.63 - 0.063).*

[e] *Assumes that of the 1% of the uncontrolled lead emissions which get through the filter, 70% ends up in the sludge and 30% is emitted into the air (see footnote b of Table 2.22).*

[f] *Assumes no fractionation.*

It should be recalled that arsenic is followed throughout the cycle even though it exists in lower concentration in lead than do some contaminants, such as tin (grid metal contains 0.02% to 0.65% tin).[7] The reason is that such contaminants are inessential and can be reduced if desired, whereas arsenic is added deliberately.

2.6.5 Impact on Existing Industry

Battery Breaking

The impact on the existing battery breaker industry of operation at a level sufficient to support an electric vehicle fleet, growing at 25% per year is shown in Table 2.27. The size of the new battery breaking industry is given in metric tons of lead/day throughput, as a percent of existing (1973) capacity (in 1973, 921 MT/day of lead, as battery plates, were recycled[20]), and in terms of large new battery breakers required.

Several things are evident from the table. By the 1990s, the battery breaking capacity would have to increase considerably. In particular, by the year 2000, an industry capacity increase of 78%, or 13 large new breakers would be required. (It is assumed that each breaker can process 20,000 MT of lead equivalent per year.[35])

Table 2.27. Impact of EV Fleet on the Existing Battery Breaking Industry[a]

	Year		
	1990	*1995*	*2000*
Electric Vehicle Fleet			
Number of Vehicles (thousands)	250	870	3,000
Megawatt Hours	6,200	22,000	75,000
Battery Breaking			
MT Pb/day	60	210	720
% of Existing Capacity[b]	6.5	23.	78.
Number Required of Large New Breakers[b]	1.1	3.7	13.
Emission			
Liquid Emissions, kg/day			
TSS	1.5	5.2	18.
Oil, Grease	0.60	2.0	7.2
Pb	0.0090	0.032	0.11
Sb	0.048	0.17	0.53
As	0.00060	0.0020	0.0072
Cu	0.00018	0.0063	0.022
Zn	0.0060	0.020	0.072
Cd	0.00060	0.0020	0.0072
Ni	0.00030	0.0100	0.037
$SO_4^=$	350	1,200	4,200
Solid Emissions, kg/day			
TSS	1.5	5.2	2.8
Oil, Grease	0.60	2.2	7.2
Pb	2.0	6.7	23.
Sb	2.2	7.5	25.
As	0.067	0.23	0.80
Cu	0.014	0.47	1.6
Zn	0.053	0.28	0.63
Cd	0.056	0.20	0.68
Ni	0.015	0.052	0.18
Sludge (dry weight) (kg/day)	780	2,600	9,300

[a] *Source: Tables 2.24 and 2.25, and Section 2.2.*

[b] *In 1973 56.5% of the total secondary lead production of 594,800 metric tons came from battery scrap (Reference 20). The average secondary smelter size, which includes a battery breaker was taken to be 20,000 metric tons of lead/yr, (Reference 35). It was assumed that in these new smelter-breaker operations, battery scrap is the only source of lead.*

In general, the emissions become appreciable during the last decade of this century. If lime neutralization is used, by the year 2000, 9.3 metric tons of $CaSO_4$ sludge (dry weight) containing 23 kg Pb, 25 kg Sb, 0.8 kg As, 1.6 kg Cu, 0.63 kg Zn, 0.68 kg Cd, and 0.2 kg Ni would be produced each day. This material would have to be disposed of in secured landfills. The amount of lead and antimony going out with the wastewater is smaller, about 0.11 and 0.53 kg/day. However, 4.2 MT/day of sulfate is released into the receiving waters.

Recycling of the sludge to recover the heavy metals would be desirable at least from an environmental standpoint. However, it is problematic due to the large amounts involved. Neutralization of the wastewaters with ammonia or lye is advantageous in this respect in that much smaller amounts of sludge are produced. Such methods, which are presently used to some extent,[35] may become more widespread in the future.

Secondary Smelting and Refining

Table 2.28 gives the impact on the secondary smelting industry of a SOA lead/acid EV fleet growing at 25%/year to 3×10^6 Evs by the year 2000. By then the industry would have to increase by 44%, or 13 large new secondary smelters each with a capacity of 20,000 MT of lead/year would be required. The percent increase of existing capacity, 44%, is about one-half the value given for battery manufacturing for the year 2000. The reason is that about half the lead recycled through secondary smelters comes from batteries and half from other sources. As indicated in the table, the impact of the SOA-lead/acid battery on secondary smelting capacity would not be felt until the 1990s, when one or more large new secondary smelters would be required.

Table 2.28. Impact of EV Fleet on Existing Secondary Lead Smelting[a]

	Year		
	1990	*1995*	*2000*
Electric Vehicle Fleet			
Number of Vehicles (thousands)	250	870	3000
Megawatt Hours	6,200	22,000	75,000
Secondary Smelting			
MT Lead/day	60	280	720
% of Existing Capacity	3.7	13.	44.
Number of Large New Smelters Required	1.1	3.6	13.
Effluents			
Controlled Air Emissions (kg/day)			
Particulates	330	1100	3800
Pb	75	260	900
Sb	1.5	5.2	18.
As	0.027	0.092	0.32
SO_2	390	1400	4700
Solid Emissions			
Slag, MT/day	27	94	320
Pb, kg/day	480	1700	5800
SO_2 Sludge, MT/day	22.	76.	260.
$CaSO_3$, MT/day	6.5	23.	80.
Pb, kg/day	12.	41.	140.
Sb, kg/day	0.23	0.83	2.8
As, kg/day	0.0042	0.014	0.040

[a] *Source: Tables 2.22 and 2.26, and Section 2.2.*

[b] *Total 1973 secondary lead production was 594,000 MT of lead. The average secondary smelter size was taken to be 20,000 MT/year (References 20 and 35).*

The controlled air emissions from the secondary smelters will increase appreciably, rising to 3800 kg particulates containing 900 kg Pb, 18 kg Sb, 32 gm As, and 4700 kg SO_2 per day in the year 2000. Furthermore, most secondary smelters are located in urban areas so as to be close to their sources of supply of used batteries.[35] However, most of the particulate and metal emissions represent fugitive dust emissions which are uncontrolled. The controlled furnace emissions represent only 6% of the total (Table 2.22). Thus, changes in housekeeping practices could well reduce the total air emissions.

The slag emission is large: 320 MT containing 5.8 MT of lead would be produced each day by the year 2000. However, the lead solubilizes to less than 1 ppm from the slag,[26] so it is not considered hazardous. The production of $CaSO_3$ sludge resulting from the wet scrubbing of the furnace gases rises to 260 MT containing 140 kg Pb, 2.8 kg Sb and 40 g As each day by the year 2000. The metals in this material are appreciably soluble.[26] As a result, the material is potentially hazardous and must be disposed of in an environmentally satisfactory way. Again it should be noted that these outputs, although considerable, are only 44% of the present output of effluents. Furthermore, the calculations of these effluents are based on the control methods available today and make no allowance for technological improvements. Thus, the effluent values given in the table are the amounts which would be emitted if no changes in the present (1970s) control technology or operations took place.

2.7 SUMMARY

1. Impacts that the state-of-the-art lead/acid battery-powered electric vehicles would have on the lead/acid battery industry were assessed. The market penetration was assumed to grow at a rate of 25% per year and to reach a total of three million lead/acid battery-powered electric vehicles on the road by the year 2000. Assessments were also made for per megawatt-hour of installed capacity, assuming a one-megawatt hour requirement for each 40 electric vehicles. The assessment included consideration of resource requirements, industrial growth, and effluents to the environment from various phases of the total battery cycle.

2. For the assumed scenario, by the year 2000:

   a. a 66% increase in lead mining and milling will be required,

   b. a 55% increase in primary lead smelting and refining will be required,

   c. a 44% increase in secondary lead smelting and refining will be required,

   d. a 94% increase in battery manufacturing facilities will be required, and

   e. a 78% increase in the battery breaking operations will be required.

3. For the assumed scenario, by the year 2000 projected increases in the air-borne emissions, assuming present day control technology, include

   a. 3.3 MT/day of lead in fugitive dust from mining and milling,

b. 1.3 MT/day of lead in fugitive dust and controlled blast furnace emissions from primary smelting and refining.

c. 0.1 MT/day of lead in fumes and particulates from battery manufacturing.

d. 4.7 MT/day of sulfur dioxide and 3.8 metric tons/day of particulates containing 0.9 metric ton/day of lead from secondary smelting and refining.

4. For the assumed scenario, by the year 2000, projected increases in the solid waste assuming present day control technology, include

a. From mine and milling -
17,000 MT/day of mill tailings containing 16 MT of lead and 0.28 MT of cadmium.

b. From primary smelting and refining -
87 MT/day of sludge containing 12 MT of lead and 0.6 MT of cadmium.

c. From battery manufacture -
1,700 MT/day of lime sludge containing 0.4 MT of lead and 0.008 MT of antimony.

d. From battery breaking 9.3 MT/day of sludge containing 23 kilograms of lead and 25 kilograms of antimony.

e. From secondary smelting and refining -
260 MT/day of sludge containing 0.14 MT of lead.

5. For the assumed scenario, by the year 2000, projected increases in water emissions, assuming present day control technology, include

a. 490 kilograms/day flotation chemicals and 18 kg/day lead from lead mining and milling.

b. 0.43 kilograms/day of lead and of cadmium and 150 kilograms/day of sulfate ions from lead smelting and refining.

c. 22 kilograms/day of lead and 600 MT/day of sodium sulfate (if and only if lye neutralization were used exclusively) from battery manufacturing by the wet process. Dry process emissions are projected to be larger by a factor of 2 to 3.

d. 0.11 kilograms/day of lead and 0.53 kilograms/day of antimony and 4.2 MT/day of sulfates from the battery breaking industry.

6. Stibine and arsine gases will be generated during charging of the batteries. These gases are unstable and will decay to oxides in the form of dust. If EVs are charged in confined areas, such as home garages, accumulations of antimony and arsenic oxide dusts on garage walls and floors are expected to occur over a period of time.

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3. EFFLUENTS FROM THE NICKEL/ZINC BATTERY CYCLE

ABSTRACT

The Ni/Zn battery cycle, from mining and milling of the raw materials through recycling of the scrap batteries and disposal of wastes, is described. Material flows and emissions for various phases of the total cycle are estimated for per megawatt hour of installed capacity and for a scenario of 8 × 10^6 electric vehicles on the road by the year 2000.

3.1 INPUT PARAMETERS

Nickel/zinc (Ni/Zn) batteries have emerged as the leading candidates for near-term EV transportation applications; the major conditions for successful commercialization remain the improvement of battery lifetime and the cost reductions necessary for major market penetration. No specific design has been chosen for electric vehicle applications of the Ni/Zn system and data are lacking on various aspects of the system itself. Therefore, in order to estimate the effluents resulting from large-scale deployment of these batteries we have utilized a synthesis* of battery compositions based on the 1976-1977 design and cost studies prepared for Argonne National Laboratory by Eagle-Picher Industries, Inc.,[1] Energy Research Corp.,[2] Gould Inc.,[3] and Yardney Electric Division.[4] Because many details of the various battery designs are of a proprietary nature, the compositions given in Table 3.1 are approximations only, but should be adequate for the purposes of this environmental assessment. The exact materials that will be used in the cases and separators, as well as various proprietary additives, are difficult to identify at this time; we shall assume that the plastic components will be polypropylene (assumed for the SOA lead-acid battery in Sec. 2.4.1). Miscellaneous materials include inorganic materials such as zirconia or asbestos that may be combined with organics to produce composite separators, assorted hardware, nylon, etc.

To quantify the materials flow in the Ni/Zn cycle, additional parameters are needed, such as battery lifetime and average annual growth rate. A Ni/Zn powered EV is expected to have a 250-km (150-mile) range.[5] For urban EVs,[5] with an expected annual traveled distance of about 16,000 km (9600 miles) and an expected lifetime of 600 cycles, the total battery lifetime would be 9.4 years (about the average ICE vehicle lifetime). An estimate** of average annual growth rate for EVs powered by Ni/Zn batteries has been based on the following scenario:

Year	Total Ni/Zn-Powered EVs in Service
1985	85,000
1990	410,000
2000	8,000,000

**Studies by ANL staff, 1979.*

***Developed by ANL staff, 1979.*

Table 3.1. Approximate Composition of the Ni/Zn Battery for EV Use

Material	*Composition*	
	Wt%	*MT/MWh*[a]
Ni	31	4.03
ZnO	23	2.99
Co	1.6	0.208
Cu	0.70	0.0909
Electrolyte	32[b]	4.16
Plastic (case, separators, etc.)	7.0	0.909
Miscellaneous Materials and Additives	5.0	0.649

[a] *Based on 77 Wh/kg.*

[b] *30 weight % aqueous KOH solution.*

As shown in Section 2 of this report, a simple exponential growth curve is reasonable to assume for this stage of development; with the above scenario we obtain a growth rate of about 30%, which is only slightly faster than the rate (25%) for the SOA lead acid battery (Sec. 2.1).

Based on arguments developed in Section 2.1, a 10% loss to the environment of inorganic battery components will be assumed as a conservative estimate. The fate of the organic components of battery (cases, separators, etc.) is not clear; we shall assume (as was done for the SOA lead-acid battery) that about 25% of the material is recycled and the remainder is used as fuel, with no more than 10% ending up in landfills.[6] The nature of this organics scenario has only a small effect on the overall cycle.

3.2 MATERIAL FLOW RATES

Overall daily flow rates of materials for the Ni/Zn battery cycle can be calculated using the methods given in Appendix A and expressed as kg/day per MWh installed; the results of these calculations are given in Table 3.2. Under "New Batteries," the column headed "Total" gives the material flow for new batteries; the "Loss" column relates to the assumed 10% loss of installed new batteries; the "Installation" column presents the flow rates calculated using the relationships developed in Appendix A; the final column, "Removal of Spent Batteries," indicates the material removed from the cycle as fully discharged, spent batteries.

3.3 PRIMARY PRODUCTION OF INPUT MATERIALS

3.3.1 Zinc

Mining and Milling

Zinc-containing ores are widely mined throughout the United States with about 30% of present extraction concentrated west of the Mississippi River. Lead, and some copper, ores are often associated with zinc; thus, the wastes associated with zinc mining and milling given in

Table 3.2. Ni/Zn Battery Component Flow Rates, (kg/day)/MWh

Battery Component	New Batteries			Removal of Spent Batteries
	Total	Loss	Installation	
Ni	3.88	0.35	3.53	0.21
ZnO	2.88	0.26	2.62	0.16
Co	0.20	0.02	0.18	0.011
Cu	0.088	0.008	0.080	0.005
Electrolyte	4.50	0.86	3.64	0.22
Plastic	0.88	0.08	0.80	0.048
Miscellaneous	0.63	0.06	0.57	0.034
TOTALS				
Dry Weight	9.91	1.04	8.87	0.53
Battery	13.1	1.64	11.42	0.69

this section are probably overestimates, because they include some wastes that should be chargeable to lead mining and milling. In 1974, U.S. ore production amounted to 18.1 million MT;[6] U.S. zinc production from domestic ores accounted for 315,000 MT (1.7%) of this total.[7] Processing after ore removal involves crushing, grinding, and separation by flotation.

Air Emissions. The chief air-emission sources during mining and milling operations are fugitive dusts resulting from blasting, ore handling, ore crushing, and exposed tailings.[8] Emissions from these operations have been estimated[8] at about 0.2 lb per ton (0.1 kg/MT) of zinc mined. A more recent study[9] indicates fugitive dust emissions of 0.2 lb/ton zinc from mining and ∿ 2 lb/ton ore emissions during crushing. Assuming current control efficiencies, we can estimate that the total particulate emissions amount to about 0.7 kg/MT zinc. Metal emissions can be estimated on the basis of crude ore compositions for a typical Missouri mine: 4.4% Pb, 2.6% Zn, 0.012% Co, 0.017% Ni, 0.012% Cd, and 0.16% Mn.[6] Material flows based on these estimates are shown in Table 3.3.

Water Emissions. Mine water drainage is not considered in the wastewater estimates, rather only water from mill discharges. EPA has estimated that about 3.2 MT wastewater is produced in the milling of one metric ton of ore;[10] from this estimate and zinc metal production data (see Sec. 3.3.1), about 200 MT wastewater is produced per metric ton of zinc metal. EPA standards for zinc mill wastes[11] can be used to calculate the emissions in the wastewater; in reality, these standards probably will be more stringent in the time frame of the EV industry. Thus, the estimates given in Table 3.3 are probably conservative.

Solid Wastes. Solid wastes from the mining and milling processes consist chiefly of tailings, as much of the waste rock from mining is used for mine backfilling. Bendersky et al.[6] reported that, in 1974, 12 million MT of zinc mill tailings were produced (dry basis); from this and the 1974 U.S. production data for zinc we can estimate that about 40 MT of tailings were produced per metric ton of zinc metal. Bendersky et al.[6] indicated the metallic composition of the tailings for a Missouri mine to be 0.093% Pb, 0.018% Cu, 0.083% Zn, 0.0045% Co, 0.0063% Ni, 0.0016% Cd, and 0.18% Mn. The estimated daily flow for mill tailings per megawatt-hour of installed batteries are given in Table 3.3.

Table 3.3. Estimates of Emissions from Zinc Mining and Milling

Emissions	*(kg/day)/MWh*
AIR	
Particulates	1×10^{-3}
Pb	6×10^{-5}
Zn	4×10^{-5}
Co	2×10^{-7}
Ni	2×10^{-7}
Cd	2×10^{-7}
Mn	2×10^{-6}
WATER	
Wastewater	400
TSS	8×10^{-3}
Zn	2×10^{-4}
Pb	1×10^{-4}
Hg	4×10^{-7}
Cd	2×10^{-5}
Cn	4×10^{-5}
Cu	6×10^{-5}
SOLIDS	
Mill Tailings	80

Smelting and Refining

Three processes are currently employed in the production of primary zinc: about 45% of domestic zinc is produced by the electrolytic process; 35% is accounted for by the electrothermic process; the remainder is from the vertical retort process. Almost all the new zinc plants use the electrolytic process.[12] In this process, the ore concentrate, after roasting, is leached with spent sulfuric acid electrolyte; manganese dioxide is added to oxidize ferrous ions, and the purified solution is plated out on aluminum cathodes in a flowing electrolysis cell, producing a high-purity zinc product.

Water Emissions. The typical electrolytic zinc plant[13] produces about 5.3 m^3 of wastewater per metric ton of zinc product, which generates about 9 kg of solids. The roasting of the zinc ores, in addition, produces about 4.7 m^3 of acid-blowdown slurry per metric ton of zinc product, containing about 17 kg of solids. Treatment of these wastewaters produces sludges with a combined analysis of: Cd, 802 ppm; Cr, 44 ppm; Cu, 2,510 ppm; Hg, 22 ppm; Mn, 8,740 ppm; Pb, 15,300 ppm; Se, 66 ppm; and Zn, 220,000 ppm.

Pyrometallurgical zinc manufacturing plants[13] produce (typically) 3.5 m^3 of slurries per metric ton of zinc product from gas cleaning operations, with a solids content of about 94 kg. The acid-blowdown slurries from roasting amount to 13.1 m^3, containing 90 kg of solids, per metric ton of zinc product. Retorting operations produce an additional 10 kg of solids per metric ton of zinc product, (these solids are the "blue powder" zinc recovered from the wet

scrubber). Analysis of the combined sludges is (typically): Cd, 822 ppm; Cr, 31 ppm; Cu, 540 ppm; Pb, 2,920 ppm; Se, 46 ppm; Zn, 306,900 ppm; and Hg, 9 ppm.

To estimate the wastewater emissions from the facilities we have used the 30-day average effluent limitation guidelines[14] for primary zinc smelters utilizing best practicable control technology currently available; these probably yield conservative estimates for facilities that could be operational in the 1990s. Even with these estimates, the quantities emitted in wastewaters are very small (see Table 3.4).

Table 3.4. Wastewater Emissions from Primary Zinc Production

Constituent Species	*Emissions*	
	Guideline (kg/MT)[13]	*Flow (kg/day)/MWh*
TSS	0.21	4.9×10^{-4}
As	0.0008	1.9×10^{-6}
Cd	0.004	9.3×10^{-6}
Pb[a]	0.004	9.3×10^{-6}
Se	0.04	9.3×10^{-5}
Zn	0.04	9.3×10^{-5}

[a] *Not explicitly listed in 40 CFR 421.83, but assumed to be the same value as Cd.*

Air Emissions. To calculate air emissions we have assumed that, when utilization of Ni/Zn batteries is widespread enough to impact the smelting industry, the chief means for primary zinc production will be the electrolytic process; uncontrolled particulate emissions will, in this case, amount to about 123 kg/MT of zinc metal.[15] Sulfur oxides would amount to 1100 kg/MT of zinc metal.[15] Particulate emissions from electrolytic zinc plants have been reported to be nil;[9] particulates lost from the roasting process typically are less than 1% of the feed.[9] Because nearly all the uncontrolled particulate emission would result from roasting, we shall estimate the particulate emission distribution using elemental distributions characteristic of roasting operations.[9] This approach, coupled with an assumed control efficiency[16] for particulate removal of 99.85%, leads to the values shown in Table 3.5 for particulate emissions. The SO_2 emissions (Table 3.5) were estimated using the 40 CFR standard for zinc roasters[17] (0.065 volume percent SO_2 in discharge gases) and reported off-gas volumes[18] from smelting plants (49 to 85 cfm per ton per day of zinc). These values are probably conservative; control technology should be significantly improved in the 1990s, and more stringent standards are likely.

Solid Emissions. Assuming that the electrolytic process for zinc smelting and refining will be the primary source of EV battery zinc, the resulting solid wastes requiring land disposal or storage will be about 26.1 kg of sludge per metric ton of zinc produced.[19] These sludges will have the composition previously discussed for electrolytic waste sludges (page 70, paragraph 1). With these values, the amounts of solid hazardous material would be as shown in Table 3.6. Much larger amounts of solid wastes are generated by the smelting process, but the majority of this solid waste is recycled for recovery of metals.

Table 3.5. Particulate and SO_2 Emissions from Primary Zinc Production

Constituent Species	Estimated Emissions	
	kg/MT Zinc Production	Flow (kg/day)/MWh
As	0.0003	7×10^{-7}
Cd	0.011	2.5×10^{-5}
Pb	0.0014	3.3×10^{-6}
Sb	0.0003	7×10^{-7}
Zn	0.17	4.0×10^{-4}
SO_2	42	0.096

Table 3.6. Solid Emissions from Primary Zinc Production

Constituent Species	Solid Emissions	
	kg/MT Zinc	Flow (kg/day)/MWh
Total Sludge	26.1	0.061
Cd	0.056	1.3×10^{-4}
Cr	0.0011	2.6×10^{-6}
Cu	0.066	1.5×10^{-4}
Hg	0.00057	1.3×10^{-6}
Mn	0.023	5.3×10^{-5}
Pb	0.40	9.3×10^{-4}
Se	0.0017	3.9×10^{-6}
Zn	5.7	0.013

Impact on Existing Zinc Smelting Industry

Unlike the situation for the nickel metals industry, the deployment of large numbers of electric vehicles powered by nickel/zinc batteries would have a miminal impact on the primary zinc industry; by the year 2000 only two additional smelters (100,000 MT annual production each) would be required to produce the zinc required by the battery industry. While the absolute amounts of effluents produced from these new smelters would be quite large, they will be undoubtedly smaller than levels indicated in Table 3.7, because the values presented there are based on current technology and standards. It is likely that improving technology and revised standards will result in a reduction of the effluents from the primary zinc industry.

3.3.2 Nickel

Mining and Milling

In the United States nickel is currently extracted from only one mine, in Oregon; the 1975 production[7] was about 15,000 MT (as ferronickel), which is neither appropriate for use in EV

Table 3.7. Impact of Ni/Zn Batteries on the Zinc Industry

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWh Installed Batteries	2,125	10,250	200,000
Daily Zinc Production, MT	4.9	24	464
% of Existing Production Capacity[a]	0.034	0.17	3.2
New Smelters Required[b]	0.02	0.08	1.6
Environmental Impact			
Total particulate emissions, MT/day	0.003	0.015	0.28
Total water emissions, MT/day	900	4,300	85,000
Total solid emissions, MT/day	170	820	16,000
Total SO_2 emissions, MT/day	0.20	0.98	19

[a] *Zinc production (world-wide) in 1975 was 5.01 × 10^6 MT (ANL-K77-3558-1 Vol. 1, Table III-10).*

[b] *Assumes annual production of 100,000 MT/yr per smelter.*

batteries nor in sufficient quantity to supply the projected nickel-zinc battery industry. The industry must therefore consider other sources, the most likely being Canadian ores.[6]

Little information is available concerning emissions related to mining of nickel ores in Canada; however, one can make crude order-of-magnitude estimates based on published information relating to the INCO Sudbury nickel operations.[19,20] From these sources, one can estimate that on the order of 150 MT of solids (requiring either[18] landfill disposal or return as fill to excavated mines) would be produced per metric ton of nickel product. It is assumed that any resulting landfill areas will be designed so that seepage and effluents from the landfill will meet appropriate standards for water quality[11] (dependent largely on local rainfall conditions). Wastewater flows from the mining and milling processes have been estimated in conjunction with smelting and refining operations and will be presented in Table 3.8.

Order-of-magnitude emission estimates for nickel mining and milling in relation to the Ni/Zn battery cycle are as follows; they should in no way be construed as being definitive.

Tailings	600 (kg/day)/MWh
Wastewater*	1300 (kg/day)/MWh

Smelting and Refining

Because the nickel smelting and refining process required for Ni/Zn batteries is likely to occur outside the United States, there would be no direct impact on the U.S. environment. However, for the purpose of this report, we have assumed that the Canadian effluents would be included in the total environmental emission from the Ni/Zn battery cycle. Data regarding

**The estimation method for liquid effluents is given on page 82. This value includes wastes from smelting and refining as well.*

Table 3.8. Estimated Emissions from Nickel Smelting

Emissions	*kg/MT Nickel*	*Flow (kg/day)/MWh*
	AIR[a]	
SO_2	780	3.0
Total Particulates	21	0.081
Ni	2.4	0.0093
Co	0.06	0.0002
Pb	0.2	0.0008
	WATER[b]	
Wastewater	340,000	1,300
TSS	5.1	0.020
Ni	0.2	0.0008
Cu	0.07	0.0003
Fe	0.2	0.0008

[a] *Assumes conformance to 40 CFR standards.*

[b] *Includes emissions for mining, milling, smelting, and refining.*

emissions from the Canadian plants are difficult to obtain; for example, there is much information regarding the effects of the INCO facility at Sudbury, Ontario, on the surroundings, but little quantitative data are available on actual facility emissions. Therefore, the estimates presented in the following sections are based on limited information and should be considered no more than preliminary guides until better values are obtainable.

Air Emissions. The major air emissions resulting from the smelting of nickel ores (chiefly pentlandite [$(Fe, Ni)_9S_8$]) are SO_2 and particulates. Based on Canadian smelter production data[7] and values for SO_2 and particulate emissions from INCO's Copper Cliff Facility[21] one can estimate that the 1977 emission level of SO_2 from such facilities was about 7.8 MT SO_2 per metric ton of nickel product. Associated with these SO_2 emissions were approximately 83 kg of particulates per metric ton of nickel. However, these emission levels are too high to conform with the 0.065 v/o SO_2 U.S. stack-gas standard and the 50 mg/m^3 standard for particulates from other types of smelters.[22] Greater dilution or more efficient particulate and SO_2 removal would serve to reduce the air emissions; we shall assume that the latter course would be taken.

Using particulate emission data for the INCO Copper Cliff Facility[21] one can estimate that the daily flue gas volume is about 1.7×10^8 m^3. Assuming the daily flue-gas output is not changed significantly by the introduction of new emission control devices, we can estimate that meeting the 40 CFR standards[22] would yield the following air emissions: 0.78 MT SO_2 and 21 kg particulates per metric ton of nickel produced. These values amount to a 90% reduction in SO_2 output over 1977 levels and a 75% reduction in particulate output.

The composition of particulates from such a facility can be estimated from data presented by Hutchinson and Whitby[23] for the Copper Cliff Smelter at Sudbury, if one assumes the control devices do not materially affect the metallic particulate distributions. On the basis of these data, emission levels have been estimated as shown in Table 3.8.

Water Emissions. Estimation of water emissions resulting from nickel production becomes even more of a crystal-gazing procedure than that for the air emissions. The Copper Cliff treatment area (∿ 8,300 acres) requires a water treatment facility with a 60-million gpd capacity to treat effluents from the mines, copper refinery, and iron ore recovery plant, as well as the nickel production facilities.[19] The design goals of this plant are 15 ppm TSS, 0.5 ppm Ni, 0.2 ppm Cu, and 0.5 ppm Fe, which are somewhat lower levels than proposed by EPA. The wastes emanating from the facility are chargeable to other products besides the nickel being produced; as a conservative estimate we shall assume that about two-thirds of the wastes are due to nickel production. In addition, we shall assume that the capacity of the water treatment plant is 10% greater than the probable water flow. Therefore, we shall choose 40 million gpd as the flow rate through the treatment system; this corresponds to about 340 MT wastewater per metric ton of nickel. Assuming the Canadian emission factors for the Copper Cliff treatment facility, the amounts of those materials emitted after the wastewater has been treated can be calculated (see Table 3.8). The values presented include emissions for mining, milling, smelting and refining of nickel from the sulfide ores.

Solid Emissions. Insufficient information is available from which to estimate solid emissions for the nickel production processes.

Impact on the Nickel Industry

It is apparent from the estimates shown in Table 3.9 that development of a mature Ni/Zn battery industry would have a significant impact on nickel utilization. Efficient recycling of nickel will become a necessity. The existing ferrous and nickel-alloy industries currently account for about 80% of primary nickel demand; competition for nickel from an EV industry could result in increased prices, making the expansion of the recycling industry attractive. Emissions from nickel refining are quite large as compared with other components of the Ni/Zn battery; it appears that significant improvement in the industry's control technology will be required to reduce the absolute quantities of pollutants released to the environment.

Table 3.9. Impact of Ni/Zn Batteries on the Nickel Industry

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWH Installed Batteries	2,125	10,250	200,000
Daily Nickel Production, MT	8.2	40	780
% of 1975 World Production[a]	0.5	2.5	48
Environmental Impact			
Total particulate emissions, MT/day	0.2	0.8	20
Total water emissions, MT/day	2,800	14,000	270,000
Total SO_2 emissions, MT/day	6	30	610

[a] *1975 world production was ∿ 582,000 MT.*

3.3.3 Cobalt

Virtually all the cobalt used in the United States is of foreign origin; U.S. refinery production in 1977 amounted to ∿ 340 MT. This is clearly inadequate for the needs of the nickel-based battery industry. We cannot at this time provide estimates of the emissions from the mining, milling, smelting and refining processes; however, their impacts on the United States will be minimal at worst.

3.3.4 Potassium Hydroxide

Manufacture of potassium hydroxide (caustic potash; KOH) is part of the U.S. chlor-alkali industry. The annual capacity[24] of U.S. producers in 1973 was about 280,000 MT of caustic potash (about one-fifth the production of sodium hydroxide). Currently about two-thirds of the U.S. chlor-alkali production is accounted for by brine electrolysis using diaphragm cells, and it is expected that nearly all future production in the United States will be chiefly by this process;[24] therefore we have chosen to make our environmental assessments based on the diaphragm cell technology.

In the diaphragm cell process, the brines are first purified of magnesium and calcium by precipitation; after filtering, the brine is electrolyzed in a diaphragm cell. Chlorine is produced at the anode and hydrogen gas and potassium hydroxide at the cathode. The diaphragm is used to hinder back-migration of hydroxyl ions into the anode compartment. The caustic solution is subjected to evaporation for purification and salt recovery. Theoretically, for each metric ton of chlorine produced, 1.58 MT of KOH would result; however, assuming the same process efficiency[24] as for the production of caustic soda (93.5%), we estimate that 1.48 MT of KOH would be produced per metric ton of chlorine. Emission standards for the chlor-alkali industry are usually formulated relative to chlorine production.

Water Emissions. We shall assume that standards for performance by new sources will be implemented in plants producing KOH for the EV battery industry. In 40 CFR 415.65 there are stated the following standards for new sources (as 30-day average emission values): 0.32 kg of suspended solids/MT of product, 0.00004 kg of lead/MT of product, and a pH within the range 6.0 to 9.0. We have converted these values into emissions per metric ton of KOH (see Table 3.10). The quantities of pollutants emitted in the liquid waste streams should be very small.

Air Emissions. The major source of air emissions from caustic potash plants using current technology is from chlorine blow gas produced during chlorine liquifaction. Normal operation employing a caustic or lime scrubber results in an emission factor of 0.005 kg chlorine per metric ton of chlorine liquified.[24] In addition, there may be a small amount of carbon monoxide emitted with the blow gas; this could amount to 0.4 volume percent[24] of the exhaust gases. Assuming no CO absorption equipment in the control system, this would correspond to about 0.08 kg CO per metric ton of chlorine liquified. These emission factors have been converted to those appropriate for production of a MT of KOH and are given in Table 3.10. The CO emission estimate is probably a very conservative one. More stringent regulations in the future could result in significant reduction of these already small air emissions.

Table 3.10. - Estimated Emissions from KOH Production

Constituent	Emissions	
	kg/MT KOH	Flow (kg/day)/MWh
	AIR	
Chlorine[a]	0.003	4×10^{-6}
Carbon Monoxide[b]	0.05	7×10^{-5}
	WASTEWATER	
TSS	0.22[c]	3×10^{-8}
Pb	0.00003[c]	4×10^{-8}
	SOLIDS[d]	
Total Solids	$\sim$ 4	$\sim 5 \times 10^{-3}$
Pb Solids	0.06	8×10^{-5}
Mg and Ca Solids	3.7	5×10^{-3}
Chlorinated Hydrocarbons	0.1	1×10^{-4}

[a] *Assumes that caustic scrubber lowers chlorine emission.*

[b] *Assumes no emission control.*

[c] *New source performance standards, 40 CFR 415.65.*

[d] *Dry basis (sludge is 30% solids).*

Solid Emissions. In 1974 about 6 kg of dry solid wastes destined for land disposal were produced by the chlor-alkali industry per metric ton of chlorine;[24] this amount includes asbestos and mercury, which should not be emitted from plants producing caustic potash for EV use. Based on data for a typical chlor-alkali plant[25] we can estimate that the total dry solid wastes would amount to about 4 kg/MT chlorine. Solid emission estimates based on these data are shown in Table 3.10; these values represent current technology and will probably be conservative in relationship to plants operating in the 1990s.

Impact on Existing Chlor-Alkali Industry

An assessment of the impact of increased caustic potash production on the chlor-alkali industry is summarized in Table 3.11. The emissions, while not large, are probably an overestimate for the situation that will exist in the 1990s. The calculations indicate, however, that in the year 2000 three additional KOH plants will be required, representing a 40% expansion of the industry. However, because existing caustic potash facilities have much smaller capacities than the average caustic soda plant, one large new plant could handle the projected increased caustic potash needs quite readily.

3.4 MANUFACTURING PROCESS

3.4.1 Case and Cover

A detailed discussion of the use of plastics in battery case and cover manufacture has been given in Section 2.4.1 in discussion of the SOA lead-acid battery. The reader is referred

Table 3.11. Impact of Ni/Zn Batteries on the Chlor-Alkali Industry (Caustic Potash Production)

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWh Installed Batteries	2,125	10,250	200,000
Daily KOH Production, MT	2.9	14	270
% of Existing U.S. Production[a] Capacity	0.3	1.7	32
New KOH Plants Required[b]	0.03	0.1	3
Environmental Impacts			
Total air emissions, MT/day	0.0002	0.0007	0.014
Total water emissions, MT/day	0.0006	0.003	0.06
Total solid emissions, MT/day	0.012	0.056	1.1

[a] *Assumed annual U.S. caustic potash production of 300,000 MT (360-day year).*

[b] *Average plant is assumed to produce 35,000 MT/yr.*

to that section for a description of polypropylene production. The estimates that follow here are based on the emission information presented in that earlier section.

Emissions

The emissions presented in Table 3.12 represent a conversion of those emissions given in Section 2.4.1 (SOA lead-acid battery) for production of polypropylene cases and covers to conditions appropriate to the nickel/zinc battery industry. The reader should keep in mind that the air emissions represent uncontrolled emissions which should be amenable to further reduction by use of additional control equipment.

Table 3.12. Emissions from Polypropylene Production[a] for Ni/Zn Battery Cases and Covers

Emission Type	*Material*	*Flow, (kg/day)/MWh*
Air[b]	Particulates	1.3×10^{-3}
	Propylene	3.1×10^{-4}
Water[c]	TSS	4.9×10^{-4}
Solid[d]	Sludge	0.001 - 0.01

[a] *Processed from propylene.*

[b] *These represent uncontrolled emissions.*

[c] *Treated wastewater.*

[d] *Equivalent dry weight of sludge.*

Impact of Ni/Zn Batteries on Existing Polypropylene Industry

Table 3.13 indicates estimated production requirements for polypropylene in an expanding nickel/zinc battery industry. Impact on the existing polypropylene industry is minimal; by the year 2000, only one new plant (of current size) would be required to produce the necessary plastics. The emissions (based on current practice) shown are probably larger than would be expected or permitted in the 1990s; thus, it is likely that significant reduction of emissions from polypropylene manufacture will occur within that time frame.

Table 3.13. Impact of Ni/Zn Batteries on the Polypropylene Industry

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWh Installed Batteries	2,125	10,250	200,000
Daily Polypropylene Production, MT	1.9	9.0	180
% of Existing U.S. Production[a] Capacity	0.09	0.42	8.4
New Plants Required	0.01	0.04	0.8
Environmental Impacts			
Total air emissions,[b] MT/day	0.003	0.016	0.32
Total water emissions, MT/day	0.001	0.005	0.098
Total solid emissions,[c] MT/day	0.012	0.056	1.1

[a] *In 1972, nine plants produced 767,000 MT; we assumed 360 days/yr operation.*

[b] *Uncontrolled emission.*

[c] *Based on mid-range of dry weight of sludge (see Table 3.12).*

3.4.2 Battery

Process Outline

To describe the manufacturing of a battery that has not yet been designed presents special difficulties when the fabrication processes proposed to date differ significantly from each other. In this section, we have chosen to base our analysis on the process proposed by Gould Inc., in its February 1977 design and cost study.[3] The rationale for this choice is Gould's status and experience in producing nickel-cadmium and nickel-zinc batteries, its position as the largest U.S. powder metallurgy company, its 60 years of experience in manufacturing automotive batteries, and its 10 years working experience in the development of the nickel-zinc system. The company is therefore well aware of the system's manufacturing requirements.

A simplified outline of the manufacturing process is given in Figure 3.1. Six process steps in which effluents from the manufacture could be released are indicated; again, since much of the detail of these processes is proprietary, many of the process emissions have had to be postulated on the basis of like technologies.

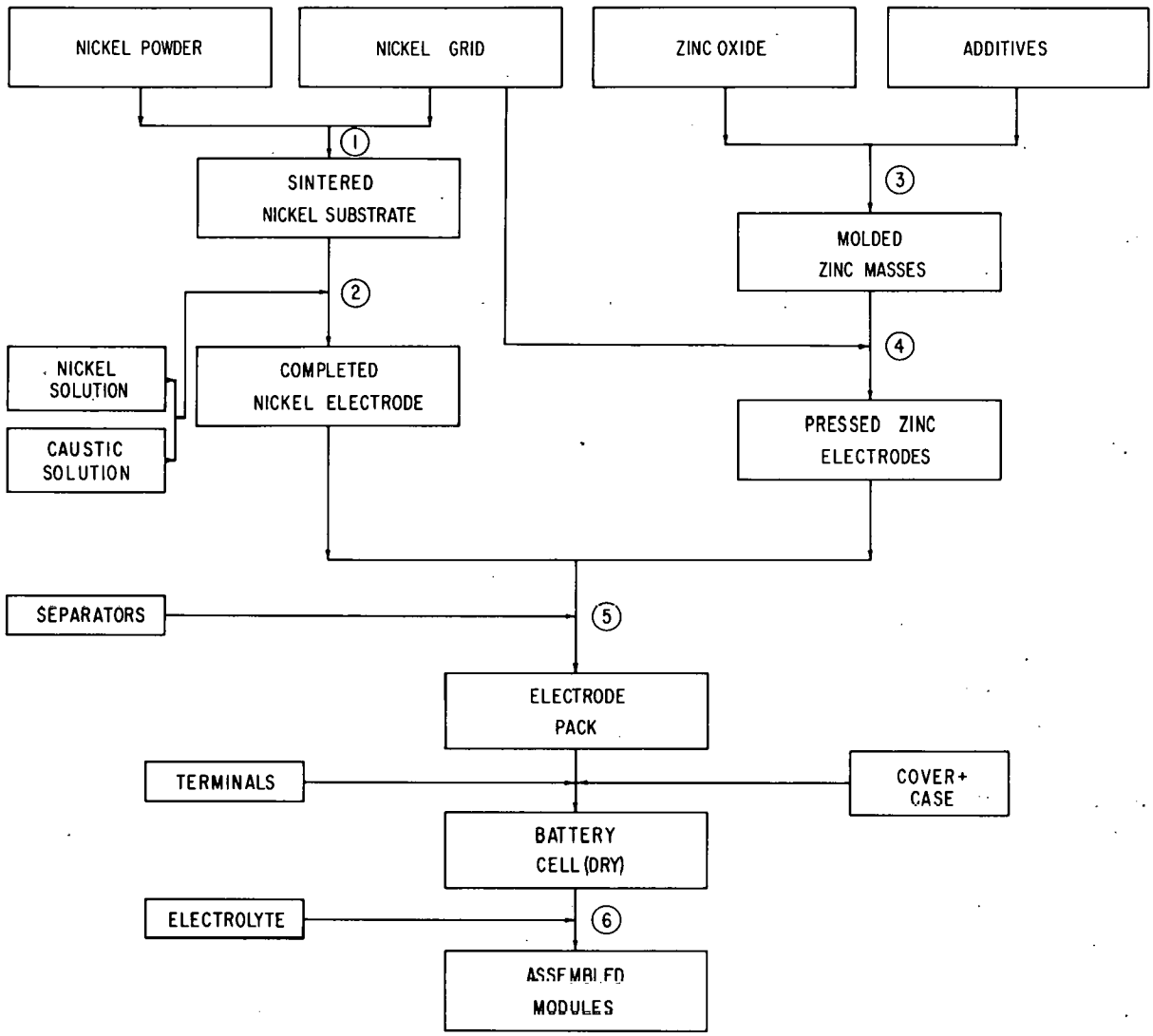


Fig. 3.1. Manufacturing Process for the Nickel/Zinc Electric Vehicle Battery.

Nickel powder and a supportive nickel grid are combined (Step 1 in Fig. 3.1) to form a molded electrode substrate; the mold is then heated to produce a thick sintered porous electrode. The porous electrode is subsequently impregnated with active material (nickel hydroxide plus additives) that is formed directly in the pores of the electrode by the interaction of a nickel solution and caustic solution.

The nickel grid used in the electrode manufacture is produced during the manufacturing process from nickel foil that is welded into the desired configuration, perforated, degreased, and annealed. The nickel solution employed for the impregnation process is produced by the dissolution (probably by nitric acid) of purchased nickel material and process scraps.

The zinc electrodes are fabricated from zinc oxide that has been blended with proprietary additives; the resulting zinc mass is then molded and pressed into two halves. The molded

zinc-halves are then combined with a nickel grid, and the three pieces are pressed into an electrode.

Separators for use in the electrodes are produced either by utilizing plastic materials available from commercial sources or by coating a suitable substrate with a slurry containing the appropriate materials, drying these sheets, and splitting into spools suitable for use in cell assembly.

The cell assembly processes should be, in many respects, analogous to those used in the production of automotive batteries, nickel-cadmium batteries and silver-zinc batteries. The electrodes and separators will be automatically wrapped to produce electrode packs; the terminals will be mounted to the pack; then the cell cover will be mounted and the electrode pack inserted into the case. After the cover has been sealed to the case, the electrolyte is added, final cell hardware put in place, and the cell inspected. Subsequently, the cells will be combined into modules.

Manufacturing Emission Sources*

The powder metallurgy involved in fabrication of the sintered nickel substrate used to manufacture the positive electrode is a possible source of metal powder dust; the major source of dust would be the loading and molding of the electrode (Step 1 in Fig. 3.1). Some dust and fumes may be produced during the welding, cutting and perforating processes in making the nickel grids. The solid scrap from this operation is reused in preparing the nickel solution. The nickel grids are degreased after fabrication, leading to possible solvent vapor emissions.

The impregnation step, which produces active nickel hydroxide materials in the sintered substrate, involves the combination of a nickel solution with a caustic solution to deposit the active material (Step 2, Fig. 3.1). The releases possible in this step are any acid or caustic materials that might be airborne as mists and also washwater containing excess reactants or their products.

Zinc oxide and additives are blended and then molded into two halves (Step 3, Fig. 3.1); during this blending and molding step, dusts containing zinc oxide and additives may be emitted. The additives often used in zinc electrodes fabricated for silver-zinc batteries are mercuric oxide, and flow and blending agents, such as Teflon powder or polyvinyl alcohol; the mercury is added to raise the hydrogen overvoltage.

The pressing of the zinc molds with the nickel grid to form the electrode (Step 4, Fig. 3.1) probably yield some small amounts of solid wastes but little airborne dusts.

Manufacturing of separators will result in small amounts of solid waste (chiefly plastics) and probably washwaters containing dissolved and particulate inorganic materials used in fabrication of the separator. These may include asbestos or zirconium oxide (Step 5, Fig. 3.1). The combination of separator with electrode to produce the electrode pack should be relatively free of emissions.

**See Figure 3.1. Emissions from fabrication of starting components and those components added during cell manufacturing are not included.*

The electrolyte added to the dry battery cells is composed chiefly of aqueous potassium hydroxide. In this step (Step 6, Fig. 3.1), release of mists containing the electrolyte can take place during the preparation of electrolyte and its addition to the batteries; leakage and spills could produce liquid emissions.

The final assembly and storage of completed batteries should not produce significant additional emissions.

Estimated Emission Magnitudes

Approximate amounts of material emitted during the manufacturing process can be estimated from information presented in the Nickel-Zinc Battery Design and Cost Study.[2,3] Detailed process stream data and material balance information are needed to provide accurate emission estimates; however, the orders of magnitude of the emissions listed in Table 3.14 are probably reasonable. Typical efficiencies for dust and mist collection were employed in making the estimates of materials released to the air. The values given in Table 3.14 are probably conservative in terms of the control technology of the 1990s. By the time large-scale manufacture of these batteries is instituted, the air emissions could be reduced even further by improved technology in order to comply with future standards.

Table 3.14. Estimated Manufacturing Emissions[a]

Components[a]	*Form*	*(kg/day)/MWh*
	BATTERY COMPONENTS	
Ni	Dust	0.0048
ZnO	Dust	0.0043
Co	Dust	0.00025
Electrolyte	Mist	0.000025
Plastic	Solid	0.049
Miscellaneous	Dust	0.0011
	PROCESS COMPONENTS[b]	
Wastewater	Liquid	20

[a] *Assumes 90% efficient dust collection, and 99% efficient mist collector.*

[b] *Based on material estimates given in "Develop Nickel-Zinc Battery Suitable for Electric Vehicle Propulsion. Task A - Design and Cost Study," Report to the U.S. Department of Energy, Argonne National Laboratory, ANL-K77-3558-1, Vol. 1, Gould Inc., 15 February 1977.*

[c] *Based on the following reports: "Final Report. Design and Cost Studies of Nickel-Zinc Batteries for Electric Vehicle," "Final Report. Design and Cost Study. Zinc/Nickel Oxide Battery for Electric Vehicle Propulsion."*

The manufacturing process will produce significant quantities of wastewater compared to the other emissions. These wastewaters, chiefly from Step 2, Figure 3.1, will probably be treated by allowing sludge to settle, with subsequent pH adjustment and filtration of the final effluent before its release. If one makes an analogy with the nickel-cadmium battery industry and assumes similar release fractions from the nickel-impregnation steps for both battery systems, the nickel content of the water effluent would be of the order 0.02 (kg/day)/MWh. This value is conservative considering the improved technology that should be available then, especially in view of probable emphasis on nickel recovery for economic and environmental reasons. The sludges produced could exit the process through sale to processors for recovery of nickel (and any other valuable metals) or be re-utilized in the cycle's nickel solution preparation.

Impact of Ni/Zn Battery Industry

The impact of the proposed Ni/Zn battery industry on other existing industries that would supply materials for use in battery manufacture is shown in Table 3.15. A cursory examination of this table reveals several important impacts by the year 2000. Sizeable portions of the nickel, cobalt, and potassium hydroxide industries will have to be dedicated to providing material for EV use. These conclusions imply that, because nickel and cobalt will have to be imported (either as ore or metal) and considerable competition from other industries will exist for these metals, efficient recycling of these metals will become a necessity. The environmental impacts of the Ni/Zn battery cycle are probably minor compared to the economic impacts of the expansion of the EV industry on supplies of these relatively scarce materials.

Table 3.15. Impact of Ni/Zn Batteries on Industry

	Year		
	1985	*1990*	*2000*
Total No. EVs	85,000	410,000	8,000,000
MWh Installed Batteries	2,125	10,250	200,000
MT Battery Material/day	27.8	134	2,620
Batteries/day	∿90	∿400	∿7,700
Projected Production of Component Materials, MT/day (% existing production)			
Nickel[a]	8.2 (0.44)	40 (2.5)	776 (48)
Zinc[a]	4.9 (0.03)	24 (0.08)	464 (1.6)
Cobalt[b]	0.42 (0.68)	2.1 (3.3)	40 (64)
Copper[c]	0.19 (0.005)	0.90 (0.02)	18 (0.44)
KOH[d]	2.9 (0.3)	14 (1.7)	270 (32)
Plastic[e]	1.9 (0.09)	9.0 (0.4)	180 (8.4)

[a] *Based on 1975 world production.*

[b] *Based on 1973 world production.*

[c] *Based on 1974 U.S. production.*

[d] *Based on U.S. annual production.*

[e] *Based on 1972 U.S. production.*

KOH production will also be affected, but this chiefly will necessitate expansion of the existing chlor-alkali facilities for KOH. The economic impacts should be less than for the metal industries discussed above.

3.5 BATTERY USE

The uses to which the nickel-zinc batteries will be put are identical to those discussed for the lead-acid battery discussed earlier (Sec. 2.5). The reader is referred to that section for charging, maintenance and driving scenarios.

As with most EV battery systems nickel-zinc cells are essentially emission-free during normal operation. Overcharging can cause the evolution of oxygen and smaller amounts of hydrogen. This factor constitutes a problem regarding design of sealed (maintenance-free) battery systems, and is not an environmental emission problem. It is conceivable that additives used to improve battery operation may need to be evaluated separately from an operational emission standpoint, but this cannot be addressed until battery design is finalized; however, because additives will be used in low concentrations, their emissions probably will be minor.

3.6 BATTERY RECYCLING

3.6.1 Process Description

Collection and Dismantling

The collection of spent Ni/Zn batteries for resource recovery may result in a specialized "scrapper" and/or other collection segment of the battery manufacturing industry. There is sufficient incentive for battery manufacturers to consider resource recovery in the design of the battery, to facilitate component removal. Consequently, it is unlikely that Ni/Zn battery scrap will be mixed with other nickel or zinc scrap of unknown composition. The recovered spent batteries collected at centralized sites, such as EV marketing and service dealerships, will be transported back to the battery manufacturers or secondary battery recovery enterprises.

It is anticipated that battery manufacturers will consider a battery design to facilitate dismantling of components, and yet provide a relatively safe product which minimizes tampering by the general public. The plates should be readily separable. In this event, battery dismantling, rather than a crushing, would be employed. A Yardney Electric Corp. report[4] indicates that certain Ni/Zn battery electrodes can be removed from the cells, washed, dried, rerolled to original thickness and rebuilt into fresh cells without loss of performance. There is some question, however, as to the number of times an electrode could be reused in such a fashion. Gould, Inc., considers the reuse of nickel electrodes, after some cleaning, as an alternative approach for recycling Ni/Zn batteries.[3] A "crushing" type of procedure similar to those previously presented is feasible, especially if component parts are not readily separable.

Metallurgical Separation

There are several hypothesized approaches to metallurgical separation following front-end operations (Collection and dismantling). Some of these options are illustrated in Figure 3.2.

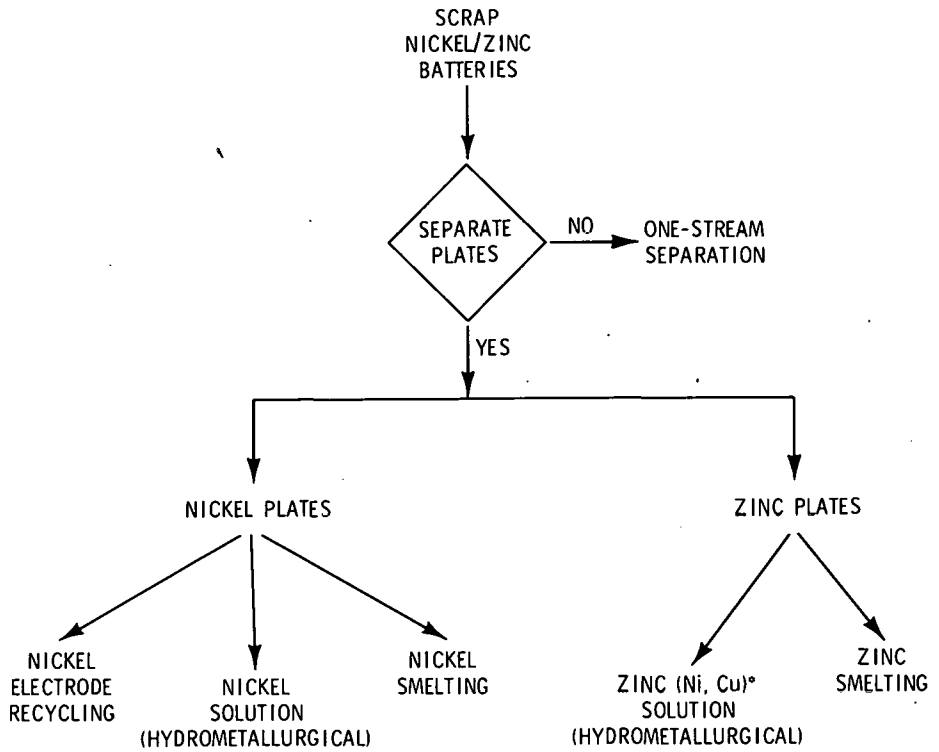


Fig. 3.2. Options for Metallurgical Separation of Scrap Nickel/Zinc Batteries.

Hydrometallurgical processes will most likely be employed rather than the pyrometallurgical or smelting processes used for the recovery of metals from lead/acid batteries. This conclusion is reflected in several studies of the Ni/Zn battery.[1,2,26] The smelting of nickel or zinc battery scrap is technologically viable. However, such an approach would eliminate some of the efficiency in direct reuse, such as recycling the nickel solutions, afforded by hydrometallurgical processes. In addition, the promulgation of the Clean Air Act Amendments of 1977 is providing an economic incentive for considering approaches other than smelting for secondary recovery.

This major difference in the metallurgical process approach will result in a significant shift in the types of emissions anticipated. There will be emphasis on wastewater treatment and aqueous versus atmospheric emissions. The sludge resulting from wastewater treatment will be a solid waste requiring special management, as in the case of the manufacturing of the lead/acid battery.

There are various specific approaches to hydrometallurgical separation and recovery of materials from spent Ni/Zn batteries. The choice of approach will, in large part, depend on the final manufacturer's design for the Ni/Zn battery. A major consideration is whether the plates will be readily separable. If they are, nickel and zinc recovery may occur in two separate streams. Otherwise, a "one-stream" approach could be employed (Fig. 3.2).

Proposed process flows can result either in the recovery of nickel electrodes in reusable form[2] or in dissolving of the electrodes to produce nickel in solution.[2,3] The sintered nickel electrode production approach favored by Gould, Inc., would facilitate the recovery of reusable nickel electrodes.[3]

In general, the processes entail an initial water washing to remove traces of electrolyte, mainly KOH. The washwater may then be filtered for recovery of suspended solids and other sludges. The aqueous portion of the washwater is sent to a neutralization tank. The plates can either be sent to a secondary smelter or dissolved into aqueous solution. Nickel, zinc and cobalt salts are then selectively removed. The nickel, zinc and cobalt salts can be readily converted to the hydroxide form, required for electrode production, by electrolysis. Other nickel-bearing solutions, for example, the waste processing solution from the grid impregnation process, can also be recycled as part of the nickel recovery process. Figure 3.3 shows an example[3] at one of these processes.

3.6.2 Air Emissions

Collection and Dismantling

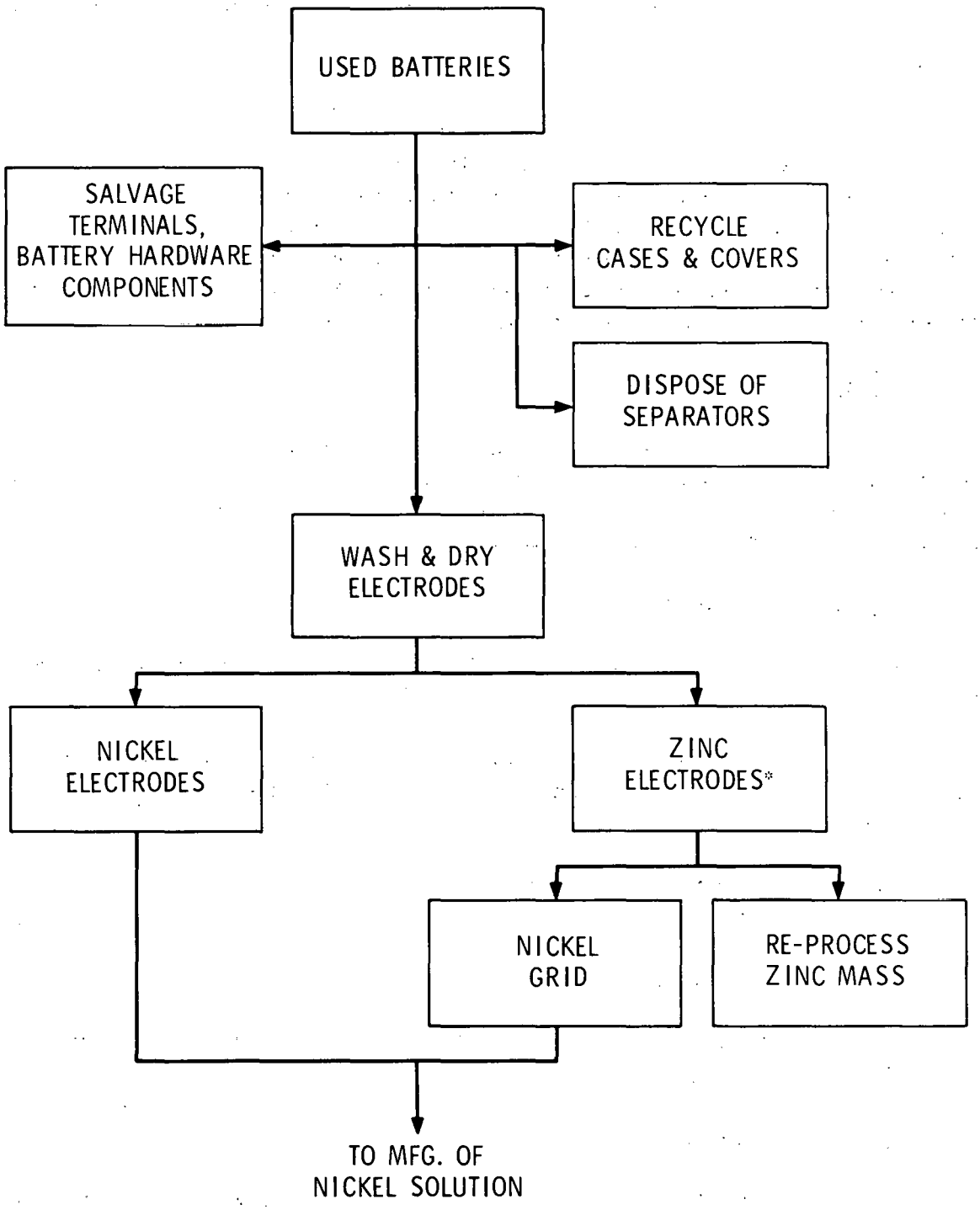
Quantifiable estimates of anticipated emissions to the atmosphere from the collection and dismantling of spent Ni/Zn batteries are not currently available, and their derivation is beyond the scope of this project. Nevertheless, some qualitative environmental and health impacts are discernible at this time.

Entrained liquid particles, such as potassium hydroxide mist resulting from the agitation in the collection, transportation and dismantling of these batteries, may pose environmental and health hazards similar to electrolytic mist generation in the lead/acid battery breaking process. Hazardous mist generation may be more prevalent in the case of Ni/Zn battery.

Although wetting the batteries will reduce the amount of dust emissions, dust will still be generated, particularly in the dismantling phase. Zinc oxide dust (the form of the active material in the discharged state) will probably be generated along with other particulate matter containing nickel. The relative amount of such material is difficult to ascertain. In general resource-recovery facilities generate considerable dust as part of normal operations.

The dismantling operations may also result in mist and dust containing materials from the separators, such as asbestos. The extent and environmental health significance of such emissions is difficult to ascertain at this time.

The mist generated from the front-end operations can be controlled in a similar fashion to that described for lead/acid batteries (Sec. 2.6). In addition, measures can also be taken to reduce the amount of mist generated in the collection and dismantling steps. Similarly, fugitive dust can be controlled through an exhaust system leading to a central control device. It is doubtful, however, that sufficient zinc oxide dust will be generated during the front-end



*Assumes nickel grid.

Fig. 3.3. A Proposed Process for Recycling Used Nickel/Zinc Batteries. Source: Gould, Inc., Report ANL-K77-3558-1, 1977.

operations to warrant recycling considerations. (The sources and control of fugitive dust were discussed generically in Sec. 2.6.2.) Overall, in the context of the limited information available and assuming that mitigating measures are implemented, no major foreseeable environmental problems are associated with the front-end operations of recycling Ni/Zn scrap batteries.

Metallurgical Separation

The argument has been made in previous sections that resource recovery and recycling associated with the Ni/Zn batteries will utilize hydrometallurgical rather than pyrometallurgical processes. One of several reasons for this conclusion is the air-emission control problems (technical and economic) associated with conventional pyrometallurgical approaches. In general hydrometallurgical processes should not lead to air pollution problems. Because it is not yet known which process will be used to recover the metallic components of the Ni/Zn battery, the air emissions will not be discussed further.

3.6.3 Liquid Emissions

Uncontrolled liquid emissions from the recycling of the Ni/Zn batteries can be a major source of pollutant emissions to the environment. Emissions include both the discarded electrolyte, which contains KOH and suspended metal particles, oxides and hydroxides, and the raw wastewater for the hydrometallurgical processes for recovering the nickel and zinc.

Collection and Dismantling

The major sources of liquid wastes which will require treatment from the front-end operations are as follows:

- Electrolyte (KOH) from spent battery dismantling. This should be a major source of pollutants because the batteries may be received with much of the electrolyte still in the cases (Sec. 3.6.1).

- The wastewater generated during dismantling operations. This contains much of the electrolyte present in the spent battery.

- The spilled electrolyte captured at storage sites.

The raw wastewater generated at these operations would then be sump-pumped to a central holding tank.

Metallurgical Separation

Several proposed processes for nickel, cobalt, and zinc recovery use solutions that dissolve the plates (or other form of feed material). Subsequently, the materials of interest are removed from the solution by the addition of chemicals that form a relatively insoluble complex with the compounds; the precipitate is either filtered or settled out.

The general sources of liquid waste which will require treatment and disposal are as follows:

- Nickel and zinc plate wash or rinse water that is contaminated.
- Wet scrubber effluent (air exhaust systems).
- Leachate from landfills (assuming these are onsite).
- Spent processing solutions formed during hydrometallurgical separation steps.
- Process tank overflows and other accidental spillage and leakage of processing solutions and spent electrolyte.

Generally, the liquid waste emissions generated from the processes will be hazardous, characterized by either high or low pH (depending on source) and containing high concentrations of suspended and dissolved solids and various heavy metal salts (Ni, Zn, Ca, and Cd oxides and sulfates), anions of the acids and possibly small quantities of oils and greases removed from battery cases and work surfaces. The influent to the wastewater treatment plant from the front-end operations will be alkaline (due to the electrolyte) and will include suspended nickel, zinc and cobalt oxides and hydroxides. Nickel hydroxide is not very soluble in the electrolyte; therefore, there will be relatively little nickel in the dissolved state. Zinc and zinc oxide, on the other hand, are somewhat soluble in the electrolyte. Consequently, heavy loadings of zinc into the wastewater treatment plant are anticipated. Alkaline wastewater from the Ni/Zn battery manufacturing plant is also anticipated, if the plant is onsite. The liquid emissions from the hydrometallurgical processes are somewhat more difficult to characterize, as several alternative processes could be employed. Generally, the liquid wastes will be low in pH with accompanying dissolved and suspended metals and their salts. Potassium, lithium, and heavy metal sulfates may be present, for example.

The wastewater treatment process steps are directed to meet design criteria. These design criteria are a function of several factors including federal, state or local requirements on discharge quality and quantity to receiving streams and/or municipal wastewater treatment plants, and individual features of the industrial processes, such as types of liquid wastes, drainage rates, and volumes. Due to the variety of possible approaches to hydrometallurgical separation as well as the lack of specific regulations for this phase of the industry, it is difficult at this time to specify the type of treatment that will be employed. Because certain constituents of the waste stream are toxic, stringent effluents limitations will most likely come into effect. National Pollutant Discharge Elimination Systems permits, as well as existing pretreatment standards for these plants discharging to sewer systems, will be required by the EPA, State or a local regulatory agency.

Although it is difficult to specify which type of wastewater system will be used, it is likely that it will include metal recovery and process solution regeneration steps. Ion-exchange treatment systems can be employed to recover nickel, zinc, copper and iron in the process solutions. Moving-bed ion-exchange colums appear to be justifiable technically and economically for such systems. Other systems which may be employed are evaporative recovery and reverse osmosis systems.

The technology is potentially available to treat wastewater streams in an environmentally acceptable manner. The economic considerations, particularly for the more advanced systems which may be required, will be important. However, it is anticipated that integrated systems which treat waste streams as well as regenerate process solutions and recover residual valuable constituents will offset the capital, operating and maintenance costs for these wastewater treatment systems.

3.6.4 Solid Wastes

It is anticipated that there will be little unreclaimed waste from the resource recovery operations for scrap Ni/Zn batteries. The climate leading to this conclusion has been variously discussed in this report. Factors that will contribute to a maximum materials-recovery effort are also, in part, those anticipated to stimulate the use of processes having minimal detrimental impact on the environment. In general, hydrometallurgical process routes will generate substantially less solid waste from air pollution control devices than lead/acid battery secondary recovery processes.

Collection and Dismantling

Battery cases are not anticipated to be a significant solid waste problem. Rather, they will be recycled. Gould, Inc., for example, is utilizing a polypropylene case for its Ni/Zn battery which is recyclable. Separator materials will require disposal. Again it is assumed that separators will be subjected to either a washing or leaching step. This will minimize the potential problems associated with their disposal.

The sludges collected during front-end, as well as metallurgical processes will contain many of the metals and metal salts present in the liquid waste stream. Sludges or muds accumulated at sump pumps in the bottom of battery cases and wastewater treatment plants represent major pollution sources. Nickel hydroxide is anticipated to be a major pollutant constituent of such sludges. The quantity and characteristic of sludge generated from wastewater treatment plants will depend on many factors, including the type of hydrometallurgical process employed and the wastewater treatment process used. The disposal of the sludges in secured landfills will probably be required by federal or local regulatory agencies. These sludges may be recycled to remove valuable metal constituents. The residual solids from the sludge recycling will require further disposal. Alternatively, more advanced wastewater treatment systems, such as those employing ion-exchange or reverse osmosis, may result in substantially less sludge.

Metallurgical Separation

The major source of solid waste requiring management will probably be the wastewater-treatment facilities. Due to an uncertainty as to the specific processes that will be employed, it is difficult to specify the sludge characteristics. The sludge, however, is anticipated to be a hazardous material due to the toxicity of its constituents, such as nickel hydroxide.

Traditionally, sludges from industrial process sources such as the metal-finishing industry have been disposed of on land. This practice entails spreading the sludges on suitable ground

area and periodically covering them to prepare a new deposit or layer base. This may be an acceptable solid-waste management option if suitable hydrogeological sites can be found. The term suitable, in large part, will be defined by responsible local regulatory agencies.

Although nickel hydroxide is anticipated to be a major pollutant constituent for the Ni/Zn battery, metals, as hydroxides or carbonates, precipitated in the sludge are generally not soluble.[27] Consequently, land spreading may be acceptable. However, pH conditions may vary, for example, in "acid rain" conditions. The resulting effect on the solubility of metals would be to make mobility through groundwater and groundwater contamination an important consideration.

The recycling of generated sludges in the hydrometallurgical process flow is a viable solid-waste management option. The residual sludges not recycled would be disposed of in secured landfill.

Increasingly, land spreading and secured landfill operations are becoming cost-intensive options. This is attributed to many factors including regulations and unavailability of suitable land. In that context, advanced wastewater treatment systems which generate relatively small amounts of sludge may become economically attractive to the industry, even though the capital expenditures may be large.

3.7 SUMMARY

1. Impacts of the nickel/zinc battery-powered electric vehicles on the total cycle of the nickel/zinc battery industry were assessed. The market penetration was assumed to grow at a rate of 30% per year and to reach a total of eight million nickel/zinc battery-powered electric vehicles on the road by the year 2000. Assessments were also made for per megawatt hour of installed capacity, assuming a one-megawatt hour requirement for each 40 electric vehicles. The assessment included consideration of resource requirements, industrial growth, and effluents to the environment from various phases of the total battery cycle.

2. For the assumed scenario, by the year 2000:

   a. a 48% increase in refined nickel production will be required as compared to current world output,

   b. a 64% increase in cobalt production will be required as compared to current world output,

   c. a 32% increase in KOH production will be required as compared to current U.S. output,

   d. a Ni/Zn battery recycle industry will need to be developed because of the scarcity and cost of nickel and cobalt.

3. For the assumed scenario, by the year 2000 major air-borne emissions are expected at the following levels (assuming current standards and/or control technology are applied).

   a. 19 MT of SO_2 per day from primary zinc production.

b. 20 MT of particulates per day from primary nickel production.

c. 610 MT of SO_2 per day from primary nickel production.

4. Liquid emissions, for the assumed scenario by the year 2000, will consist chiefly of wastewaters from mining and milling.

a. 80,000 MT of water per day from zinc mining and milling.

b. 260,000 MT of water per day from primary nickel production (includes mining, milling, smelting, and refining).

5. For the assumed scenario, by the year 2000 16,000 MT per day of solid wastes from primary zinc production are expected to be produced (assuming current standards and/or control technology are applied).

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4. EFFLUENTS FROM THE NICKEL/IRON BATTERY CYCLE

ABSTRACT

The Ni/Fe battery cycle from mining and milling of the raw materials through recycling of the scrap batteries and disposal of wastes has been described. Material flows and emissions for various phases of the total cycle have been estimated for per megawatt hour of installed capacity and for a scenario of 8×10^6 electric vehicles on the road by the year 2000.

4.1 INPUT PARAMETERS

The nickel/iron (Ni/Fe) storage battery was developed by Thomas A. Edison at the turn of the century in his work to replace horse-drawn vehicles with electric vehicles. However, his durable nickel/iron battery could not match the internal combustion engine in cost or performance, and subsequently the battery had its chief applications in railroad lighting and industrial trucks. The Ni/Fe battery has changed remarkably little since its development; one of its chief advantages remains the proven long life-cycle under conditions where mechanical and electrical abuse are common (thousands of charge-discharge cycles are not uncommon). However, the Ni/Fe system has a lower energy density than the nickel/zinc (Ni/Zn) battery, and performs poorly at low temperatures. As with the Ni/Zn system, cost reductions are necessary before wide deployment of Ni/Fe batteries can be expected.

The following discussion utilizes information presented in the Design and Cost Study[1] prepared by Westinghouse for Argonne National Laboratory in 1977; the details do not necessarily represent a Westinghouse system but rather are a composite based on all the information available about such batteries. Emission values are order-of-magnitude estimates, as no specific manufacturing process or battery design can be identified at the present time.

As in the previous discussion for the Ni/Zn battery, we shall assume an approximate battery composition (see Table 4.1). The plastic components are again assumed to be polypropylene. Miscellaneous battery materials and additives, including inorganics and assorted hardware, are not discussed in the following sections, because details are not available and the amounts involved are small. If we assume an EV range of 180 km, a life of 2000 cycles and an annual traveled distance of 16,000 km, the total Ni/Fe battery lifetime will be 22.5 years. Operational lifetimes of 30 years for present-day Ni/Fe batteries are not unheard of. With a growth scenario identical to that given in Table 3.2 for the Ni/Zn cycle, the same annual growth rate--about 30%--is obtained. As assumed previously for the Ni/Zn battery, a 10% loss of battery components to the environment was factored into the environmental scenario.

Table 4.1. Approximate Composition for the Ni/Fe Battery for EV Use

Material	Composition	
	Weight %	MT/MWh[a]
Ni	16	2.7
Co	0.76	0.13
Steel	25	4.2
Fe_2O_3	17	2.8
Cu	3.8	0.63
Electrolyte	26[b]	4.3
LiOH	1.4	0.23
Rubber	0.056	0.0093
Plastic	8.2	1.4
Miscellaneous	2.2	0.37

[a] *Based on 60 Wh/kg.*

[b] *Electrolyte solution contains 25 wt. % KOH.*

4.2 MATERIAL FLOW RATES

Overall daily flow rates for materials used in the Ni/Fe battery cycle have been calculated using the methods found in Section 2, with results as shown in Table 4.2. The column headed "Total" presents the materials flow for new batteries; the "Loss" column relates component loss involved in manufacture plus the 10% loss assumed for completed new batteries; "Installation" presents the calculated flow using parameters given in Section 2; the final column, "Removal of Spent Batteries," gives material removed from the cycle as fully discharged, spent batteries.

Table 4.2. Ni/Fe Battery Component Flow Rates, (kg/day)/MWh

Battery Component	New Batteries			Removal of Spent Batteries
	Total	Loss	Installation	
Ni	2.44	0.24	2.20	0.0026
Co	0.11	0.01	0.10	0.00012
Steel	4.01	0.58	3.43	0.0040
Fe_2O_3	2.72	0.39	2.33	0.0027
Cu	0.57	0.05	0.52	0.0061
Electrolyte	4.47	0.91	3.56	0.0042
LiOH	0.21	0.02	0.19	0.0022
Rubber	0.009	0.001	0.008	0.00001
Plastic	1.30	0.17	1.13	0.0013
Miscellaneous	0.33	0.03	0.30	0.00035
TOTALS				
Dry Weight	12.82	1.72	11.10	0.0204
Battery	16.17	2.40	13.77	0.0236

4.3 PRIMARY PRODUCTION OF INPUT MATERIALS

4.3.1 Iron and Steel

It will be assumed here that the iron and steel used in production of the iron electrodes for the Ni/Fe battery are the products of integrated steel mill production. Since special properties may be required for these ferrous materials, their production will also be assumed to be carried out by electric furnaces charged chiefly with cold scrap.

Emissions from Mining and Milling

The total amounts of steel required in the Ni/Fe battery constitute at most a very minor perturbation to the output of the U.S. steel industry; only about 0.2% of U.S. total steel production is needed for 8 million EVs. In addition, the electric furnace uses mostly scrap metal, of which there is an enormous backlog.[2] Because the emissions from mining and milling of the ores for these scrap materials have already been accounted for in the production of the virgin material and because the total amounts of material involved are insignificant when compared with the total output of the U.S. steel industry, the impact of the Ni/Fe battery on iron-mining and -milling emissions in the United States need not be discussed in this report.

Emissions from Electric Furnaces

The emissions from electric furnaces using dry emission controls are chiefly slag and dust generated at the rates of 120 kg/MT and 12.8 kg/MT of steel, respectively.[3] The slag is a dense, hard material composed principally of iron, silica and calcium compounds, which do not leach significantly; the slag is usually dumped after recovery of the metallic content. The particulate emissions from an electric furnace can be reduced significantly using electrostatic precipitators or baghouses; a conservative 98% removal[4] of dusts (baghouse collection) would yield emissions of about 0.3 kg/MT of steel. Even lower (0.045 kg/MT) particulate emissions with baghouses have been reported.[5] Carbon monoxide is also emitted from the electric air furnace at a level of 9 kg/MT of steel.[6] Liquid wastes are associated with other steel processes--chiefly pickle liquors from cold rolling mills. Standards of performance for new sources (40 CFR 420.105) set average effluent limitations per metric ton of steel at 0.0052 kg total suspended solids, 0.0042 kg fluoride, and 0.0010 kg zinc. We will assume that the liquid emissions associated with the product of the electric furnace meet these standards.

Table 4.3 presents the estimated emissions associated with the production of Ni/Fe batteries. We have assumed, for our purposes, that the production of metal powders for use in the battery processes yields small amounts of emissions compared with those from iron and steel production.

Impact on the U.S. Steel Industry

The impact of the Ni/Fe battery on the U.S. steel industry is summarized in Table 4.4. The impact on the U.S. electric furnace steel-making capability will not be significant, even in the year 2000.

Table 4.3. Emissions from Production of Iron and Steel for Use in Ni/Fe Batteries

Emissions	*(kg/Day)/MWh*
AIR[a]	
Particulates	0.001
Cr	1×10^{-6}
Cu	2×10^{-6}
Mn	4×10^{-5}
Ni	2×10^{-7}
Pb	2×10^{-5}
Zn	1×10^{-4}
CO[b]	0.04
WATER[c]	
TSS	2×10^{-5}
F	2×10^{-5}
Pb	4×10^{-6}
SOLIDS[a]	
Slag (landfill disposal)	0.5

[a] *Based on data in R.P. Leonard et al., PB 276171, 1977.*

[b] *From "Compilation of Air Pollutant Emission Factors," 2nd Ed., U.S. Environmental Protection Agency, AP-42, 1972.*

[c] *40 CFR 420.105.*

Table 4.4. Impact of the Ni/Fe Battery on the U.S. Steel Industry

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWh Installed Batteries	2125	10,250	200,000
Daily Steel Requirement, MT	8.5	41	800
% of Existing Production Capacity[a]	0.01	0.05	1.1
Environmental Impacts			
Total air emissions, MT/day	0.08	0.38	7.4
Total solid emissions, MT/day	1.0	4.9	96
Total liquid emissions, MT/day[b]	0.20	0.94	18

[a] *Based on 1974 U.S. electric furnace steel capacity of 27×10^6 MT/yr.*

[b] *Based on a pickle liquor discharge of 23 kg/MT steel (see R.P. Leonard, PB 276171, 1977).*

4.3.2 Nickel

Mining and Milling

The nickel-mining capabilities of the U.S. industry are inadequate (Sec. 3.3.2) for the needs of a burgeoning nickel-based battery cycle; thus, the supply of ores must come from outside the U.S., probably from Canada. The order-of-magnitude estimates of emissions given in Section 3.2.3 for the Ni/Zn battery cycle will also be applied here to the Ni/Fe battery; these are: 150 MT solids and 340 MT wastewater per metric ton of nickel metal. The value for wastewater is the sum of the mining, milling, smelting and refining processes. These values are at best crude first-order approximations that should be refined when data become available. Estimated waste flows for the nickel mining and milling associated with the Ni/Fe battery cycle are as follows:

Tailings	400 (kg/day)/MWh
Wastewater*	700 (kg/day)/MWh

Emissions from Smelting and Refining of Nickel

Wastes from nickel refining and smelting were estimated in Section 3.3.1 (Ni/Zn battery cycle). The same arguments apply to the Ni/Fe battery cycle: namely, that U.S. nickel production capacity is inadequate for the later stages of an EV battery industry, and that foreign sources of supply will be required. The emission estimates given for the Ni/Zn cycle (again based on very limited published information) will be applied to the Ni/Fe battery cycle. The estimation methods are discussed in Section 3.3.1.

Air emissions from nickel smelting and refining are assumed to meet the standards given in 40 CFR 60.172-3; this results in 780 kg SO_2 and 21 kg particulate matter per metric ton of nickel. The composition of particulates can be estimated using data of Hutchinson and Whitby[6] for the INCO Copper Cliff Smelter at Sudbury, Ontario, Canada. Materials flows for air emissions for the Ni/Fe battery cycle are shown in Table 4.5.

Water emissions are estimated to be about 340 MT wastewater per metric ton of nickel metal; we have assumed that these emissions will meet the design specifications proposed by INCO for its Copper Cliff facility[7] (somewhat more stringent than EPA standards). The estimated flows based on these values are also given in Table 4.5.

Impact on the Nickel-Refining Industry

The deployment of large numbers of EVs using Ni/Fe batteries would have a significant effect on nickel utilization, similar to the situation for the Ni/Zn battery (see Sec. 3.3.2). The magnitude of this impact is shown in Table 4.6. Again, as for the Ni/Zn battery cycle, the likelihood of the establishment of a sizable nickel-recycling industry seems great. Large amounts of pollutants are expected to be emitted, and significant improvements in control technology will be necessary to reduce the absolute quantities of pollutants released to the environment.

**See Section 3.3.2 for the estimation method. This value includes wastes from smelting and refining as well.*

Table 4.5. Estimated Emissions from Nickel Smelting for the Ni/Fe Battery Cycle

Emissions	*Flow (kg/day)/MWh*
AIR	
SO_2	1.9
Total Particulates	0.051
Ni	0.006
Co	0.0001
Pb	0.0005
WATER[a]	
Wastewater	830
TSS	0.01
Ni	0.0005
Cu	0.0002
Fe	0.0005

[a] *Includes emissions for mining, milling, smelting and refining.*

Table 4.6. Impact of the Ni/Fe Battery on the Nickel-Refining Industry

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWh Installed Batteries	2125	10,250	200,000
Daily Nickel Requirements, MT	5.2	25	490
% of 1975 World Production[a]	0.3	1.5	30
Environmental Impacts			
Total particulate emissions, MT/day	0.1	0.5	10
Total water emissions, MT/day	1800	8,500	170,000
Total SO_2 emissions, MT/day	4	20	380

4.3.3 Cobalt

Virtually all the cobalt used in the United States is of foreign origin. U.S. refinery production in 1977 amounted to about 340 MT, which is clearly inadequate for the needs of the nickel-based battery industry. Because the cobalt used will be of foreign origin, we cannot at this time provide estimates of the emissions from these mining, milling, smelting and refining processes; however, their impacts on the U.S. environment will be minimal at worst.

4.3.4 Potassium Hydroxide

Caustic potash (potassium hydroxide) manufacturing and the emissions likely to occur from large-scale deployment of nickel-based battery-powered EVs are discussed in Section 3.4.

Estimate of Emissions from KOH Production

Estimates of emissions in the production of potassium hydroxide (KOH) as a result of deployment of EVs using Ni/Fe batteries are given in Tables 4.7 and 4.8. The estimates are based on the emission information presented in Section 3.3.4 for the chlor-alkali industry and on the cycle flow data given in Table 4.2. Table 4.7 has the estimates for the various effluents.

Impact on the Chlor-Alkali Industry

Table 4.8 shows the impact of Ni/Fe battery EV usage on the chlor-alkali industry. The impact of the increased caustic potash requirements on the chlor-alkali industry is assessed in Table 4.8. The emissions are slightly less than those estimated for the Ni/Zn battery cycle (Table 3.11), which in turn were not large. The impact of the EV industry on KOH production should not be significant until large-scale utilization occurs at the end of the century; at that time an expansion of about 26% over currently existing production would be required.

4.3.5 Lithium Hydroxide

Mining and Milling of Lithium Ores

The most important U.S. mineral ore source of lithium consists of spodumene deposits in North Carolina. The spodumene ore is obtained by conventional open-pit mining techniques; the ore is crushed and wet-ground prior to flotation. The tailings from processing consist chiefly of feldspar, mica and quartz. U.S. spodumene production figures are proprietary; however, using 1973 statistics on U.S. lithium minerals deliveries[8] of about 4300 MT of contained lithium, and assuming this to result solely from spodumene refining, we can estimate a daily production of about 12 MT of contained lithium. This corresponds to about 1900 MT of spodumene ore, or a ratio of 160 MT ore per metric ton of lithium.

Estimate of Emissions from Mining and Milling

Slimes and tailings from one producer of ore concentrates[9] amount to 620 kg per metric ton of feed; this would correspond to about 100 MT of solid wastes per metric ton of lithium.

Water emissions from such a facility[9] (including mine water) amount to about 0.65 MT water per metric ton of ore; this corresponds to about 100 MT wastewater per metric ton of lithium produced. This processing involves 95% recycle of process water and 100% recycle of cooling water, resulting in these relatively low wastewater releases. The best practical control technology effluents from the spodumene processing industry have been estimated (Ref. 9, p. 248) as 0.11 kg TSS and 0.017 kg fluoride per metric ton of ore processed (monthly averages).

Table 4.7. Estimate of Emissions from KOH Production for the Ni/Fe EV Battery

Emissions	*(kg/day)/MWh*
AIR	
Chlorine[a]	3×10^{-6}
Carbon Monoxide[b]	6×10^{-5}
WASTEWATER[c]	
TSS	2×10^{-4}
Pb	3×10^{-8}
SOLID[d]	
Total Solids	$\sim 4 \times 10^{-3}$
Pb Solids	7×10^{-5}
Mg and Ca Solids	4×10^{-3}
Chlorinated Hydrocarbons	1×10^{-4}

[a] *Assumes caustic scrubber lowers chlorine emission.*

[b] *Assumes no emission control.*

[c] *40 CFR 415.65, new source performance standards.*

[d] *Dry basis (sludge is 3% solids).*

Table 4.8. Impact of the Ni/Fe Battery on the Chlor-Alkali Industry (Caustic Potash Production)

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWh Installed Batteries	2,125	10,250	200,000
Daily KOH Requirement, MT	2.4	11	220
% of Existing U.S. Production Capacity[a]	0.3	1.3	26
New KOH Plants Required[b]	0.02	0.1	2
Environmental Impacts			
Total air emissions, MT/day	0.0001	0.0006	0.012
Total water emissions, MT/day	0.0005	0.002	0.05
Total solid emissions, MT/day	0.01	0.04	0.9

[a] *Assumes annual U.S. caustic potash production of 300,000 MT (360 day/yr).*

[b] *Assumes average plant produces 35,000 MT/yr.*

The materials flows based on these estimates, relative to the Ni/Fe battery cycle, are:

	(kg/day)/MWh
Tailings	6
Wastewater	6
TSS	0.001
F (soluble)	0.0001

Estimate of Emissions from LiOH Manufacture

No emission data for the LiOH manufacturing processes have been reported in the literature; however, estimates can be made based on descriptions of current commercial processes for spodumene processing.[10] We have chosen to base estimates on the Foote Mineral Company process for spodumene, with the assumption of 90% lithium recovery, 98% water recycle, and 99% efficiency of particulate control technology. The process steps are: calcination of lithium ore concentrates (one part ore to 3.5 parts limestone); grinding of the resultant clinker; separation in a six-stage thickener; filtration; evaporation; and centrifugation to yield $LiOH \cdot H_2O$.

Estimates made of the emission rates based on the Foote Mineral Co. process are as follows: 4 kg particulates, 10 MT wastewater, and 40 MT solids (dry basis) per metric ton LiOH produced. It should be kept in mind, again, that these are order-of-magnitude estimates, and that the assumptions used in their calculation are somewhat arbitrary. Emission flows corresponding to the Ni/Fe battery cycle are given in Table 4.9. The relatively large solid emissions are due to the process requirement of 3.5 parts limestone per one part ore concentrate.

Table 4.9. Estimated Emissions from LiOH Manufacturing

Emission Type	*Form*	*Emissions, (kg/day)/MWh*
Air	Particulates	0.0008
Water	Wastewater	2
Solids[a]	Filter sludges	8

[a] *Dry basis.*

Impact on the U.S. LiOH Industry

Table 4.10 compares the impact of LiOH requirements through the year 2000 for an expanding Ni/Fe battery industry. From the requirements shown in the table, it is evident that although the LiOH quantities used in each battery are small, lithium will be a critical component. Competition for the lithium supply may come from other battery systems and electronics development.

Table 4.10. Impact of Ni/Fe Battery Requirements on the LiOH Industry

	Year		
	1985	*1990*	*2000*
Total EV's (thousands)	85	410	8,000
MWh Installed Batteries	2,125	10,250	200,000
Daily LiOH Requirement, MT	0.45	2.2	42
% 1973 U.S. Li Production Capacity[a]	1.1	5.3	100
New LiOH Plants Required[b]	0.05	0.2	5
Environmental Impacts[c]			
Total air emissions, MT/day	0.002	0.01	0.2
Total waste emissions, MT/day	20	80	1,600
Total solid emissions, MT/day	30	140	2,800

[a] *Annual production equivalent to 41.4 MT LiOH/day (360 day/yr).*

[b] *Assumes typical plant produces the equivalent of 5000 short tons per year as lithium carbonate.*

[c] *Includes mining, milling, and LiOH manufacture.*

4.4 MANUFACTURING PROCESS

4.4.1 Case and Cover

A detailed discussion of the use of plastics in battery case and cover manufacture has been given in Section 2.4.1. The reader is referred to that section for a description of polypropylene production. The estimates that follow here are based on the emission information presented in Section 2.4.1.

Emissions

The emissions reported in Table 4.11 represent a conversion of the emissions given in Section 2.4.1 (SOA lead/acid battery) for production of polypropylene cases and covers to conditions appropriate to the Ni/Fe battery industry. The air emission values represent uncontrolled emissions and should be amenable to further reduction by the use of additional control equipment.

Impact on the Polypropylene Industry

Table 4.12 shows the estimated production requirements for polypropylene in an expanding Ni/Fe battery industry. As can be seen from the table, the impact on the existing polypropylene industry is minimal; by the year 2000, only one new plant (of current size) would be required to produce the necessary plastics. The emissions, based on current practice, are probably larger than would be expected or permitted in the 1990s; thus, it is likely that significant reduction of emissions from polypropylene manufacture will occur within that time frame.

Table 4.11. Emissions from the Production of Polypropylene (from Propylene) for Ni/Fe Battery Cases and Covers

Emissions	*Flow (kg/day)/MWh*
AIR[a]	
Particulates	2.0×10^{-3}
Propylene	4.5×10^{-4}
WATER[b]	
TSS	7.3×10^{-4}
SOLID[c]	
Sludge	0.001 - 0.01

[a] *Uncontrolled emissions.*

[b] *Treated wastewater.*

[c] *Equivalent dry weight of sludge.*

Table 4.12. Impact of the Ni/Fe Battery on the Polypropylene Industry

	Year		
	1985	*1990*	*2000*
Total EVs (thousands)	85	410	8,000
MWh Installed Batteries	2,125	10,250	200,000
Daily Polypropylene Requirement, MT	2.6	13	250
% Existing U.S. Production Capacity[a]	0.1	0.6	12
New Plants Required	0.01	0.05	1
Environmental Impacts			
Total air[b] emissions, MT/day	0.005	0.025	0.49
Total water emissions, MT/day	0.002	0.007	0.15
Total solid[c] emissions, MT/day	0.01	0.06	1

[a] *In 1972, nine plants produced 767,000 MT; we have assumed 360 days of operation per year.*

[b] *Uncontrolled emissions.*

[c] *Based on mid-range of dry weight of sludge (see Table 4.11).*

4.4.2 Battery

Process Outline

The manufacturing of Ni/Fe batteries in the traditional fashion is described in detail in the literature, e.g., Falk and Salkind;[11] the process described below is based, however, on a process outlined in the Westinghouse Design and Cost Study.[1] The Westinghouse process, given only in barest outline in that report, appears to differ from the classic technique of Ni/Fe cell manufacture; especially novel is the use of a nickel-electroplated steel substrate as the positive electrode. Figure 4.1 is a simplified flow diagram for the manufacture of Ni/Fe

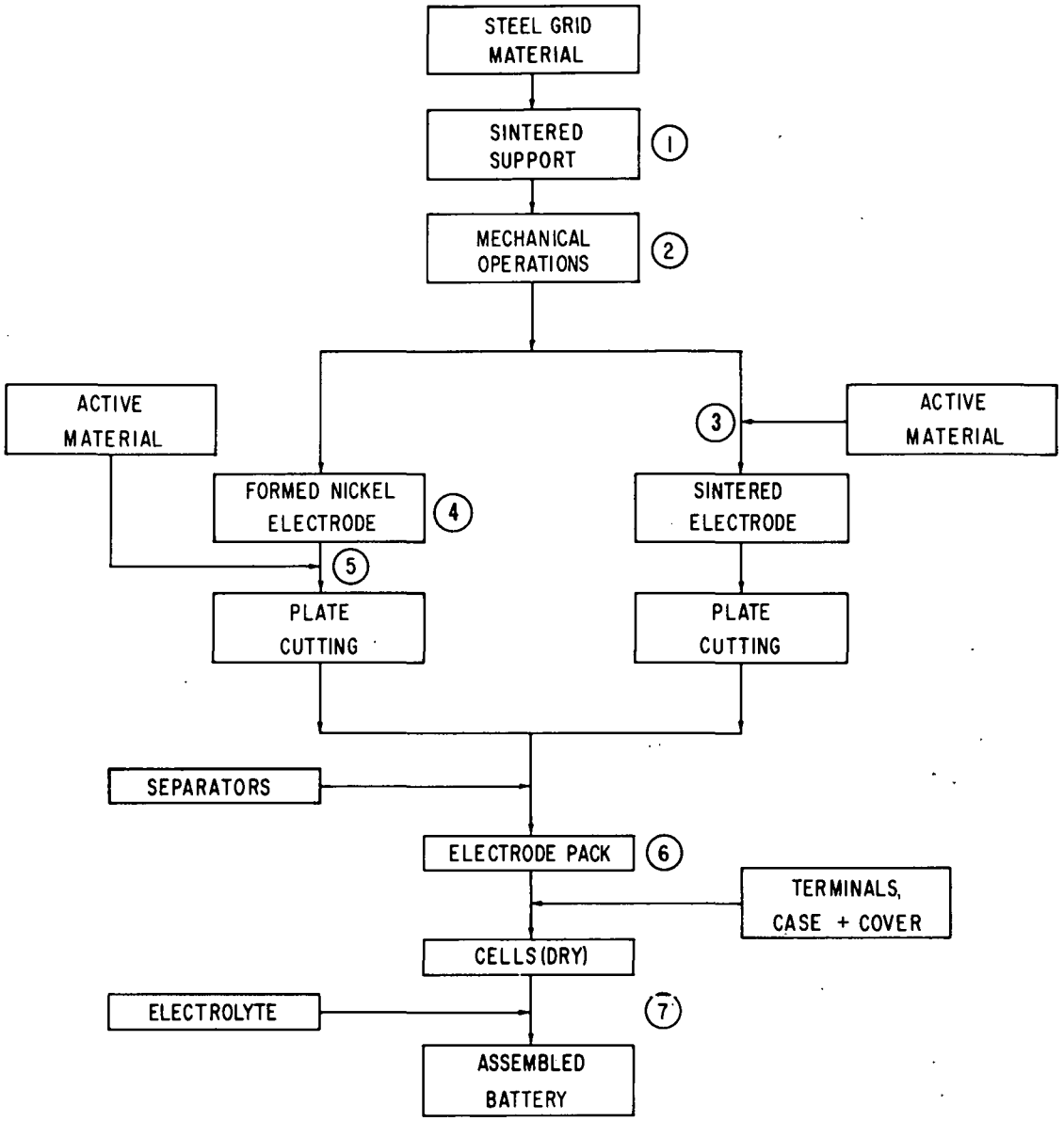

Fig. 4.1. Simplified Diagram of the Westinghouse Nickel/Iron Battery Manufacturing Process.

batteries based on the Westinghouse process; their manufacturing procedure has been modified here so that the same sintered steel support is used to fabricate both electrodes. The figure indicates seven process steps (circled and numbered) in which effluents could be released during the battery manufacture process. Because the process is not yet operational and details of Westinghouse's battery fabrication procedures are proprietary, many of the process emissions estimates given below have been based on situations that occur in like technologies.

A porous sintered grid support (Step 1 in Fig. 4.1) is prepared by powder metallurgical methods for use with both electrodes. Mechanical operations such as rolling, slitting and cutting are performed to prepare the grids for loading the active materials in the electrode preparation steps. The nickel electrode (Step 3) is electrochemically formed by nickel plating, and active material is then deposited directly in the substrate. The iron electrode (Step 4) is formed by pressing iron-active materials into the steel grid structure and sintering.

The iron and nickel plates are then stacked (Step 6) alternately (with separators between them) into an electrode pack; terminals are added; the assemblies are placed in the case; and the covers are welded to the case. Subsequently electrolyte is added and the battery module assembly is complete.

Manufacturing Emission Sources

The details of the Ni/Fe battery manufacturing process, as indicated above, are less certain than those for the Ni/Zn battery process; therefore, it is difficult to assess the sources and amounts of the emissions resulting from battery manufacture. However, the steps in which emissions could be generated (identified in Fig. 4.1) are discussed below.

During fabrication of the steel grid support for the electrodes, the preparation of the sintered support (Step 1, Fig. 4.1) can produce dust and solid cuttings, and mechanical operations (Step 2) to prepare the electrodes for impregnation with active material can produce solid cuttings and trimmings with oil residues. Subsequent vapor degreasing of the electrodes probably would produce minor amounts of solvent vapor emissions.

The iron electrode preparation involves impregnation with active material and subsequent sintering; these steps (Step 3) probably involve some wastewater emissions from active material preparation and washing, as well as some dusts and mists. The nickel electrode is prepared chiefly by electrochemical means and the emissions from these processes (Steps 4 and 5) should be very minor; probably the chief emissions would be nickel-containing waste liquids from the electroplating and impregnating steps. Wastes from the final cutting and trimming operations for the completed electrodes would be similar to those included in Step 2.

Wastes from the combination of the electrodes with the separators into an electrode pack and addition of the terminals, case and cover (Step 6) are chiefly solids. In Step 7, the electrolyte is added to the completed cells, and emissions of some mists and liquids from leakage and spillage are likely.

Estimate of Emission Magnitudes

Estimation of the amounts of materials emitted during the Ni/Fe battery manufacturing process is very difficult; however, use of the cost information presented in the Westinghouse Design and Cost Study[1] does allow order-of-magnitude estimation of these emissions. Currently available technology for dust and mist collection was assumed in estimating air emissions; this probably results in conservative values for the battery technologies predicted for the 1990s and beyond. Wastewater effluents are more difficult to estimate; we shall assume they are the same as for the Ni/Zn battery, which is probably an overestimate for the Ni/Fe battery manufacturing process. Table 4.13 summarizes the results of these estimates.

Table 4.13. Estimated Magnitude of Emissions from the Ni/Fe Battery Manufacturing Process

Effluent	*Form*	*Emissions, (kg/Day)/MWh*
Ni	In aqueous solution	0.002
Steel	Dust[a]	0.007
Fe_2O_3	Dust[a]	0.005
Electrolyte	Mist[b]	0.00005
Plastic	Solid	0.06
Wastewater	Liquid	~20

[a] *Assumes 90% efficient dust collection.*

[b] *Assumes 99% efficient mist removal.*

Impact on Other Industries

The impact of the Ni/Fe battery industry on other existing industries that would supply materials for use in battery manufacture is summarized in Table 4.14. The nickel, cobalt, KOH and LiOH industries would be significantly impacted by the year 2000. As in the case for the Ni/Zn EV battery, the cobalt and nickel requirements (chiefly from foreign suppliers) will necessitate efficient recycling of these metals in order to maintain the metal input supply. In the case of the domestic KOH and LiOH industries, significant expansion of production will be needed; the lithium requirements may be in serious competition with the needs of other EV battery systems (e.g., Li/metal sulfide) as well as the fusion power program. It seems likely that the economic impacts generated will be greater than the anticipated environmental impacts.

4.5 BATTERY USE

The anticipated uses for the Ni/Fe batteries are identical to those discussed in Section 2.5 for the lead/acid battery. Charging, maintenance and driving scenarios are outlined in that section.

Table 4.14. Impact of the Ni/Fe Battery on Industry

	Year		
	1985	*1990*	*2000*
Total EVs	85,000	410,000	8,000,000
MWh Installed Batteries	2,125	10,250	200,000
MT Battery/day	34	166	3,230
Batteries/day	~80	~400	~7,700
Projected Production of Component Materials, MT/day (% of existing production)			
Nickel[a,b]	5 (0.3)	25 (1.6)	490 (30)
Steel[a,c]	8 (0.01)	41 (.05)	800 (1.1)
Cobalt[a,d]	0.2 (0.3)	1.1 (1.8)	22 (35)
Copper[a,c]	1.2 (0.03)	5.8 (0.2)	110 (2.7)
KOH[a,e]	2.4 (0.3)	11 (1.3)	220 (26)
LiOH[a,d]	0.4 (1.1)	2.2 (5.3)	42 (100)
Plastic[a,f]	2.6 (0.1)	13 (0.6)	250 (12)

[a] *% of existing production.*

[b] *Based on 1975 world production.*

[c] *Based on 1974 U.S. production.*

[d] *Based on 1973 world production.*

[e] *Based on U.S. annual production.*

[f] *Based on 1972 U.S. production.*

The Ni/Fe cell is essentially emission-free during normal operation; however, during recharge, hydrogen evolution is relatively large, and provisions are needed to recombine the hydrogen with oxygen to form water. This, nevertheless, presents more of a design consideration than an environmental emission problem.

4.6 RECYCLING

4.6.1 Process Description

Because the Ni/Fe battery is similar to the Ni/Zn battery in many ways, the recycling operations are expected to be similar. As a result the discussion here will be brief. Section 3.6 contains a more complete discussion. The material differences in recycling are the iron electrode and the presence of some lithium hydroxide in the electrolyte.

Collection and Dismantling

The collection scheme for Ni/Fe batteries would be similar to that for lead/acid batteries. Ni/Fe batteries will be heavier than Ni/Zn batteries on a specific energy basis, which would impact transportation costs. Case dismantling could involve either complete case crushing or plate removal and separation. The latter is more likely, if sufficient nickel is present for hydrometallurgical recovery.

Metallurgical Separation

A proposed resource recovery process flow is illustrated in Figure 4.2. The process is directed primarily toward nickel recycling, whereas the other components of the battery are either disposed in a secured manner or available to other secondary scrappers. The amount of iron available for recovery is relatively insignificant in relationship to the amount of secondary iron scrap available. Consequently, the incremental environmental impacts attributed to viable secondary recovery of iron from Ni/Fe battery materials are not deemed to be significant and will not be considered further in this report.

Whereas the iron plates probably will be discarded, the nickel plates, after washing, will be processed for further recovery. The nickel plates may be dissolved in H_2SO_4 to produce $NiSO_4$ solution. The nickel solution is then recycled to produce more active material for the nickel electrode. Otherwise, although a less likely route, the grid nickel may be transported to a secondary smelter. The nickel electrodes may also be removed from the cells, washed, dried, rerolled to their original thickness and rebuilt into fresh cells, as previously reported for nickel recovery from the Ni/Zn battery.

4.6.2 Air Emissions

Collection and Dismantling

If the electrolyte is removed prior to the shipment of the batteries from the collection sites, the production of electrolytic mists may not be as prevalent as for the Ni/Zn battery. Nevertheless, the emission of caustic mists to the environment as well as in the workplace is a potential problem. Control of such mists in the workplace and beyond the plant site can be achieved by employing measures similar to those discussed for the lead/acid and Ni/Zn batteries.

Fugitive dust also will be generated in a number of steps in the front-end operations, but should not be as much a concern as in Ni/Zn batteries, as zinc or zinc oxide will not be present. Nevertheless, exhaust systems probably will be warranted, as is the case for resource recovery in general.

In the context of the limited information available and assuming control measures similar to those discussed are instituted, air emissions from front-end operations are not anticipated to be significant.

Metallurgical Separation

The air emissions from hydrometallurgical processes proposed for the Ni/Fe battery are similar to those for the Ni/Zn battery.

4.6.3 Liquid Emissions

Liquid emissions from the recycling of Ni/Fe batteries are expected to be similar to those for the Ni/Zn battery, except that oxides and hydroxides of iron, rather than zinc, are emitted. Also the discarded electrolyte includes some lithium hydroxide.

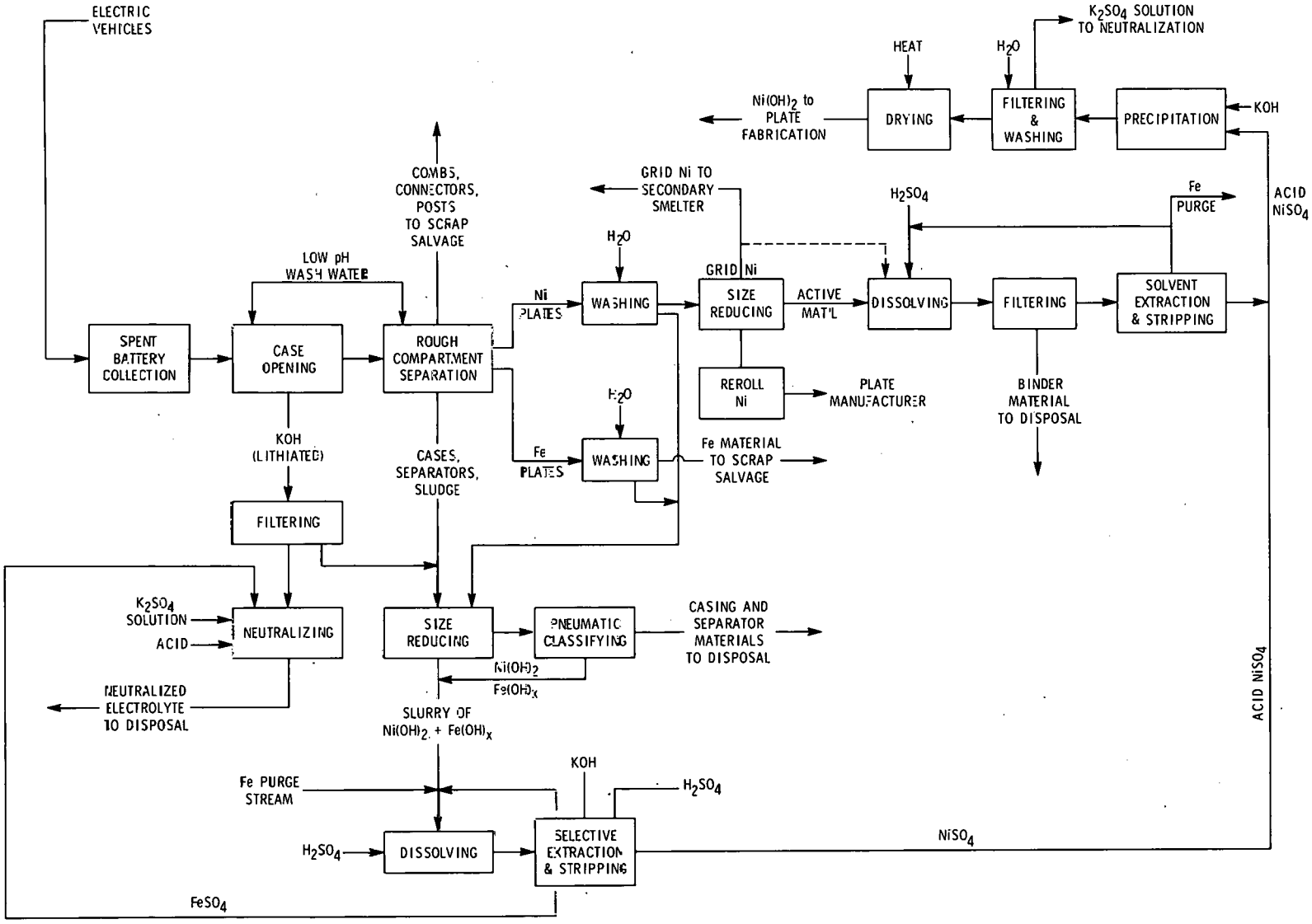

Fig. 4.2. Flowsheet for the Recovery of Nickel/Iron Electric Vehicle Batteries. Source: EIC Corp. *A Survey of Metallurgical Recycling Processes.* Draft Report to Argonne National Laboratory, ANL/OEPM-79-2, 1979.

Collection and Dismantling

The sources of liquid emissions are essentially the same as those given for the Ni/Zn battery. Most of the electrolyte will probably be discarded before the batteries are shipped to the recycling plants, and wetting the batteries during transport is not as important as for the Ni/Zn batteries. The greater density of the Ni/Fe batteries suggests that scrappers may arrange to discharge the electrolyte prior to transportation.

Metallurgical Separation

The recovery process for the nickel plates is similar to that for the Ni/Zn battery, and pollutants in the raw wastewater would be similar. The main difference is that suspended particles of iron as the metal, oxide or hydroxide would occur rather than of zinc, and that some LiOH will be present.

The waste pollution control technology is essentially the same as that for the liquid emissions associated with the Ni/Zn battery.

4.6.4 Solid Wastes

Collection and Dismantling

It is possible that because less electrolyte will be treated, less sludge may be generated than from Ni/Zn metallurgical processing site. If this is the case, more electrolyte will be shipped elsewhere, with a possible increase in sludge at those sites. The total amount of electrolyte to be disposed of is similar to that for the Ni/Zn battery.

Metallurgical Separation

The main difference in the metallurgical processing for the Ni/Fe battery compared to the Ni/Zn battery is the electrode material. The spent electrode can either be discarded or recycled; however, since iron is cheap and abundant and it is not considered as a toxic metal, the electrodes will probably be discarded in landfills.

Solid waste management options and processes are expected to be the same as those used for the Ni/Zn battery (see Sec. 3.6.4).

4.7 SUMMARY

1. Impacts of the nickel/iron battery-powered electric vehicles on the total cycle of the nickel/iron battery industry were assessed. The market penetration was assumed to grow at a rate of 30% per year and to reach a total of eight million nickel/iron battery-powered electric vehicles on the road by the year 2000. Assessments were also made for per megawatt hour of installed capacity, assuming one-megawatt hour requirement for each 40 electric vehicles. The assessment included consideration of resource requirements, industrial growth, and effluents to the environment from various phases of the total battery cycle.

2. For the assumed scenario, by the year 2000:

   a. a 30% increase in refined nickel production will be required as compared to current world output,

   b. a 35% increase in cobalt production will be required as compared to current world output,

   c. a 26% increase in KOH production will be required, as compared to current U.S. output;

   d. a 100% increase in LiOH production will be required over current U.S. output;

   e. a Ni/Fe battery recycle industry will need to be developed because of the scarcity and cost of nickel and cobalt.

3. For the assumed scenario, by the year 2000 major air-borne emissions are expected at the following levels (assuming current standards and/or control technology are applied).

   a. 380 MT/day of SO_2 from primary nickel production;

   b. 8 MT/day of CO from iron and steel production;

   c. 10 MT/day of particulates from primary nickel production.

4. Liquid emissions for the assumed scenario, by the year 2000, are expected as follows:

   a. 20 MT/day of wastewater discharges from steel production;

   b. 170,000 MT/day of wastewater discharges from nickel production (includes mining, milling, smelting, and refining);

   c. 1600 MT/day of wastewater discharges from the LiOH industry.

   Treatment of the wastewaters before discharge will be necessary.

5. For the assumed scenario, by the year 2000 the following solid wastes are expected to be produced (assuming current standards and/or control technology):

   a. 100 MT/day of slag from steel production;

   b. 2800 MT/day of slag from LiOH production (chiefly from mining and milling).

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5. DISPERSION OF EFFLUENTS

ABSTRACT

Generic dispersion analyses are given for battery-related emission sources identified in Sections 2 through 4. Dispersion data in terms of resulting environmental concentrations are summarized for the year 2000. The following scenario is used: with 40 EVs equivalent to 1 MWh in each case, 3×10^6 EVs with lead/acid batteries totaling 7.5×10^4 MWh, and 8×10^6 EVs with either Ni/Zn or Ni/Fe batteries totaling 2.0×10^5 MWh. The information presented serves as a data base for subsequent sections of this report. These dispersion data are referred to wherever appropriate in Sections 6 through 8.

5.1 ATMOSPHERIC DISPERSION ANALYSIS

The effluent dispersion scenarios used here are ones that approach worst-case conditions for atmospheric concentrations, deposition rates, deposition rates into the soil, and runoff concentrations. The calculations are based on the atmospheric dispersion model of Turner.[1] In this model the short-term ground level concentration, $\chi(x,y)$ of an effluent at a position (x,y) where the source is at the origin ($x = 0$ and $y = 0$) is given by:

$$\chi(x,y) = \frac{Q}{u\pi\sigma_y(x)\sigma_z(x)} \exp\left[-\frac{1}{2}\left(\frac{y}{\sigma_y(x)}\right)^2\right] \exp\left[-\frac{1}{2}\left(\frac{h}{\sigma_z(x)}\right)^2\right] . \qquad (1)$$

where:

Q = effluent emission rate of the source,

u = wind velocity,

$\sigma_y(x)$ = horizontal dispersion coefficient of the plume,

$\sigma_z(x)$ = vertical dispersion coefficient of the plume, and

h = effective release height of the effluent (the height at which the plume becomes horizontal).

In the above the wind velocity is assumed to be fixed along the x axis and in the direction of increasing x. $\sigma_y(x)$ and $\sigma_z(x)$ depend on both the downwind distance from the source and the atmospheric stability class that is being considered.

The ground-level effluent concentration directly under the plume $\chi(x,0)$ is obtained from the above by setting $y = 0$. One has

$$\chi(x,0) = \frac{Q}{u\pi\sigma_y(x)\sigma_z(x)} \exp\left[-\frac{1}{2}\left(\frac{h}{\sigma_z(x)}\right)^2\right] . \qquad (2)$$

For the atmospheric stability class D, which is the one considered here,[1] the graphs of the horizontal and vertical dispersion coefficients as a function of downwind distance given by

Turner[1] are well represented by simple functions. That is, $\sigma_y(x)$ and $\sigma_z(x)$, in km, are given by

$$\sigma_y(x) = 0.070x^{0.9} \tag{3}$$

and

$$\sigma_z(x) = 0.032x^{d} , \tag{4}$$

where d = 0.83 if $0 \leq x < 1$ and d = 0.63 if $x \geq 1$. The wind speed is taken here to be 6 m/s.

The worst-case deposition rate is that obtained directly under the plume if one assumes that the wind always blows in the same direction and that at each point the deposition rate is proportional to the ground-level atmospheric concentrations (which are calculated assuming total reflection from the ground). Based on this assumption effluent deposition rates are calculated for sectors of successive rings or annuli whose angular width is sufficiently small so that Equation 2 is valid for all points in the sectors.

The normalization factor is obtained by assuming that all the material omitted is deposited within 50 km downwind of the source. Then the fraction of material deposited in a small sector lying between radial distances r_1 and r_2 under the plume divided by the area of the sector is given by

$$G(r_1,r_2) = \frac{\sqrt{\frac{2}{\pi}}\int_{r_1}^{r_2} \chi(r,0)rdr}{\left(r_2^2 - r_1^2\right)\int_0^{50} \sigma_y(x)\chi(x,0)dx} . \tag{5}$$

To obtain this result it is convenient to convert to polar coordinates and to note that in the annular section under consideration $x \simeq r$. $\chi(r,0)$ is given by Eq. 2 with $x \simeq r$. The integral in the denominator of Equation 5 was obtained by integrating Equation 1 over y from $-\infty$ to $+\infty$.

The effluent deposition rate under the plume and between r_1 and r_2 is given by

$$aQG(r_1,r_2) , \tag{6}$$

where Q is the effluent emission rate and a is a constant that depends on the units. If Q is given in kg/day and $\sigma_y(x)$ and $\sigma_z(x)$ in km and the deposition rate is in $\mu g/m^2 \cdot yr$, then $a = 3.65 \times 10^5$ ($\mu g/kg$) × (day/yr) × (km^2/m^2).

The incremental soil concentration of an effluent is obtained from the deposition rate by assuming that all the effluent deposited is retained in the top 3 cm of the soil and that the bulk density of the soil is 1.5 g/cm^3.[2] The incremental soil concentration of an effluent in $\mu g/g \cdot yr$ is given by

$$baQG(r_1,r_2) , \tag{7}$$

where $b = (10 \times 3 \times 1.5)^{-1} = 2.22 \times 10^{-5}$ m^2/gm.

The effluent runoff concentration under the plume was obtained by assuming an annual precipitation of one meter water equivalent and that all the effluent deposited is dissolved or suspended in the water and none retained in the soil. This worst-case runoff concentration in μg/L is given by

$$caQG(r_1,r_2) \ , \tag{8}$$

where $c = 10^{-3}$ m^2 yr/L.

Under the assumptions used to obtain the runoff concentration one sees that the concentration is independent of what fraction of the precipitation runs off and what fraction is retained by the soil. Later on it will be assumed that one fourth of the precipitation runs off and the remainder is retained by the soil.

Based on existing industries, an effective release height of 70 m is assumed for stack releases from the primary and secondary lead smelter-refineries, primary zinc smelters, caustic potash and LiOH producing complexes, and primary nickel smelters. An effective release height of 5 m is assumed for lead mine-mills, zinc mine-mills, battery manufacturing plants, and polypropylene manufacturing plants. Fugitive emissions are considered to be point sources. Emission rates, reported earlier, were used for calculations. Controlled rates were used when reported. The primary and secondary lead smelter emissions consist of both fugitive and stack emissions.

It must be emphasized that the model used here is very approximate. Besides the assumption of a fixed wind direction, variations in atmospheric conditions, source characteristics, topographical influences, vegetative cover, and a host of other variables all influence dispersion and accuracy of predicted concentrations. No currently available models can predict concentrations with great accuracy. Turner[1] indicated that centerline concentrations, used in this report for a ground-level release, are accurate to a factor of 3, but that elevated stacks and uneven terrain can lead to predictions that disagree with observations by even larger factors.

Resulting environmental concentrations are presented in tabular form for battery-related emissions. Details for intermediate distances are found in Appendix C. The tables for atmospheric dispersion are given as outlined below.

5.1.1 Lead/Acid Battery Cycle

Primary Production of Input Materials

Model Lead Mine-Mill Complex - Table 5.1

Model Primary Lead Smelting and Refining Complex - Table 5.2

Model Secondary Lead Refinery Complex

Model Secondary Lead Smelter - Table 5.3

Manufacturing Processes

Model Battery Manufacturing Plant - Table 5.4

Model Polypropylene Manufacturing Plant - Table 5.5

5.1.2 Ni/Zn Battery Cycle

Primary Production of Input Materials

Model Zinc Mine-Mill Complex - Table 5.6

Model Primary Zinc Smelter - Table 5.7

Model Primary Nickel Smelter - Table 5.8

Model Caustic Potash Plant - Table 5.9

Manufacturing Processes

Model Battery Manufacturing Plant - Table 5.10

Model Polypropylene Manufacturing Plant - Table 5.11

5.1.3 Ni/Fe Battery Cycle

Primary Production of Input Materials

Model Iron and Steel Complex - Table 5.12

Model Lithium Hydroxide Plant - Table 5.13

Model Primary Nickel Smelter - Table 5.14

Model Caustic Potash Plant - Table 5.15

Manufacturing Processes

Model Battery Manufacturing Plant - Table 5.16

Model Polypropylene Manufacturing Plant - Table 5.17

5.2 WASTEWATER DISPERSION ANALYSIS

Generic wastewater dispersion analyses are made here for battery-related, potentially hazardous, emission sources identified and quantified in earlier chapters. Polypropylene industries and LiOH manufacturing industries are therefore not included. The dispersion analyses are based on recommended or promulgated effluent concentration limitations, when possible, or on representative effluent constituent concentrations.[3-6] Resulting environmental concentrations are presented in tables as outlined below for battery-related emissions.

5.2.1 Lead/Acid Battery Cycle

Primary Production of Input Materials

Model Lead Mine-Mill Complex - Table 5.18

Model Primary Lead Smelter-Refinery Complex - Table 5.19

Model Battery Manufacturing Plant

Wet Process - Table 5.20

Dry Process - Table 5.21

Battery Recycling - Table 5.22

5.2.2 Ni/Zn Battery Cycle

Primary Production of Input Materials

Model Zinc Mine-Mill Complex - Table 5.23

Model Nickel Mining, Milling, Smelting, and Refining Operations - Table 5.24

Model Primary Zinc Industry - Table 5.25
Model Caustic Potash Producing Complex - Table 5.26

Model Battery Manufacturing Plant - Table 5.27

5.2.3 Ni/Fe Battery Cycle

Primary Production of Input Materials

Model Lithium Mine-Mill Complex - Table 5.28
Model Nickel Mining, Milling, Smelting and Refining Operations - Table 5.29
Model Caustic Potash Producing Complex - Table 5.30
Model Iron-Steel Producing Complex - Table 5.31

Model Battery Manufacturing Plant - Table 5.32

5.3 SOLID WASTES DISPERSION ANALYSIS

It is assumed that all potentially hazardous wastes generated from battery-related activities will be contained and disposed of as required by the Resource Conservation and Recovery Act of 1976 (P.L. 94-580). Solid wastes, therefore, will not be discussed further because secured disposal sites are assumed to be permanent sinks for solid wastes. However, the potential for release of hazardous solid wastes into the environment from secured disposal sites does exist.

5.4 SUMMARY

1. The generic dispersion analyses are presented for emissions from the total battery cycle for each of the near-term batteries. The amounts and sources of emissions used in the dispersion analyses are as identified in Sections 2, 3, and 4.

2. Atmospheric concentrations, deposition rates, soil concentrations, surface water runoff concentrations, and lake water concentrations were calculated for conditions approaching the worst case.

3. Wastewater dispersion analyses were made only for potentially hazardous emissions.

4. Detailed dispersion analyses are not provided for solid wastes; it is assumed that potentially hazardous wastes will be contained and disposed of as required under the Resource Conservation and Recovery Act of 1976.

5. Analyses are presented for the total installed capacity in the year 2000 for various near-term batteries, as well as for unit megawatt hour.

6. Analyses are presented in tabular form only; concentration levels for near-field and far-field are given in the tables in the main text and for intermediate distances in tables in Appendix C.

TABLE 5.1. DISPERSION OF TOTAL EMISSIONS FROM MODEL LEAD MINE-MILL COMPLEX

	DISTANCE KM	PARTICULATES	LEAD	COPPER	ZINC	IRON	COBALT	NICKEL
GROUND-LEVEL	1	3.3E+03	1.5E+02	6.7E+00	8.7E+01	5.3E+01	4.0E-01	5.7E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	8.4E+00	3.7E-01	1.7E-02	2.2E-01	1.4E-01	1.0E-03	1.4E-03
DEPOSITION RATES	1- 2	1.2E+09	5.5E+07	2.5E+06	3.2E+07	2.0E+07	1.5E+05	2.1E+05
(MICROGRAMS/M**2/YR)	30-50	8.6E+06	3.8E+05	1.7E+04	2.3E+05	1.4E+05	1.0E+03	1.5E+03
INCREMENTAL SOIL	1- 2	2.7E+04	1.2E+03	5.5E+01	7.2E+02	4.4E+02	3.3E+00	4.7E+00
CONCENTRATIONS	30-50	1.9E+02	8.5E+00	3.8E-01	5.0E+00	3.1E+00	2.3E-02	3.3E-02
(MICROGRAMS/G/YR)								
RUNOFF	1- 2	1.2E+06	5.5E+04	2.5E+03	3.2E+04	2.0E+04	1.5E+02	2.1E+02
CONCENTRATIONS	30-50	8.6E+03	3.8E+02	1.7E+01	2.3E+02	1.4E+02	1.0E+00	1.5E+00
(MICROGRAMS/L)								

	DISTANCE KM	CADMIUM	MANGANESE	CARBON MONOXIDE	SULFUR OXIDES	HYDRO-CARBONS	ALDEHYDES	ORGANIC ACIDS
GROUND-LEVEL	1	4.0E-01	5.3E+00	1.1E+01	4.7E+00	6.0E+00	6.7E-01	4.7E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	1.0E-03	1.4E-02	2.7E-02	1.2E-02	1.5E-02	1.7E-03	1.2E-03
DEPOSITION RATES	1- 2	1.5E+05	2.0E+06	4.0E+06	1.7E+06	2.2E+06	2.5E+05	1.7E+05
(MICROGRAMS/M**2/YR)	30-50	1.0E+03	1.4E+04	2.8E+04	1.2E+04	1.6E+04	1.7E+03	1.2E+03
INCREMENTAL SOIL	1- 2	3.3E+00	4.4E+01	8.8E+01	3.9E+01	5.0E+01	5.5E+00	3.9E+00
CONCENTRATIONS	30-50	2.3E-02	3.1E-01	6.2E-01	2.7E-01	3.5E-01	3.8E-02	2.7E-02
(MICROGRAMS/G/YR)								
RUNOFF	1- 2	1.5E+02	2.0E+03	4.0E+03	1.7E+03	2.2E+03	2.5E+02	1.7E+02
CONCENTRATIONS	30-50	1.0E+00	1.4E+01	2.8E+01	1.2E+01	1.6E+01	1.7E+00	1.2E+00
(MICROGRAMS/L)								

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.1-C.4 IN APPENDIX C USING A CONVERSION FACTOR OF 12295 MWH/COMPLEX (TABLE 2.7)

TABLE 5.2 DISPERSION OF TOTAL STACK EMISSIONS FROM MODEL PRIMARY LEAD SMELTING AND REFINING COMPLEX

	DISTANCE KM	PARTICULATES	LEAD	SULFUR DIOXIDE
GROUND-LEVEL	1	2.6E+01	7.8E+00	6.1E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	7.0E-01	2.1E-01	1.6E-02
DEPOSITION RATES	1- 2	9.3E+07	2.7E+07	1.6E+06
(MICROGRAMS/M**2/YR)	30-50	8.2E+05	2.5E+05	2.3E+04
INCREMENTAL SOIL	1- 2	2.1E+03	5.9E+02	3.6E+01
CONCENTRATIONS	30-50	1.8E+01	5.7E+00	5.0E-01
(MICROGRAMS/G/YR)				
RUNOFF	1- 2	9.3E+04	2.7E+04	1.6E+03
CONCENTRATIONS	30-50	8.2E+02	2.5E+02	2.3E+01
(MICROGRAMS/L)				

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.5-C.8 IN APPENDIX C USING A CONVERSION FACTOR OF 18750 MWH/COMPLEX (TABLE 2.11)

TABLE 5.3 DISPERSION OF TOTAL EMISSIONS FROM MODEL SECONDARY LEAD SMELTER

	DISTANCE KM	PARTICULATES	LEAD	ANTIMONY	ARSENIC	SULFUR DIOXIDE
GROUND-LEVEL	1	7.4E+00	1.7E+00	3.4E-02	5.9E-04	9.1E+00
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	2.0E-01	4.6E-02	9.2E-04	1.6E-05	2.5E-01
DEPOSITION RATES	1- 2	2.9E+07	6.7E+06	1.3E+05	2.3E+03	2.4E+07
(MICROGRAMS/M**2/YR)	30-50	2.1E+05	4.9E+04	9.8E+02	1.7E+01	3.4E+05
INCREMENTAL SOIL	1- 2	6.5E+02	1.5E+02	3.0E+00	5.1E-02	5.4E+02
CONCENTRATIONS (MICROGRAMS/G/YR)	30-50	4.7E+00	1.1E+00	2.2E-02	3.7E-04	7.6E+00
RUNOFF	1- 2	2.9E+04	6.7E+03	1.3E+02	2.3E+00	2.4E+04
CONCENTRATIONS (MICROGRAMS/L)	30-50	2.1E+02	4.9E+01	9.8E-01	1.7E-02	3.4E+02

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.9-C.12 IN APPENDIX C USING A CONVERSION FACTOR OF 5770 MWH/COMPLEX (TABLE 2.28)

TABLE 5.4 DISPERSION OF TOTAL EMISSIONS FROM MODEL LEAD/ACID BATTERY MANUFACTURING PLANT

	DISTANCE KM	PARTICULATES	LEAD	LEAD FUMES	LEAD OXIDES	LEAD SULFATES
GROUND-LEVEL	1	2.5E+00	1.2E+00	8.5E-02	9.7E-01	1.4E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	6.3E-03	3.1E-03	2.2E-04	2.5E-03	3.6E-04
DEPOSITION RATES	1- 2	9.2E+05	4.6E+05	3.2E+04	3.6E+05	5.3E+04
(MICROGRAMS/M**2/YR)	30-50	6.4E+03	3.2E+03	2.2E+02	2.5E+03	3.7E+02
INCREMENTAL SOIL	1- 2	2.1E+01	1.0E+01	7.0E-01	8.1E+00	1.2E+00
CONCENTRATIONS (MICROGRAMS/G/YR)	30-50	1.4E-01	7.1E-02	4.9E-03	5.6E-02	8.2E-03
RUNOFF	1- 2	9.2E+02	4.6E+02	3.2E+01	3.6E+02	5.3E+01
CONCENTRATIONS (MICROGRAMS/L)	30-50	6.4E+00	3.2E+00	2.2E-01	2.5E+00	3.7E-01

	DISTANCE KM	ANTIMONY	ARSENIC	EXPANDER	SEPARATOR	SULFURIC ACID
GROUND-LEVEL	1	1.7E-03	3.0E-05	2.3E-03	6.4E-04	6.7E-02
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	4.3E-06	7.6E-08	5.8E-06	1.6E-06	1.7E-04
DEPOSITION RATES	1- 2	6.3E+02	1.1E+01	8.6E+02	2.4E+02	2.5E+04
(MICROGRAMS/M**2/YR)	30-50	4.4E+00	7.8E-02	6.0E+00	1.7E+00	1.7E+02
INCREMENTAL SOIL	1- 2	1.4E-02	2.5E-04	1.9E-02	5.3E-03	5.6E-01
CONCENTRATIONS (MICROGRAMS/G/YR)	30-50	9.7E-05	1.7E-06	1.3E-04	3.7E-05	3.9E-03
RUNOFF	1- 2	6.3E-01	1.1E-02	8.6E-01	2.4E-01	2.5E+01
CONCENTRATIONS (MICROGRAMS/L)	30-50	4.4E-03	7.8E-05	6.0E-03	1.7E-03	1.7E-01

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.13-C.16 IN APPENDIX C USING A CONVERSION FACTOR OF 3260 MWH/COMPLEX (TABLE 2.20)

TABLE 5.5 DISPERSION OF TOTAL EMISSIONS
FROM MODEL POLYPROPYLENE MANUFACTURING PLANT

	DISTANCE KM	PARTICULATES	PROPYLENE
GROUND-LEVEL	1	7.7E+01	1.8E+01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	2.0E-01	4.5E-02
DEPOSITION RATES	1- 2	2.9E+07	6.6E+06
(MICROGRAMS/M**2/YR)	30-50	2.0E+05	4.6E+04
INCREMENTAL SOIL	1- 2	6.4E+02	1.5E+02
CONCENTRATIONS	30-50	4.5E+00	1.0E+00
(MICROGRAMS/G/YR)			
RUNOFF	1- 2	2.9E+04	6.6E+03
CONCENTRATIONS	30-50	2.0E+02	4.6E+01
(MICROGRAMS/L)			

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.17-C.20 IN APPENDIX C USING A CONVERSION FACTOR OF 75,000 MWH/COMPLEX (TABLE 2.15)

TABLE 5.6 DISPERSION OF TOTAL EMISSIONS FROM MODEL ZINC MINE-MILL COMPLEX

	DISTANCE KM	PARTICULATES	LEAD	ZINC	COBALT	NICKEL	CADMIUM	MANGANESE
GROUND-LEVEL	1	1.2E+01	7.0E-01	4.7E-01	2.3E-03	2.3E-03	2.3E-03	2.3E-02
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	3.0E-02	1.8E-03	1.2E-03	5.9E-06	5.9E-06	5.9E-06	5.9E-05
DEPOSITION RATES	1- 2	4.4E+06	2.6E+05	1.7E+05	8.7E+02	8.7E+02	8.7E+02	8.7E+03
(MICROGRAMS/M**2/YR)	30-50	3.0E+04	1.8E+03	1.2E+03	6.1E+00	6.1E+00	6.1E+00	6.1E+01
INCREMENTAL SOIL	1- 2	9.7E+01	5.8E+00	3.9E+00	1.9E-02	1.9E-02	1.9E-02	1.9E-01
CONCENTRATIONS	30-50	6.7E-01	4.0E-02	2.7E-02	1.3E-04	1.3E-04	1.3E-04	1.3E-03
(MICROGRAMS/G/YR)								
RUNOFF	1- 2	4.4E+03	2.6E+02	1.7E+02	8.7E-01	8.7E-01	8.7E-01	8.7E+00
CONCENTRATIONS	30-50	3.0E+01	1.8E+00	1.2E+00	6.1E-03	6.1E-03	6.1E-03	6.1E-02
(MICROGRAMS/L)								

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.21-C.24 IN APPENDIX C USING AN ASSUMED LARGE MINE-MILL PRODUCTION OF 36,000 MTZN/YR (MINERALS YEARBOOK VOL. I METALS, MINERALS, AND FUELS, U.S. DEPT OF INTERIOR, 1976) AND A FLOW RATE OF 0.84 MTZN/YR*MWH (TABLE 3.2) TO GIVE 43,000 MWH/COMPLEX

TABLE 5.7 DISPERSION OF TOTAL EMISSIONS FROM MODEL PRIMARY ZINC SMELTER

	DISTANCE KM	ARSENIC	ANTIMONY	CADMIUM	LEAD	ZINC	SULFUR DIOXIDE
GROUND-LEVEL	1	2.2E-03	2.2E-03	7.8E-02	1.0E-02	1.3E+00	3.0E+02
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	5.9E-05	5.9E-05	2.1E-03	2.8E-04	3.4E-02	8.1E+00
DEPOSITION RATES	1- 2	5.8E+03	5.8E+03	2.1E+05	2.7E+04	3.3E+06	8.0E+08
(MICROGRAMS/M**2/YR)	30-50	8.2E+01	8.2E+01	2.9E+03	3.9E+02	4.7E+04	1.1E+07
INCREMENTAL SOIL	1- 2	1.3E-01	1.3E-01	4.6E+00	6.1E-01	7.4E+01	1.8E+04
CONCENTRATIONS	30-50	1.8E-03	1.8E-03	6.5E-02	8.6E-03	1.0E+00	2.5E+02
(MICROGRAMS/G/YR)							
RUNOFF	1- 2	5.8E+00	5.8E+00	2.1E+02	2.7E+01	3.3E+03	8.0E+05
CONCENTRATIONS	30-50	8.2E-02	8.2E-02	2.9E+00	3.9E-01	4.7E+01	1.1E+04
(MICROGRAMS/L)							

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.25-C.28 IN APPENDIX C USING A CONVERSION FACTOR OF 125,000 MWH/COMPLEX (TABLE 3.7)

TABLE 5.8 DISPERSION OF TOTAL EMISSIONS FROM MODEL PRIMARY NICKEL SMELTER

	DISTANCE KM	PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
GROUND-LEVEL	1	2.0E+02	2.3E+01	5.0E-01	2.0E+00	7.5E+03
ATMOSPHERIC CONCENTRATIONS,	50	5.5E+00	6.3E-01	1.4E-02	5.4E-02	2.0E+02
(MICROGRAMS/M**3)						
DEPOSITION RATES	1- 2	5.4E+08	6.2E+07	1.3E+06	5.3E+06	2.0E+10
(MICROGRAMS/M**2/YR)	30-50	7.6E+06	8.7E+05	1.9E+04	7.5E+04	2.8E+08
INCREMENTAL SOIL	1- 2	1.2E+04	1.4E+03	3.0E+01	1.2E+02	4.4E+05
CONCENTRATIONS	30-50	1.7E+02	1.9E+01	4.2E-01	1.7E+00	6.3E+03
(MICROGRAMS/G/YR)						
RUNOFF	1- 2	5.4E+05	6.2E+04	1.3E+03	5.3E+03	2.0E+07
CONCENTRATIONS	30-50	7.6E+03	8.7E+02	1.9E+01	7.5E+01	2.8E+05
(MICROGRAMS/L)						

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.29-C.32 IN APPENDIX C USING AN ESTIMATED CONVERSION FACTOR OF 100,000 MWH/COMPLEX

TABLE 5.9 DISPERSION OF TOTAL EMISSIONS FROM MODEL CAUSTIC POTASH PLANT

	DISTANCE KM	CHLORINE	CARBON MONOXIDE
GROUND-LEVEL	1	1.0E-02	1.8E-01
ATMOSPHERIC CONCENTRATIONS,	50	2.7E-04	4.7E-03
(MICROGRAMS/M**3)			

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.33 IN APPENDIX C USING A CONVERSION FACTOR OF 100,000 MWH/COMPLEX (TABLE3.11)

TABLE 5.10 DISPERSION OF TOTAL EMISSIONS FROM MODEL NICKEL/ZINC BATTERY MANUFACTURING PLANT

	DISTANCE KM	NICKEL	ZINC OXIDE	COBALT	ELECTROLYTE
GROUND-LEVEL	1	5.2E+00	4.7E+00	2.7E-01	2.7E-02
ATMOSPHERIC CONCENTRATIONS,	50	1.3E-02	1.2E-02	6.9E-04	6.9E-05
(MICROGRAMS/M**3)					
DEPOSITION RATES	1- 2	1.9E+06	1.7E+06	1.0E+05	1.0E+04
(MICROGRAMS/M**2/YR)	30-50	1.4E+04	1.2E+04	7.0E+02	7.0E+01
INCREMENTAL SOIL	1- 2	4.3E+01	3.9E+01	2.2E+00	2.2E-01
CONCENTRATIONS	30-50	3.0E-01	2.7E-01	1.6E-02	1.6E-03
(MICROGRAMS/G/YR)					
RUNOFF	1- 2	1.9E+03	1.7E+03	1.0E+02	1.0E+01
CONCENTRATIONS	30-50	1.4E+01	1.2E+01	7.0E-01	7.0E-02
(MICROGRAMS/L)					

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.34-C.37 IN APPENDIX C USING AN ESTIMATED CONVERSION FACTOR OF 4000 MWH/COMPLEX

TABLE 5.11 DISPERSION OF TOTAL EMISSIONS
FROM MODEL POLYPROPYLENE MANUFACTURING PLANT

	DISTANCE KM	PARTICULATES	PROPYLENE
GROUND-LEVEL	1	7.0E+01	1.7E+01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	1.8E-01	4.3E-02
DEPOSITION RATES	1- 2	2.6E+07	6.3E+06
(MICROGRAMS/M**2/YR)	30-50	1.8E+05	4.4E+04
INCREMENTAL SOIL	1- 2	5.8E+02	1.4E+02
CONCENTRATIONS	30-50	4.1E+00	9.7E-01
(MICROGRAMS/G/YR)			
RUNOFF	1- 2	2.6E+04	6.3E+03
CONCENTRATIONS	30-50	1.8E+02	4.4E+01
(MICROGRAMS/L)			

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.38-C.41 APPENDIX CUSING A CONVERSION FACTOR OF 200,000 MWH/COMPLEX (TABLE 3.13)

TABLE 5.12 DISPERSION OF TOTAL EMISSIONS FROM MODEL IRON AND STEEL COMPLEX

	DISTANCE KM	PARTICULATES	CHROMIUM	COPPER	MANGANESE	NICKEL	LEAD	ZINC
GROUND-LEVEL	1	5.0E+00	5.0E-03	1.0E-02	2.0E-01	1.0E-03	1.0E-01	5.0E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	1.4E-01	1.4E-04	2.7E-04	5.4E-03	2.7E-05	2.7E-03	1.4E-02
DEPOSITION RATES	1- 2	1.3E+07	1.3E+04	2.7E+04	5.3E+05	2.7E+03	2.7E+05	1.3E+06
(MICROGRAMS/M**2/YR)	30-50	1.9E+05	1.9E+02	3.8E+02	7.5E+03	3.8E+01	3.8E+03	1.9E+04
INCREMENTAL SOIL	1- 2	3.0E+02	3.0E-01	5.9E-01	1.2E+01	5.9E-02	5.9E+00	3.0E+01
CONCENTRATIONS	30-50	4.2E+00	4.2E-03	8.4E-03	1.7E-01	8.4E-04	8.4E-02	4.2E-01
(MICROGRAMS/G/YR)								
RUNOFF	1- 2	1.3E+04	1.3E+01	2.7E+01	5.3E+02	2.7E+00	2.7E+02	1.3E+03
CONCENTRATIONS	30-50	1.9E+02	1.9E-01	3.8E-01	7.5E+00	3.8E-02	3.8E+00	1.9E+01
(MICROGRAMS/L)								

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.42-C.45 IN APPENDIX C USING AN ESTIMATED CONVERSION FACTOR OF 200,000 MWH/COMPLEX

TABLE 5.13 DISPERSION OF TOTAL EMISSIONS
FROM MODEL LITHIUM HYDROXIDE PLANT

	DISTANCE KM	PARTICULATES
GROUND-LEVEL	1	8.0E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	2.2E-02
DEPOSITION RATES	1- 2	2.1E+06
(MICROGRAMS/M**2/YR)	30-50	3.0E+04
INCREMENTAL SOIL	1- 2	4.7E+01
CONCENTRATIONS	30-50	6.7E-01
(MICROGRAMS/G/YR)		
RUNOFF	1- 2	2.1E+03
CONCENTRATIONS	30-50	3.0E+01
(MICROGRAMS/L)		

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.46-C.49 IN APPENDIX C USING A CONVERSION FACTOR OF 40,000 MWH/COMPLEX (TABLE 4.10)

TABLE 5.14 DISPERSION OF TOTAL EMISSIONS FROM MODEL PRIMARY NICKEL SMELTER

	DISTANCE KM	TOTAL PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
GROUND-LEVEL	1	1.3E+02	1.5E+01	2.5E-01	1.3E+00	4.8E+03
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	3.5E+00	4.1E-01	6.8E-03	3.4E-02	1.3E+02
DEPOSITION RATES	1- 2	3.4E+08	4.0E+07	6.7E+05	3.3E+06	1.3E+10
(MICROGRAMS/M**2/YR)	30-50	4.8E+06	5.6E+05	9.4E+03	4.7E+04	1.8E+08
INCREMENTAL SOIL	1- 2	7.5E+03	8.9E+02	1.5E+01	7.4E+01	2.8E+05
CONCENTRATIONS	30-50	1.1E+02	1.3E+01	2.1E-01	1.0E+00	4.0E+03
(MICROGRAMS/G/YR)						
RUNOFF	1- 2	3.4E+05	4.0E+04	6.7E+02	3.3E+03	1.3E+07
CONCENTRATIONS	30-50	4.8E+03	5.6E+02	9.4E+00	4.7E+01	1.8E+05
(MICROGRAMS/L)						

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.50-C.53 IN APPENDIX C USING AN ESTIMATED CONVERSION FACTOR OF 100,000 MWH/COMPLEX

TABLE 5.15 DISPERSION OF TOTAL EMISSIONS FROM MODEL CAUSTIC POTASH PLANT

	DISTANCE KM	CHLORINE	CARBON MONOXIDE
GROUND-LEVEL	1	7.5E-03	1.5E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	2.0E-04	4.1E-03

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.54 IN APPENDIX C USING A CONVERSION FACTOR OF 100,000 MWH/COMPLEX (TABLE 4.8)

TABLE 5.16 DISPERSION OF TOTAL EMISSIONS FROM MODEL NICKEL/IRON BATTERY MANUFACTURING PLANT

	DISTANCE KM	STEEL	FERRIC OXIDE	ELECTROLYTE
GROUND-LEVEL	1	3.8E+01	2.7E+01	2.7E-01
ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	50	9.6E-02	6.9E-02	6.9E-04
DEPOSITION RATES	1- 2	1.4E+07	1.0E+07	1.0E+05
(MICROGRAMS/M**2/YR)	30-50	9.9E+04	7.0E+04	7.0E+02
INCREMENTAL SOIL	1- 2	3.1E+02	2.2E+02	2.2E+00
CONCENTRATIONS	30-50	2.2E+00	1.6E+00	1.6E-02
(MICROGRAMS/G/YR)				
RUNOFF	1- 2	1.4E+04	1.0E+04	1.0E+02
CONCENTRATIONS	30-50	9.9E+01	7.0E+01	7.0E-01
(MICROGRAMS/L)				

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.55-C.58 IN APPENDIX APPENDIX C USING A CONVERSION FACTOR OF 4000 MWH/COMPLEX (TABLE 4.8)

TABLE 5.17 DISPERSION OF TOTAL EMISSIONS FROM MODEL PROPYLENE MANUFACTURING PLANT

	DISTANCE KM	PARTICULATES	PROPYLENE
GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)	1	1.1E+02	2.4E+01
	50	2.8E-01	6.2E-02
DEPOSITION RATES (MICROGRAMS/M**2/YR)	1- 2	4.0E+07	9.1E+06
	30-50	2.8E+05	6.3E+04
INCREMENTAL SOIL CONCENTRATIONS (MICROGRAMS/G/YR)	1- 2	9.0E+02	2.0E+02
	30-50	6.3E+00	1.4E+00
RUNOFF CONCENTRATIONS (MICROGRAMS/L)	1- 2	4.0E+04	9.1E+03
	30-50	2.8E+02	6.3E+01

THE VALUES IN THE TABLE WERE OBTAINED FROM SELECTED ENTRIES OF TABLES C.59-C.62 IN APPENDIX APPENDIX C USING A CONVERSION FACTOR OF 200,000 MWH/COMPLEX (TABLE 4.12)

TABLE 5.18. DISCHARGE RATES FOR MODEL LEAD MINE-MILL COMPLEX, ASSUMING A DISCHARGE RATE OF 2200 (L/DAY)/MWH IN THE YEAR 2000 [a,b]

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	2.0E+01	4.4E-02	3.3E+03
COPPER	1.5E-01	3.3E-04	2.5E+01
ZINC	5.0E-01	1.1E-03	8.2E+01
LEAD	3.0E-01	6.6E-04	5.0E+01
MERCURY	1.0E-03	2.2E-06	1.6E-01
CADMIUM	5.0E-02	1.1E-04	8.2E+00
CYANIDE	1.0E-01	2.2E-04	1.6E+01

[a] DERIVED FROM PROMULGATED MILL EFFLUENT LIMITATIONS (43 FR 29771, 1978; EPA-440/1-75/061) AND DATA PRESENTED EARILER IN THIS REPORT.
[b] EFFLUENT FLOATATION REAGENTS HAVE BEEN POORLY QUANTIFIED AND ARE NOT INCLUDED.

TABLE 5.19. DISCHARGE RATES FOR MODEL PRIMARY LEAD SMELTER-REFINERY COMPLEX, ASSUMING A DISCHARGE RATE OF 822 L/MT IN THE YEAR 2000 [a]

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	2.5E+01	3.0E-04	2.2E+01
CADMIUM	5.0E-01	5.8E-06	4.4E-01
LEAD	5.0E-01	5.8E-06	4.4E-01
ZINC	5.0E+00	5.8E-05	4.4E+00
ARSENIC	1.4E-02 [b]	1.8E-07	1.4E-02
NICKEL	1.7E-01 [b]	2.1E-06	1.6E-01
SULFATE	5.1E+02 [b]	6.0E-03	4.5E+02

[a] DERIVED FROM PROMULGATED PRIMARY LEAD SUBCATEGORY EFFLUENT CONCENTRATIONS (40 CFR 421.70, 1977, AND EPA-440/1-75/032-0) AND DATA PRESENTED EARLIER IN THIS REPORT.
[b] DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.20. DISCHARGE RATES FROM MODEL BATTERY MANUFACTURING PLANT USING THE WET PROCESS WITH SODIUM HYDROXIDE OR CALCIUM HYDROXIDE TREATMENT IN THE YEAR 2000

	SODIUM HYDROXIDE			CALCIUM HYDROXIDE		
PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY	MG/L	(KG/DAY)/MWH	KG/DAY
SULFATE	4.4E+04	5.5E+00	4.1E+05	-------	-------	-------
LEAD	2.3E+00	2.9E-04	2.2E+01	2.6E+00	2.9E-04	2.2E+01
ANTIMONY	4.8E-02	6.0E-06	4.5E-01	5.4E-02	6.0E-06	4.5E-01
ARSENIC	8.0E-04	1.0E-07	7.5E-03	9.0E-04	1.0E-07	7.5E-03
WATER	-------	1.3E+02	9.8E+06	-------	1.1E+02	8.2E+06

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.21. DISCHARGE RATES FROM MODEL BATTERY MANUFACTURING PLANT USING THE DRY PROCESS WITH SODIUM HYDROXIDE OR CALCIUM HYDROXIDE TREATMENT IN THE YEAR 2000

	SODIUM HYDROXIDE			CALCIUM HYDROXIDE		
PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY	MG/L	(KG/DAY)/MWH	KG/DAY
SULFATE	4.0E+04	1.8E+01	1.4E+06	-------	-------	-------
LEAD	6.0E-01	2.9E-04	2.2E+01	7.0E-01	2.9E-04	2.2E+01
ANTIMONY	1.4E-02	6.0E-06	4.5E-01	1.5E-02	6.0E-06	4.5E-01
ARSENIC	2.0E-04	1.0E-07	7.5E-03	2.0E-04	1.0E-07	7.5E-03
WATER	-------	4.4E+02	3.3E+07	-------	4.0E+02	3.0E+07

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.22. DISCHARGE RATES FOR MODEL BATTERY BREAKING OPERATIONS WITH LIME TREATMENT AND A DISCHARGE RATE OF 8 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	3.0E+01	2.4E-04	1.8E+01
OIL, GREASE	1.2E+01	9.6E-05	7.2E+00
LEAD	1.8E-01	1.4E-06	1.0E-01
ANTIMONY	9.6E-01	7.6E-06	5.7E-01
ARSENIC	1.0E-02	9.6E-08	7.2E-03
COPPER	3.6E-02	2.9E-07	2.2E-02
ZINC	1.2E-01	9.6E-07	7.2E-02
CADIUM	1.2E-02	9.6E-08	7.2E-02
NICKEL	6.0E+02	4.8E-07	3.6E-02
SULFATE	6.8E+03	5.5E-02	4.1E+03

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.23. DISCHARGE RATES FOR MODEL ZINC MINE-MILL COMPLEX, ASSUMING A DISCHARGE RATE OF 400 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	2.0E+01	8.0E-03	1.6E+03
COPPER	1.5E-01	6.0E-05	1.2E+01
ZINC	5.0E-01	2.0E-04	4.0E+01
LEAD	3.0E-01	1.2E-04	2.4E+01
MERCURY	1.0E-03	4.0E-07	8.0E-02
CADIUM	5.0E-02	2.0E-05	4.0E+00
CYANIDE	1.0E-01	4.0E-05	8.0E+00

DERIVED FROM PROMULGATED MILL EFFLUENT CONCENTRATIONS (43 FR 29771, 1978; EPA-440/1-75/061) AND DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.24. DISCHARGE RATES FROM MODEL NICKEL MINING, MILLING,SMELTING AND REFINERY OPERATIONS, ASSUMING A DISCHARGE RATE OF 1300 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	1.5E+01	2.0E-02	4.0E+03
NICKEL	5.0E-01	8.0E-04	1.6E+02
COPPER	2.0E-01	3.0E-04	6.0E+01
IRON	5.0E-01	8.0E-04	1.6E+02

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.25. DISCHARGE RATES FROM MODEL PRIMARY ZINC INDUSTRY, ASSUMING A DISCHARGE RATE OF 19 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	2.5E+01	4.9E-04	1.0E+02
ARSENIC	1.0E-01	1.9E-06	3.8E-01
CADIUM	5.0E-01	9.3E-06	1.8E+00
SELENIUM	5.0E+00	9.3E-05	1.8E+01
ZINC	5.0E+00	9.3E-05	1.8E+01

DERIVED FROM PROMULGATED PRIMARY ZINC SUBCATEGORY EFFLUENT LIMITATIONS (40 CFR 421.83, 1977; EPA-440/1-75/032, 1975) AND DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.26. DISCHARGE RATES FROM MODEL CAUSTIC POTASH PRODUCING COMPLEX, ASSUMING A DISCHARGE RATE OF .08 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	6.4E+01	3.0E-04	6.0E+01
LEAD	5.0E-01	4.0E-08	8.0E-03

DERIVED FROM PROMULGATED EFFLUENT LIMITATIONS (40 CFR 415.65, 1977) AND DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.27. DISCHARGE RATES FROM MODEL NICKEL/ZINC BATTERY MANUFACTURING PLANT, ASSUMING A DISCHARGE RATE OF 20 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
NICKEL	1.0E+03	2.0E-02	4.0E+03

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.28. DISCHARGE RATES FROM MODEL LITHIUM MINE-MILL COMPLEX, ASSUMING A DISCHARGE RATE OF 6 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	1.7E+02	1.0E-03	2.0E+02
FLUORINE	1.7E+01	1.0E-04	2.0E+01

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.29. DISCHARGE RATES FROM MODEL NICKEL MINING, MILLING, SMELTING AND REFINERY OPERATIONS, ASSUMING A DISCHARGE RATE OF 830 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	1.5E+01	1.0E-02	2.0E+03
NICKEL	5.0E-01	5.0E-04	1.0E+02
COPPER	2.0E-01	2.0E-04	4.0E+01
IRON	5.0E-01	5.0E-04	1.0E+02

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.30. DISCHARGE RATES FROM MODEL CAUSTIC POTASH PRODUCING COMPLEX, ASSUMING A DISCHARGE RATE OF 6.4 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
TSS	6.4E+01	2.4E-04	4.8E+01
LEAD	5.0E-01	3.2E-08	6.2E-03

DERIVED FROM PROMULGATED EFFLUENT LIMITATIONS (40 CFR 415.65, 1977) AND DATA PRESENTED EARLIER IN THIS REPORT.

TABLE 5.31. DISCHARGE RATES FROM MODEL IRON-STEEL PRODUCING COMPLEX, ASSUMING A DISCHARGE RATE OF 1 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	(KG/DAY)/MWH	KG/DAY
TSS	2.0E-05	4.0E+00
FLUORINE	2.0E-05	4.0E+00
ZINC	4.0E-06	8.1E-01

DERIVED FROM PROMULGATED EFFLUENT LIMITATIONS (40 CFR 420.105, 1977) AND DATA PRESENTED EARLIER IN THIS REPORT. A DISCHARGE RATE OF 1 (L/DAY)/MWH IS ASSUMED.

TABLE 5.32. DISCHARGE RATES FROM MODEL NICKEL/IRON BATTERY MANUFACTURING PLANT ASSUMING A DISCHARGE RATE OF 20 (L/DAY)/MWH IN THE YEAR 2000

PARAMETER	MG/L	(KG/DAY)/MWH	KG/DAY
NICKEL	1.0E+02	2.0E-03	4.0E+02

DERIVED FROM DISCHARGE DATA PRESENTED EARLIER IN THIS REPORT.

References

1. D. B. Turner, *Workbook of Atmospheric Dispersion Estimates*, U.S. Environmental Protection Agency, Office of Air Programs, Research Triangle Park, North Carolina, 1970.

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3. U.S. Environmental Protection Agency, *Ore Mining and Dressing Point Source Category - Effluent Limitations Guidelines for Existing Sources.* Fed. Reg. 43(133): 29771-29781, 1978.

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6. U.S. Environmental Protection Agency, *Development Document for Interim Final Effluent Limitations Guidelines and Proposed New Source Performance Standards for the Zinc Segment of the Nonferrous Metals Manufacturing-Point Source Category*, EPA-440/1-75/032, 1975.

6. ECOLOGICAL IMPACTS OF EMISSIONS FROM BATTERY CYCLES

ABSTRACT

The first part of the section provides a generic discussion of the biogeochemistry and impacts of the major expected constituents of emissions from the battery cycle. This lays the foundation for assessing the types of impacts that may result from further development of battery technology. Our quantitative assessments are based upon the numbers generated in Section 5 and represent our predictions of anticipated impacts. The estimates are tentative and will change as the pathways to be followed by the developing technologies become better defined.

6.1 BIOGEOCHEMISTRY OF MAJOR EMISSION CONSTITUENTS

Discussion of the biogeochemistry* of major emission constituents (SO_2, Sb, As, Cd, Fe, Ni, Pb and Zn) from the principal battery cycles (lead/acid, Ni/Zn and Ni/Fe) is presented here as background information for an assessment of ecological impacts of these emissions. A generalized biogeochemical cycle is presented in Figure 6.1. Elements, even those nonessential to life, are cycled among physical and biological components of ecosystems. This ensures the continued availability of essential elements for new biomass production. This process involves (1) transfers from anthropogenic sources (man's activities) to atmosphere (gases surrounding the Earth), pedosphere (soils), and hydrosphere;** (2) uptake by biosphere (living things); (3) transfer to saprosphere (decaying matter); (4) mineralization; and (5) subsequent recycling. The discussion will identify permanent and temporary sinks for emission constituents and impacts to terrestrial and aquatic systems. Data on bioaccumulation (biological accumulation of emission constituents above levels in growth medium) and biomagnification (increasing tissue concentration of emissions constituents during sequential transfer along food chains) are provided where available. Emission constituent pathways to humans are identified.

6.1.1 Antimony

Biogeochemical Cycle

Antimony is found naturally in the atmosphere at concentrations of less than 4 ng/m^3 but natural distributions have been altered by anthropogenic sources. Little information is available on antimony releases within the United States. Most domestic antimony produced is primary antimony ore, coproduct or byproduct of other mining operations, or byproduct at primary lead smelters, usually antimonial lead.[1] The largest use of antimony in the U.S. is in the

**Geochemistry is a study of the circulation of the elements in nature (amounts, distribution, stability, form); biogeochemistry studies the effects of life processes on the distribution and fixation of these chemical elements.*

***Surface waters of the Earth, including glaciers, ice, snow, and water vapor; distinct from the lithosphere (rocks).*

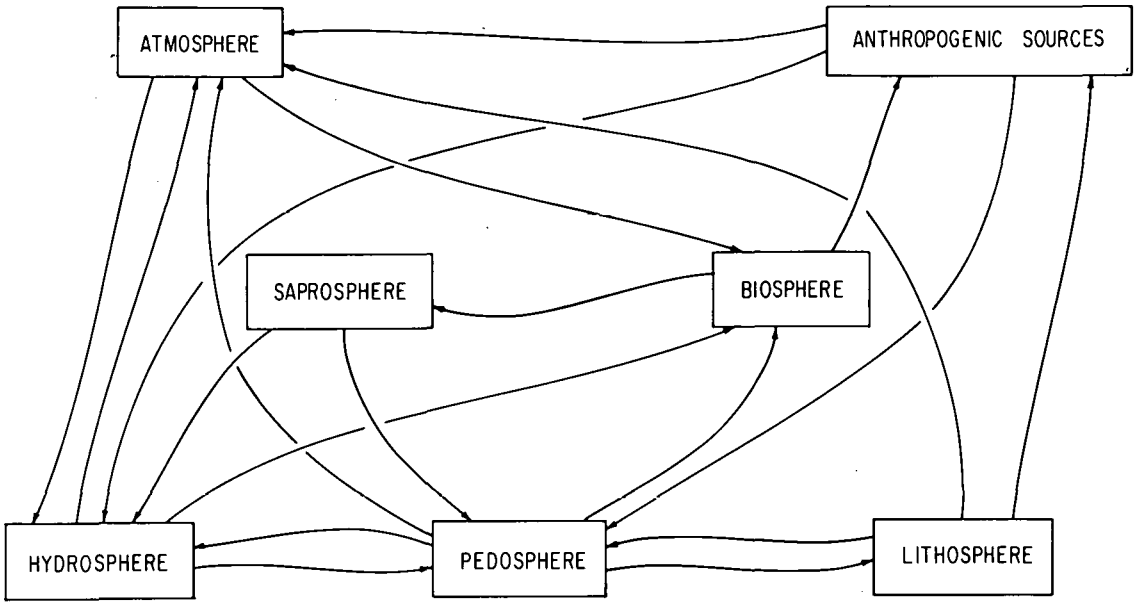

Fig. 6.1. Schematic Representation of Components and Pathways within the Biogeochemical Cycle.

manufacture of storage batteries.[2] Antimony is released into the environment from a variety of sources including industrial and municipal wastes. Antimony is a contaminant in nonferrous ores and is released into the environment during smelting of these ores as antimony oxides. Crecelius et al. reported elevated antimony levels in the environment around a large copper smelter, originating in atmospheric emissions, wastewater discharges and slag.[3]

Antimony enters the soil from the atmosphere by dry deposition or precipitation. Abundance of antimony in the earth's crust ranges from 0.2 to 0.5 μg/g.[2] Little information is available concerning the biogeochemistry of antimony in soil. Furthermore, the movement of antimony in soil systems is poorly understood. Under natural conditions antimony has a valence of +3 or +5, but has physicochemical properties similar to those of arsenic (see Sec. 6.1.2).

Antimony enters surface freshwater systems through many pathways including atmospheric deposition, surface runoff, groundwater flow, and waste disposal. Because little information is available on the aquatic chemistry of antimony, antimony cycling in aquatic systems is poorly understood. Antimony probably behaves similarly to arsenic in aquatic systems.

McKee and Wolf reported that dissolved antimony enters natural waters by precipitation and is removed by sedimentation and/or adsorption.[4] Antimony tends to be precipitated as oxides or sulfides. Crecelius et al. reported that less than 10% of the antimony was bound in sediments of Puget Sound by readily oxidized organic matter.[3] Fifty percent of the antimony was bound to extractable iron and aluminum compounds in an unpolluted area. Lisk reported an antimony level in river water of 0.13 μg/L.[5]

Though antimony occurs naturally in atmospheric, terrestrial and aquatic systems, inputs from anthropogenic sources are primarily responsible for its role as a potentially hazardous

substance. Antimony compounds are most mobile when released into the atmosphere or water, and are of greatest concern to the general population surrounding sites of emission. Antimony dispersion to and cycling within natural systems are poorly understood, as is its potential toxicity to exposed biota. Representative natural concentrations of antimony in various components of the biogeochemical cycle are shown in Table 6.1; concentration ratios in biota are shown in Table 6.2.

Table 6.1. Representative Natural Concentrations of Antimony in Various Components of the Biogeochemical Cycle

Component	*Concentration*
Atmosphere, μg/m^3	< 0.004
Hydrosphere	
Seawater, μg/g	0.00033
Freshwater, μg/g	0.00027 - 0.0049
Pedosphere, μg/g	2 - 10
Biosphere	
Marine animals, μg/g	0.2
Terrestrial plants, μg/g	0.06
Terrestrial animals, μg/g	0.14

Sources: H.J.M. Bowen, "Trace Elements in Biochemistry," Academic Press, New York, 1966.

H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee, ORNL-5361, 1978.

Table 6.2. Concentration Ratios for Antimony in Aquatic or in Terrestrial Ecosystems[a]

Biota	*Habitat*	*Concentration Ratio*
Invertebrates	Marine[b]	5.0
Fish	Marine[b]	40.0
Invertebrates	Freshwater[b]	10.0
Fish	Freshwater[b]	1.0
Plants	Terrestrial[c]	0.02

[a] $[Sb]_{biota}/[Sb]_{ambient}$.

[b] *H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee, ORNL-5361, 1978.*

[c] *D.J. Lisk, "Trace Metals in Soils, Plants, and Animals," Adv. Agron. 24:267-325, 1972.*

Impacts on Terrestrial Ecosystems

Atmospheric antimony is available for foliar uptake by plants and inhalation by animals in exposed populations. However, toxicity, bioconcentration and biomagnification of antimony have not been quantified sufficiently for discussion.

Impacts on Aquatic Ecosystems

Little information is available concerning antimony toxicity to aquatic organisms. Bioconcentration and biomagnification of antimony by aquatic organisms are not quantified sufficiently for discussion here.

Pathways to Humans

Atmospheric antimony is available for inhalation in exposed human populations. Little information is available concerning the bioconcentration and biomagnification of antimony in food chains leading to human populations.

6.1.2 Arsenic

Biogeochemical Cycle

Arsenic is released through natural processes and as a result of anthropogenic activities. Arsenic is a contaminant in many nonferrous metal ores, such as lead, zinc and copper ores. During smelting of these ores arsenic compounds, such as arsenic trioxide, volatilize and must be removed from smelter exhaust gases. In the United States, arsenic is recovered by a smelter in Tacoma, Washington. Approximately 97% of the arsenic produced enters end-product manufacture as white arsenic and the remaining 3% as metallurgic additives in special lead and copper alloys.[6]

Serious incidents of air pollution by arsenic from U.S. smelters have been recorded. Some degree of arsenical air pollution is possible at every smelter treating arsenical ores, especially when dust-collecting equipment is inadequate or not working properly. Sullivan reported atmospheric arsenic concentrations of less than 0.02 $\mu g/m^3$ in areas not contaminated by industrial activities.[7] Urban arsenic concentrations varied from less than 0.01 to 0.16 $\mu g/m^3$.

Atmospheric arsenic is removed by dry deposition or precipitation. Deposition rates are dependent on distance from source, pollution type, initial release rate and height, and weather conditions. Woolson reported that atmospheric contamination from smelter stack emissions resulted in elevated arsenic levels in surrounding ecosystems.[0] Colburn et al. reported average arsenic concentrations of 5.7 $\mu g/g$ in uncontaminated soils, with considerable variation among geographic regions.[9]

Arsenic soil chemistry is analogous to that of phosphorus, and arsenate usually exists in soil solution as a trivalent anion resembling phosphate. Probabilities of arsenical adsorption, ion exchange and salt formation range from high to very high. These reactions yield biologically inactive forms of arsenic. Salt formation occurs in presence of iron, aluminum, calcium and

magnesium, all of which result in insoluble arsenic salts. Adsorption, occurring in presence of fine soil (colloidal and organic matter), results in soil-arsenic complexes. Ion exchange occurs in soils with high exchange capacity and results in soil-arsenic complexes.[10]

Under normal aerobic soil conditions arsenic is oxidized to pentavelent forms. Pentavalent arsenical compounds have high probabilities of salt formation, adsorption and ion exchange, and low probabilities of arsenical demethylation, reduction, and methylation. Demethylated arsenicals, produced by aerobic microorganisms, are likely to undergo salt formation, adsorption, and ion exchange, yielding biologically inactive forms of arsenic.[10]

Reduction and methylation of arsenic produces biologically active forms of arsenic. Arsines produced by reduction under aerobic or anaerobic conditions or by specific microorganisms react rapidly to form pentavalent arsenicals. Methylarsines produced by methylation in the presence of specific bacterial microorganisms (aerobic and anaerobic) react rapidly to form pentavalent arsenicals.[10]

Arsenic enters surface freshwater systems through many pathways, including atmospheric deposition, surface runoff, groundwater flow and waste disposal. Durum et al. reported that 79% of 727 samples examined in the United States contained arsenic concentrations of less than 10 µg/L, 21% greater than 10 µg/L, and 2% greater than 50 µg/L.[11] Arsenic moves within streams by dissolved load or within suspended sediment.[12] Dissolved arsenates and arsenites quickly form insoluble salts with cations and settle out in river sediments. Thus, below outfall areas arsenic stream loads are removed with resultant increases in sediment arsenic concentrations.[10]

Arsenic in aquatic systems has a complex chemistry, including oxidation-reduction, ligand exchange, precipitation and adsorption reactions. Arsenic is stable in four oxidation states (+5, +3, 0, -3) under Eh conditions present in aquatic systems. At high Eh values in oxygenated waters, arsenic acid species (H_3AsO_4, $H_2AsO_4^-$, $HAsO_4^{2-}$, and AsO_4^{3-}) are stable. Arsenous acid species (H_3AsO_3, $H_2AsO_3^-$, and $HAsO_3^{2-}$) are characteristic of mildly reducing conditions.[13] Braman and Forebach reported arsenite, arsenate and methylated arsenic forms in aquatic environments.[14] Arsenite species can be present in surface waters if the Eh is less than about 0.1 volt or if oxidation to arsenate is incomplete. Arsenic is removed to the sediment through co-precipitation with metal ions or precipitation by adsorption onto clays. Arsenic in sediments can be solubilized through microbial transmethylation or reduction, and can re-enter the water column by diffusion through the sediments, or mixing by currents or burrowing organisms.[13] Organisms affect arsenic distribution by accumulating, transporting, and transforming it.

Arsenic cycling in natural aquatic systems has not been quantified to any extent. The ratio of dissolved organic to dissolved inorganic arsenic pools is unknown, and arsenic speciation is unknown within each pool. Between active and storage pools, relative arsenic concentrations and rates of arsenic movement are unknown. The extent of bioconcentration and biomagnification is unknown. Arsenic methylation is poorly understood as are the quantitative effects of temperature, redox potential, pH, and other physicochemical factors on the regeneration of arsenic from sediments. Bacterial influence on arsenic cycling is poorly understood.

Arsenic compounds are most mobile when released into the atmosphere or water and are of greatest concern to the general population surrounding sites of emission. Cases of severe environmental degradation have practically disappeared with the introduction of modern arsenic

recovery technologies.[10] Representative natural concentrations of arsenic in various components of the biogeochemical cycle are shown in Table 6.3; concentration ratios for biota are presented in Table 6.4.

Impacts on Terrestrial Ecosystems

Natural arsenic absorption by plants from the atmosphere is negligible. Smelter fumes and dust can be deposited on plant foliage, but there is no evidence of foliar uptake. Plants are affected, however, by arsenic from smelter fallout. The effects are strongly modified by a host of variables, including plant species, geographical region, soil type and climatic conditions.[10]

According to Allaway, plants grown on uncontaminated soils rarely contain greater than 1 µg/g arsenic.[15] In arsenic toxicity, plant roots are usually severely affected, and plant growth is limited before much arsenic is translocated to aerial foliage.[15-17] Around smelters, high arsenic soil concentrations can cause the area to be devoid of higher plants.[10] Arsenic residues in plants probably will not be toxic to animals because plant injury occurs before sufficient bioconcentration occurs.[10]

Arsenic compounds do not accumulate in mammals.[18,19] Differences in the toxicity of various compounds have been correlated with animal excretion rates; compounds most slowly excreted tend to be the most toxic.[18,19] The biological half-lives for excretion of arsenic compounds are reported to range from 30 to 60 hours.[19,20] The greatest danger to animals from arsenic emissions may be through the ingestion of arsenic-contaminated dust or soil on forage. Arsenic and its compounds do not appear to be carcinogenic, although there is some dispute about this in the literature.[15,19,20] Frost,[19] Buchanan,[21] and Vallee et al.[18] have reviewed the toxicity and biological effects of arsenic in animals.

Impacts on Aquatic Ecosystems

Natural waters are usually low in arsenic and plants reflect this in their arsenic contents. Natural waters high in arsenic can be toxic to plants as well as animals.

Bioaccumulation of arsenic occurs, but there is little biomagnification.[13] Isensee et al. reported that arsenic biomagnification did not occur through their study food chain, and they suggested that adsorption was important in the distribution pattern.[22] Lower food-chain organisms contain the highest concentration of residues.[23] However, Braunstein et al. indicated that arsenic may be biologically concentrated through aquatic food chains; benthic algae, mollusks, crustacea and fish can concentrate arsenic to levels 200, 650, 400, and 700 times as great as those in the environment, respectively.[24] Freshwater organisms generally contain lower concentrations of arsenic than their marine counterparts; concentration ratios reported for saltwater fish range from 10 to 100 times higher than those reported for freshwater fish.[23]

In general, arsenic is toxic to aquatic organisms within the range of 1 to 45 µg/L arsenite.[4] The arsenite ion is generally considered more toxic than arsenate.[13,24] Toxic levels of arsenic to aquatic organism are highly dependent on physicochemical conditions.

Table 6.3. Representative Natural Concentrations of Arsenic in Various Components of the Biogeochemical Cycle

Component	*Concentration*
Lithosphere, μg/g	15 - 2
Atmosphere, μg/m^3	<0.01
Hydrosphere	
Seawater, μg/g	0.003
Freshwater, μg/g	0.0004
Pedosphere, μg/g	6.0
Biosphere	
Marine plants, μg/g	30
Marine animals, μg/g	0.005 - 150
Freshwater animals, μg/g	<1.0 - 3.0
Terrestrial plants, μg/g	20
Terrestrial animals, μg/g	≤ 0.2

Sources: "Arsenic," National Academy of Sciences, Washington, DC, 1977.

H.J.M. Bowen, "Trace Elements in Biochemistry," Academic Press, New York, 1966.

Table 6.4. Concentration Ratios for Arsenic in Aquatic or Terrestrial Ecosystems[a]

Biota	*Habitat*	*Concentration Ratio*
Brown algae[b]	Marine	6000.
Invertebrates[b]	Marine	330.
Fish[b]	Marine	330.
Invertebrates[b]	Freshwater	330.
Fish[b]	Freshwater	330.
Plants[c]	Terrestrial	0.14
Plants[d]	Terrestrial	0.12
Plants[e]	Terrestrial	0.03

[a] $[As]_{biota}/[As]_{ambient}$.

[b] *H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Overview," Oak Ridge National Laboratory, Tennessee, ORNL/5361, 1977.*

[c] *A.J. Dvorak et al., "Impacts of Coal-fired Power Plants on Fish, Wildlife, and Their Habitats," U.S. Department of the Interior, Fish and Wildlife Service, FWS/OBS-78/29, 1978.*

[d] *J.K. Hodgson, "Chemistry of Trace Elements in Soils With Reference to Trace Element Concentration in Plants," pp. 45-58 in Proceedings of the Third Annual Conference on Trace Substances in Environmental Health," Vol. III, D.D. Hemphill, ed., University of Missouri Press, Columbia, 1970.*

[e] *D.J. Lisk, "Trace Metals in Soils, Plants, and Animals," Adv. Agron. 24:267-325, 1972.*

Pathways to Humans

Environmental contamination, with subsequent human exposure to arsenic compounds, has resulted from air pollution from smelters. Atmospheric arsenic is available for inhalation in exposed human populations. Normally, man's intake of arsenic from water is not significant, except in areas of abnormally high arsenic concentrations. Except for locations around smelters and other anthropogenic sources or where natural arsenic concentrations are high, there is little concern that arsenic will enter pathways to human exposure.

6.1.3 Cadmium

Biogeochemical Cycle

Cadmium is released by man into the environment either as a result of manufacture, use, or disposal of cadmium products or as a contaminant in other man-made substances. Approximately 1800 MT of cadmium was released into the environment in 1974. Land waste disposal, air emissions, and wastewater effluents constituted 82%, 16%, and 1% respectively, of this release. About 20% of the 1974 release originated from the primary nonferrous metals industry; 30% from the manufacture, use and disposal of cadmium products and 50% from cadmium contaminants released from sources such as phosphatic fertilizers, sewage sludge and combustion of fossil fuels.[25]

Atmospheric cadmium concentrations in the United States are usually a few hundredths or thousandths of a $\mu g/m^3$; higher concentrations have been reported in areas of zinc and lead mining and smelting.[25] Atmospheric cadmium is removed by dry deposition or precipitation. Soils receive 96% of the total environmental cadmium released by man. Cadmium appears to be less mobile in soils than most other heavy metals: studies indicate that 95% of the cadmium entering the soil is retained within the soil.[26]

Haghiri reported that soil cation exchange capacity, organic matter, temperature and zinc affected cadmium uptake by plants.[27] The greater the cation exchange capacity of the soil, the greater the absorbancy of exogenous cadmium by the exchange complex.[28] Street et al. reported that $CdCO_3$ and $Cd_3(PO_4)_2$ most likely limit Cd^{2+} activities in soils.[29] Under alkaline conditions (in sandy soils having low cation-exchange capacities or low organic matter contents) Cd^{2+} activity decreases approximately 100-fold for each unit increase in pH. Allaway reported an average cadmium concentration in soil of 0.06 µg/g with a range of 0.01 to 7 µg/g.[15] Cadmium is removed from soil systems through direct surface runoff, leaching, and solute interflow by underground pathways or through biochemical activities.[25]

A median dissolved cadmium concentration of <1 µg/L was reported by Hem in 726 samples from rivers and lakes.[30] In river water, cadmium concentrations are usually below the limiting equilibrium solubilities for cadmium carbonate and hydroxide complexes. Concentrations above 10 µg/L can be stable in water having low total solute concentrations and low pH. A substantial portion of the total cadmium in surface waters is usually present as the free cadmium ion; this proportion will be larger with lower pH values. Humic substances usually account for most cadmium complexation, followed in importance by carbonate complexation.[31] Shephard reported that precipitation of cadmium adsorbed on particulate matter is the primary control on dissolved cadmium solubility, with precipitation of cadmium carbonate acting as a secondary control on

cadmium solubility.[32] Sediment adsorption and desorption processes are likely to be major factors in controlling the cadmium concentrations in natural waters. Humic material appears to be a major sediment component responsible for adsorption. Sediment concentration factors varying between 5,000 and 50,000 were reported by Gardener.[33] Sediments in localized areas exposed to industrial or mining waste have been reported to contain very high levels of cadmium.[25]

Reddy and Patrick reported that redox potential and pH are two of the major factors influencing cadmium immobilization in sediments, and thus the availability of cadmium to plants.[34] Cadmium uptake increased with an increase in suspension redox potential and decrease in pH. Although cadmium does form coordination complexes with a variety of ligands in biological systems, no metabolic interconversions have been demonstrated. Cadmium is not methylated in the environment because alkyl-metals of cadmium are not stable in aqueous systems and methyl-B_{12} does not transfer methyl groups to cadmium.[35]

Anthropogenic sources are primarily responsible for cadmium as a potentially hazardous substance, although cadmium occurs naturally in atmospheric, terrestrial and aquatic systems. Cadmium compounds are most mobile when released into the atmosphere or water, and are of greatest concern to the general population surrounding sites of emission. Cadmium releases to the environment appear to be declining; however, the cadmium content in fossil fuels, nonferrous ores and fertilizers is only partially controllable, and these sources may set the lower bounds of attainable minimums in cadmium emissions. Most of the released cadmium eventually becomes immobile in soil, sediment and ocean sinks.

Accumulations of cadmium are found in most living organisms.[25] Representative natural concentrations of cadmium in various components of the biogeochemical cycle are shown in Table 6.5. Concentration ratios for cadmium in aquatic and terrestrial ecosystems are presented in Table 6.6.

Impacts on Terrestrial Ecosystems

Although plants normally contain <0.5 µg/g cadmium,[15] many species accumulate much higher concentrations (up to several hundred µg/g) when they are grown in soils with elevated cadmium concentrations.[27,36-41] Higher cadmium concentrations in biota have been reported near smelters.[25]

Plant species vary in their ability to accumulate and translocate cadmium to aerial portions. The gross effects of cadmium toxicity in plants include wilting, chlorosis, necrosis, and a reduction of growth.[42,43] Huang et al. reported that cadmium generally inhibited soybean metabolism and adversely affected nitrogen fixation and photosynthesis.[44] Cadmium has not been demonstrated to be essential for plant growth.[45]

Little information has been documented on cadmium content in terrestrial invertebrates, which play an important role in the vertical transport of trace elements in the environment.[25] Gish and Christensen found that earthworm cadmium concentrations were approximately ten times soil cadmium levels along the Baltimore-Washington, D.C. Parkway.[46] Munshower reported that grasshopper cadmium concentrations were slightly higher than their food cadmium concentrations.[47]

Table 6.5. Representative Natural Concentrations of Cadmium in Various Components of the Biogeochemical Cycle

Component	*Concentration*
Lithosphere, μg/g	0.5
Atmosphere, μg/m^3	0.0004 - 0.028
Hydrosphere	
Seawater, μg/g	0.00011
Freshwater, μg/g	<0.08
Pedosphere, μg/g	600
Biosphere	
Marine plants, μg/g	0.4
Marine animals, μg/g	1.5 - 13.0
Terrestrial plants, μg/g	0.6
Terrestrial animals, μg/g	≤0.5

Sources: H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee, ORNL-5361, 1978.

D. Wagoner, "Compilation of Ambient Trace Substances," Draft Report Prepared for the U.S. Environmental Protection Agency by Research Triangle Institute Under Contract No. 68-02-1325, Research Triangle Park, North Carolina, 1976.

H.J.M. Bowen, "Trace Elements in Biochemistry," Academic Press, New York, 1966.

Table 6.6. Concentration Ratios for Cadmium in Aquatic and Terrestrial Ecosystems[a]

Biota	*Habitat*	*Concentration Ratio*
Invertebrates[b]	Freshwater	2,000.
Fish[b]	Freshwater	200.
Invertebrates[b]	Marine	250,000.
Fish[b]	Marine	3,000.
Scallops[b]	Marine	2,300,000.
Plants[c]	Terrestrial	11.
Plants[d]	Terrestrial	5.5

[a] $[Cd]_{biota}/[Cd]_{ambient}$.

[b] *H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee, ORNL-5361, 1978.*

[b] *A.J. Dvorak et al., "Impacts of Coal-fired Power Plants on Fish, Wildlife, and Their Habitats," U.S. Department of the Interior, Fish and Wildlife Service, FWS/OSB-78/29, 1978.*

[d] *D.J. Lisk, "Trace Metals in Soils, Plants, and Animals," Adv. Agron. 24:267-325, 1972.*

Cadmium is a cumulative poison in animals.[16,42] Unlike plants, species-dependency of the cadmium level in animals has not been demonstrated. No clear indication of any differences in the cadmium content of herbivorous and carnivorous animals has been observed.[25]

Impacts on Aquatic Ecosystems

Aquatic biota have been shown to concentrate cadmium in their tissues several orders of magnitude greater than that found in their immediate environment.[25] Aquatic macrophytes have concentrated cadmium in the hundreds of μg/g range in contaminated areas near smelters and nickel-cadmium battery plants.

Comparison of data on cadmium levels in aquatic invertebrates with those for fish indicate a much greater concentration of cadmium in invertebrates. Huckabee and Blaylock reported that cadmium accumulated in sediments more rapidly than in living organisms and that fish accumulated cadmium more slowly than snails in a microcosm study.[26] Cadmium values appeared to be lower in marine and estuarine fish than in freshwater fish in an EPA investigation.[25]

Kinkade and Erdman reported that water hardness influences cadmium uptake and accumulation rates, which in turn influence cadmium toxicity.[48]

Pathways to Humans

Atmospheric cadmium is available for inhalation in exposed human populations. Higher cadmium concentrations have been reported in areas of zinc and lead mining and smelting.[25] Crop plants accumulate cadmium through aerial uptake and through uptake from soil.[16,49] Yamagata and Shigematsu demonstrated that cadmium from polluted soils can be incorporated into the food chain in quantities sufficient to be toxic to human populations.[50] Additionally, cadmium can be incorporated into food chains from waters in quantities sufficient to be toxic to human populations.[51]

6.1.4 Iron

Biogeochemical Cycle

Iron is essential to all biota, as an enzyme activator and a mediator in oxidation-reduction reactions; iron is considered nontoxic or minimally toxic to biota. Iron is abundant in the lithosphere (Table 6.7), which is its primary source; it occurs in nature mainly as weakly soluble oxides and sulfides.

The sulfides are the major ores processed to extract iron, and on weathering, release sulfuric acid and iron hydroxides. Iron is found in soil principally as relatively insoluble oxides and hydroxides, and is relatively immobile.[51,52] Iron solubility and mobility in soils are influenced by pH; solubility increases with acidity. Under the acid conditions associated with many smelter-polluted soils, leaching and biotic uptake of iron would be higher than under more neutral or alkaline conditions; however, the generally low mobility of iron suggests that soils fumigated by iron emissions will tend to accumulate iron and become the major sink for iron emissions in terrestrial ecosystems.

Table 6.7. Representative Natural Concentrations of Iron in Components of the Biogeochemical Cycle

Component	*Concentration*
Lithosphere, µg/g	3,800 - 56,000
Pedosphere, µg/g	8,000 - 38,000
Atmosphere, µg/g[3]	< 3
Hydrosphere	
Freshwater, µg/g	0.67
Seawater, µg/g	0.003 - 0.01
Saprosphere, µg/g	900 - 3,600
Biosphere	
Marine plants, µg/g	700 - 4,800
Marine animals, µg/g	400
Terrestrial plants, µg/g	160

Source: H.J.M. Bowen, "Trace Elements in Biochemistry," Academic Press, New York, 1966.

The concentration ratio for iron in terrestrial biota is generally less than 1 (Table 6.8). In areas near Wawa, Ontario, that have been contaminated by emissions from an iron sintering plant, the concentration ratios in foliage ranged from 0.01 to 0.05.[53] Data from the Wawa area indicate that iron accumulated faster in vegetation (up to 200-fold in the direction of the pollution source than in soil (2- to 20-fold). The low toxicity of iron makes it unlikely that pollutant iron would accumulate to toxic levels within terrestrial ecosystems near iron processing plants.

The hydrosphere contains little iron (Table 6.7), because of the low solubility of iron compounds. The sediment is the major sink for iron.[53-55] Biomagnification beyond the primary producer trophic level does not appear to occur. However, aquatic biota show higher concentration ratios for iron than terrestrial biota, and the likelihood of accumulating toxic amounts of iron is higher in aquatic than in terrestrial systems.

In the natural atmosphere, suspended iron is rare (Table 6.7). The major source of atmospheric iron is the processing of iron ores. In the vicinity of a steel mill in Sault Ste. Marie, Ontario, Canada, iron concentrations in air as high as 14 µg/m^3 have been measured.[56]

Impacts on Terrestrial Ecosystems

Few studies have been conducted that show the impacts of iron emissions on terrestrial ecosystems; most have examined the smelting regions of Sudbury and Wawa, Ontario, Canada.[52,57,58] Sulfur dioxide is the primary toxic pollutant in these areas, although nickel has been implicated in inhibiting revegetation in the Sudbury region. It has been difficult to isolate the impacts of iron emissions. In only a few cases did the iron in vegetation near Wawa exceed levels considered excessive, but not necessarily toxic, by the Ontario Ministry of the Environment (ca. 800 µg/g).[52] Therefore, it does not appear that iron emissions *per se* have impacted

Table 6.8. Concentration Ratios for Iron[a]

Taxon	*Habitat*	*Concentration Ratio*
Phytoplankton[b]	Marine	2,400
Algae/Higher Plants[c]	Aquatic	2,400 - 200,000
Invertebrates[c]	Aquatic	125
Zooplankton[b]	Marine	5,400
Fish[c]	Aquatic	10,000
Fish[b]	Marine	130 - 660
Vascular Plants[d]	Terrestrial	0.008
Vascular Plants[e]	Terrestrial	0.01 - 0.06
Plants[c]	Terrestrial	1
Mammals[c]	Terrestrial	0.2 - 0.8

[a] $[Fe]_{biota}/[Fe]_{ambient}$.

[b] *M. Waldichuk, "Some Biological Concerns in Heavy Metal Pollution," pp. 1-57 in "Pollution and Physiology of Marine Organisms," F.J. Vernberg and W.B. Vernberg, eds., Academic Press, New York, 1974.*

[c] *D.E. Reichle, P.B. Dunaway and D.J. Nelson, "Turnover and Concentration of Radionuclides in Food Chains," Nucl. Safety 11:43-55, 1970.*

[d] *J.K. Hodgson, "Chemistry of Trace Elements in Soils with Reference to Trace Element Concentrations in Plants," pp. 45-58 in "Proceedings of the Third Annual Conference on Trace Substances in Environmental Health, Vol. III, D.D. Hemphill, ed., University of Missouri Press, Columbia, 1970.*

[e] *W.D. McIlveen, R. Potvin and W. Keller, "Environmental Studies in the Wawa Area, 1969-1977," Ontario Ministry of the Environment, Ottawa, Canada, 1979.*

these ecosystems. The possibility of synergistic interactions with other pollutants was not evaluated in these studies.

Iron has been shown to inhibit nitrification at soil concentrations of about 5 µg/g above background levels.[59]

Impacts on Aquatic Ecosystems

One study has examined the impacts of iron on riverine communities in Lancashire, England.[60] The pollutant source was coal mine drainage dominated by iron compounds. The acidity of the drainage was buffered by bicarbonate salts from the river and lime treatment. Iron concentrations in the affected areas were 20 to 100 times concentrations upstream; other metals increased 1- to 20-fold. Species diversity and macrofaunal abundance were depressed in the impacted stretches; benthic fauna were markedly impacted by high iron concentrations in sediment below the drainage inflow. The responses of this system to the pollutants were similar to those of other systems.

Pathways to Humans

Few data are available on iron-emissions inputs to the human food chain. One would expect the likelihood of effects to be greater for aquatic food chains because aquatic biota tend to concentrate iron. Areas in which water concentrations of iron are greatly increased could be a threat to human health.

6.1.5 Lead

Biogeochemical Cycle

Anthropogenic activities have altered the equilibrium in the natural biogeochemical cycling of lead. Anthropogenic sources of atmospheric lead emissions during 1974 and 1975 accounted for 4.4×10^{11} g of the total 4.6×10^{11} g. Automobiles, base metal production, and fossil fuel combustion contributed 61%, 30%, and 5% of this total.[61] In 1975, U.S. gasoline combustion contributed 88.1% of the 1.6×10^5 MT released by inventoried atmospheric release categories, while base metal production contributed about 3% of the total.[25]

Numerous studies show significant soil contamination by lead emissions from mobile and stationary sources and by disposal of waste products.[25] The atmosphere is the principal medium for transport of lead from anthropogenic sources.

Lead pollution from lead mining and smelting activities is localized, but may be severe. Windblown ore concentrates from storage dumps, open trucks, railroad cars and tailings ponds are principal pollution sources.

Physical and chemical characteristics of lead releases are variable and depend on ore composition, processing procedures and pollution abatement equipment. Jennett et al. found lead, zinc, cadmium and copper comprised 16.3%, 1.63%, 0.82%, and 0.04% by weight, respectively, of particulates in smelter stack emissions.[62] Corrin and Natusch reported that 60% of particulate lead was associated with particulates smaller than 2.4 μm.[63] Lead species in baghouse effluents from lead smelting and refining activities included PbS, $PbSO_4$, elemental lead and a mineral tentatively identified as $PbO \cdot PbSO_4$.[63] Aerosols from mining activities contain PbS, $PbCO_3$, $PbSO_4$, $Pb_5(PO_4)_3Cl$, $PbS \cdot Bi_2S_3$, PbO_x and Pb-silicates.[64] Metal smelting operations release elemental lead, PbO_x, $PbSO_4$, $PbCO_3$, $PbO \cdot PbSO_4$, $(PbO)_2PbCO_3$, lead in other metal oxides and Pb-silicates.

Nriagu has calculated that if present emissions were uniformly distributed to a height of 20 km, the atmospheric concentration would be about 3.7 $\mu g/m^3$.[64] Average lead concentrations in urban areas range from 0.5 to 10 $\mu g/m^3$; in rural areas from 0.1 to 1.0 $\mu g/m^3$; in areas far removed from urban centers are <0.1 $\mu g/m^3$. Atmospheric inorganic particulate lead is removed by dry deposition and precipitation.[25] Deposition rates depend on distance from source, pollution type, inital release rate and height, and weather conditions.

Lead deposited on the soil surface is not leached substantially into lower soil horizons because of its low geochemical mobility. Thus, soils are the major sink for most atmospheric lead released by man. Getz et al. found that most lead entering a study watershed remained

within the watershed.[65] Lead sulfide and lead chloride are thought to be the major lead constituents in soils in the vicinity of mining industries, and that lead oxide is the major constituent in the vicinity of smelters. Nriagu reported an average lead concentration of 17 µg/g in uncontaminated soils.[61]

Lead chemistry in soils is determined by (a) absorption at soil/mineral interfaces; (b) formation of relatively stable organolead complex ions and insoluble organolead chelates, particulates and residues; (c) precipitation of sparingly soluble lead compounds; and (d) coprecipitation of lead with common soil minerals. The relative importance of these mechanisms to lead retention in soils is unknown.[66] Zimdahl and Hassett reported increases in solubility of most lead salts as pH decreases because the hydrogen ion is a good competitor for available sorption sites.[67]

The formation of insoluble lead compounds (particularly phosphates) has two important ecological implications: lead fixation in soils apparently regulates the quantity of lead that cycles annually through aquatic systems, and lead associated with insoluble compounds may not be available to plants.[66] Lead can be more available for plant uptake in acidic soils because it is sorbed to a lesser extent and is less likely to be precipitated; however, this depends on which ionic species are present. Environmental factors, plant age, and plant species are important variables in lead uptake by plant roots. Under certain soil conditions (low pH, low cation exchange capacity, low organic matter, and low phosphate levels) large amounts of lead can be taken up by roots.

Lead enters the aquatic environment through many pathways including atmospheric deposition, surface runoff, groundwater flow, and waste disposal. High concentrations of lead in surface freshwaters usually occur near sources of contamination. The average lead level in surface freshwaters throughout the world has been calculated as 0.5 µg/L.[68]

Lead is partitioned quickly within aquatic systems into three principal storage pools (organic biomass, suspended organic and inorganic particulates, and sedimentary pools) and two active pools (dissolved organoleads and dissolved inorganic lead).[66]

A significant fraction of the lead in lotic systems is transported in an undissolved state. Industrial lead concentrations in rivers, however, can be so excessive that exchange between particulate and dissolved forms provides large quantities of dissolved lead even though the major lead fraction is adsorbed onto solids.[68]

Partitioning of lead between aqueous and solid phases in aquatic environments is determined by many factors including composition and textural characteristics of sediments, temperature, pH, redox potential, ionic competition, lead input, and biological activities.[61] Chow reported that the following interactions were important in determining lead concentrations: (1) formation of lead-inorganic anion complexes, (2) formation of lead-organic ligand complexes, (3) dissolution and precipitation of organic and inorganic lead compounds, (4) lead sorption onto hydrous and clay colloids; (5) lead sorption onto organic colloids and particulates, (6) decomposition of organic biomass to liberate associated lead, and (7) coprecipitation of lead with other inorganic mineral phases.[68] Hydroxides, carbonates, sulfides, and more rarely sulfates, act as lead-solubility controls.[25] Lead tends to form compounds of low solubility with major anions in

Table 6.9. Representative Natural Concentrations of Lead in Various Components of the Biogeochemical Cycle

Component	*Concentration*
Lithosphere, μg/g	16
Atmosphere, μg/m^3	<0.2
Hydrosphere	
Seawater, μg/g	0.00003
Freshwater, μg/g	0.005
Pedosphere, μg/g	10
Biosphere	
Marine plants, μg/g	8.4
Marine animals, μg/g	0.5
Freshwater plants, μg/g	<1.0 - 2.3
Terrestrial plants, μg/g	2.7
Terrestrial animals, μg/g	2.0

Sources: H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee ORNL-5361, 1978.

H.J.M. Bowen, "Trace Elements in Biochemistry, Academic Press, New York, 1966.

K. J. Yost, "Some Aspects of Cadmium Flow in the UNS.," Environ. Health Perspect. 28; 5-16, 1979.

Table 6.10. Concentration Ratios for Lead in Aquatic or Terrestrial Ecosystems[a]

Biota	*Habitat*	*Concentration Ratio*
Invertebrates[b]	Freshwater	100.
Fish[b]	Freshwater	300.
Invertebrates[b]	Marine	1000.
Fish[b]	Marine	200.
Plants[c]	Terrestrial	0.45
Plants[d]	Terrestiral	2.3

[a] $[Pb]_{biota}/[Pb]_{ambient}$.

[b] *H.M. Braunstein et al. "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee ORNL-5361, 1978.*

[c] *A.J. Dvorak et al., "Impacts of Coal-fired Power Plants on Fish, Wildlife, and Their Habitats," U.S. Department of the Interior, Fish and Wildlife Service, FWS/OBS-78-29, 1978.*

[d] *D.J. Lisk, "Trace Metals in Soils, Plants, and Animals," Adv. Agron. 24:267-325, 1972.*

natural water. The undissolved lead fraction consists of lead carbonates, oxides, hydroxides, sorbed ions and components of living and nonliving organic matter. Lead carbonate complexes dominate the inorganic chemistry of dissolved lead in surface-water systems.[68]

The physicochemical forms of lead determine reaction mechanisms and rates, availability, and thus the impact of lead in aquatic ecosystems.[68] Lead can enter biomass pools as soluble lead ions, as organolead molecules, or with particulate materials. Lead is highly toxic in aquatic systems with the degree of toxicity to aquatic organisms being dependent on physicochemical conditions.

Lead enters sedimentary pools as biomass and particulate pools settle to the bottom. Lead flow into aerobic sediments tends to be unidirectional, and sediments represent the primary sink for lead in aquatic environments.[66] In anaerobic sediments lead can be immobilized as PbS; but also returned to overlying waters as alkylated lead and other organolead compounds. Particulate lead undergoes only minor change when deposited in sediments. Degradation of organic materials can release lead which is likely to react with hydrous oxides. Increased use of lead products is reflected in lake bottom sediment lead profiles.

Lead cycling in natural aquatic systems has not been extensively quantified. The ratio of dissolved organic to dissolved inorganic lead and lead speciation are unknown within each pool. Rates of lead movement between active and storage pools and relative pool concentrations are unknown. Lead methylation is poorly understood, as are the quantitative effect of temperature, redox potential, pH, and other physicochemical factors on the regeneration of lead from sediments. Bacterial influence on lead cycling is poorly understood also.[66]

Anthropogenic sources are primarily responsible for lead as a potentially hazardous substance. Lead compounds from human activities are most mobile when released into the atmosphere or into water, and are of greatest concern to the general population surrounding the site of emission. Representative natural concentrations of lead in various components of the biogeochemical cycle are shown in Table 6.9. Concentration ratios for biota in aquatic and terrestrial ecosystems are presented in Table 6.10.

Impacts on Terrestrial Ecosystems

In lead-polluted soils, lead-sensitive organisms generally will be eliminated while lead-tolerant organisms will survive and predominate.[69] Soil microfauna play an important role in accumulation and transport of lead, but little is known on how lead affects these microfauna. Rühling and Tyler[70] and Ebregt and Boldewijn[71] reported negative correlations between decomposition rates and lead concentrations. Doelman reported that microfauna inhibition by lead is correlated with soil type.[69]

Most lead deposited on soil is adsorbed onto organic matter and remains close to the soil surface.[72] The amount of lead accumulated by plant roots and translocated to aerial portions depends on plant species, soil and environmental factors, but is generally low.[72] Most lead accumulated by plant roots remains in the roots. Substantial quantities of lead must accumulate in soil before lead hinders the growth of higher plants.[67]

Little evidence for foliar uptake of aerosol-deposited lead exists, but deposition on leaf surfaces cannot be ignored as a potential hazard in food chains.[73] Nriagu has shown that the lead content of various plants is related to sources of air pollution and to atmospheric lead concentrations.[64] Around smelters and other industrial installations, highly significant relationships between lead in vegetation and both distance from point source and the direction of prevailing winds have been found.

Animals grazing near smelters can be poisoned, likewise animals fed vegetation harvested in the vicinity of smelters, mines and industrial plants from which lead fumes and/or dusts are being emitted.[25] The deaths of animals grazing in pastures near primary and secondary smelters have been reported as directly attributed to high body burdens of lead.[72] Williamson and Evans reported no evidence of biomagnification of lead in the food chain of warm-blooded vertebrates.[74]

Impacts on Aquatic Ecosystems

Lead is highly toxic to all species of aquatic biota. Its toxicity depends on a number of factors including types and physiological stages of biota as well as a host of environmental physicochemical parameters.

Sediments are widely recognized as primary sinks for heavy metals in aquatic ecosystems, however, few studies have related sediment metal content to lead concentrations in benthic organisms.[75] Little is known about biomagnification of lead through successive trophic levels. Rickard and Nriagu have indicated that bioconcentration and biomagnification can occur as lead is transferred up food chains.[66] Studies by Getz et al.,[65] Drifmeyer and Odum,[76] and others, however, indicate that biomagnification of lead does not occur and that lead accumulation is a function of niche and habitat; biota associated with sediments have higher lead concentrations.[75] Although biomagnification of lead does not appear to occur, lead is concentrated by biota from water and sediments.

Pathways to Humans

Atmospheric lead can be inhaled by exposed human populations. The presence of secondary smelters in urban areas is considered to be a health risk to local communities because atmospheric lead can be inhaled while lead fallout and dust can be ingested.[77] Deposition of lead on plant foliage occurs in areas of lead pollution and is a potential hazard in food chains leading to humans. Biomagnification through pathways leading to humans is apparently not a severe problem in either terrestrial or aquatic ecosystems.

6.1.6 Nickel

Biogeochemical Cycle

The role of nickel in metabolism remains obscure; although some evidence shows it may be essential for some species, many researchers do not consider it essential for life.[78-81] Nickel is cycled through the major components of the biogeochemical system; unfortunately, the manner of cycling has not been well documented. Nickel is considered very toxic to plants and moderately

toxic to mammals;[78,79] in general, plants exhibit symptoms of toxicity at tissue concentrations in excess of about 50 μg/g; lethal, oral doses for laboratory animals range from 100 to 1600 μg/g.[81]

Some data are available on the distribution of nickel in the environment (Table 6.11). The major source of nickel is the lithosphere, most nickel ore is currently extracted as nickel-iron sulfide minerals. Generally, nickel occurs in the divalent oxidation state. Weathering yields $NiSO_4$ and other soluble nickel salts which enter the pedosphere or are transferred to the hydrosphere in runoff.

Table 6.11. Representative Natural Concentrations of Nickel in Various Components of the Biogeochemical Cycle

Component	*Concentration*
Lithosphere, μg/g	1 - 110
Atmosphere, μg/m^3	0 - 0.1
Hydrosphere	
Seawater, μg/g	0.001 - 0.023
Freshwater, μg/g	0.002 - 0.56
Pedosphere, μg/g	2 - 3000
Biosphere	
Marine plants, μg/g	0.3 - 18
Marine animals, μg/g	~17
Freshwater plants, μg/g	5 - 60
Freshwater animals, μg/g	3 - 16
Terrestrial plants, μg/g	1 - 630
Terrestrial animals, μg/g	<1

Sources: H.J.M. Bowen, "Trace Elements in Biochemistry," Academic Press, New York, 1966.

H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee, ORNL-5361, 1978.

T.C. Hutchinson et al., "Movement and Compartmentation of Nickel and Copper in an Aquatic Ecosystem," pp. 565-585 in "Environmental Biogeochemistry," Vol. 2, Metals Transfer and Ecological Mass Balances, J.O. Nriagu, ed., Ann Arbor Science Publishers, Ann Arbor, Michigan, 1976.

Nickel is highly mobile under neutral to acid conditions.[82] Rates of leaching from soils increase rapidly with decreases in pH below 4.[83] Nickel solubility and its availability to plants may also be increased by complexing with some organic molecules; thus, nickel toxicity may be particularly severe in soils high in soluble organic matter. The soil serves as the primary sink for nickel in terrestrial systems. Under anthropogenic input of nickel, concentrations increase more rapidly in the soil than in the biotic components. In the Sudbury

smelting region, nickel concentrations in polluted soil were 50 times that in unpolluted sites; whereas,[84] the nickel in polluted foliage was less than 25 times that in foliage from unpolluted sites. This suggests that soils serve as the major accumulators of pollutant nickel in the terrestrial system.

Nickel is taken up by plants as a divalent cation,[78] but has been reported to have a lower concentration ratio in terrestrial plants (0.045) than many divalent, transition metals.[85] Foliar concentration ratios of 0.02 to 1.0 have been measured, which are generally less than for other divalent metals.[86] It is not known how large a role is played by direct uptake of nickel from atmospheric suspension.

Concentration ratios in aquatic systems are several orders of magnitude higher than in terrestrial systems (Table 6.12). Aquatic primary producers concentrate nickel 10 to 100 times higher than consumers. There does not appear to be any biomagnification of nickel entering the higher trophic groups. Nickel concentrations are generally low in the hydrosphere (Table 6.11), and the sediment is the major sink in aquatic system.[87,88] Hutchinson et al. noted that sediment nickel levels were ten times higher near a pollution source than in unpolluted waters.[87] Concentrations in water and most biota increased only 2- to 6-fold as the pollutant source was approached.

Table 6.12. Concentration Ratios for Nickel in Aquatic Systems[a]

Taxon	*Habitat*	*Concentration Factors*
Periphyton[b]	Riverine	∿20,000
Vascular plants[b]	Riverine	∿11,500
Seaweed[c]	Marine	1100 - 2800
Invertebrate[c]	Marine	250
Invertebrate[c]	Freshwater	100
Invertebrate[b]	Riverine	250 - 950
Fish[b]	Riverine	200 300
Fish[c]	Freshwater	100
Fish[c]	Marine	100

[a] $[Ni]_{biota}/[Ni]_{ambient}$.

[b] *T.C. Hutchinson et al., "Movement and Compartmentation of Nickel and Copper in an Aquatic Ecosystem," pp. 565-585 in "Environmental Biogeochemistry," Vo. 2, Metals Transfer and Ecological Mass Balances, J. O. Nriagu, ed., Ann Arbor Science Publishers, Ann Arbor, Michigan, 1976.*

[c] *H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An information Assessment," Oak Ridge National Laboratory, Tennessee, ORNL-5361, 1978.*

Under natural conditions the atmosphere contains little nickel, anthropogenic sources are fossil fuel combustion and metallurgical processes. Potvin and Balsillie reported aerial concentrations near a smelter that exceeded 1 $\mu g/m^3$.[89]

Impacts on Terrestrial Ecosystems

The primary impact of nickel on soils is the depression of microbial activity. Giashuddin and Cornfield found that additions of nickel sulfide inhibit the dynamics of nitrogen and carbon cycling.[90] Nickel depressed nitrification to a greater extent than it depressed mineralization of either nitrogen or carbon. Decreased activity was noted at the lowest concentration of nickel, 10 µg/g. About 6 µg/g of nickel was reported to depress nitrification by about two-thirds.[59] Natural revegetation in the Sudbury region has probably been prevented by high levels of nickel and other metals in the soil; extensive soil erosion has occurred in heavily impacted areas.[56,57]

In the Sudbury region, researchers have been unable to distinquish among the effects of the several metal pollutants, but nickel levels exhibit the greatest increase over background.[84] In one study Whitby and Hutchinson irrigated laboratory plants with aqueous extracts from these soils.[86] Although germination was not inhibited, root elongation was reduced about 33% in seedlings growing in extracts from soils containing 2300 µg/g total nickel (extracts contained ∿ 1 to 6 µg/g Ni). Irrigation with solutions of nickel salts alone reduced elongation nearly 70% at concentrations around 2 µg/g. Nickel appeared to be the major metal toxicant in this case; however, the soils contained other metals at levels toxic to many plants. Impacts to fauna were not measured, but it is likely that nickel deleteriously impacts the animal populations, directly and indirectly, in a similar manner as the plants.

Impacts on Aquatic Ecosystems

Hutchinson et al., studying the distributions of heavy metals in a riverine environment in the Sudbury region, noted no impacts on the biotic community, but they did not rigorously examine the community.[88] The responses of aquatic communities would be expected to be similar (reduced diversity, inhibited decomposition, change in species composition, etc.) to impacts by other pollutants. The levels of nickel at which such responses would be discernible are not known.

Pathways to Humans

Few studies have examined the role of nickel in the human food chain. The National Research Council has summarized the available information.[81] It notes that mammalian species may have mechanisms for preventing nickel accumulation until dieting concentrations are quite high (< 1500 µg/g). However, this conclusion is based on limited data from studies of cattle, and may not be generalizable to mammals as a whole. Because of the tendency of aquatic organisms to accumulate nickel, fish and shellfish exposed to high levels are more likely to pose a threat to humans than meat.

6.1.7 Sulfur

Biogeochemical Cycle

Sulfur is a chemical element required by all biota for the synthesis of a number of compounds; its action on biota ranges from the beneficial to the highly toxic, depending upon its

form and concentration.[78] In recent years concern has arisen over the increasing anthropogenic releases of sulfur to the environment. Sulfur emissions are associated with the mining and processing of many metal ores, and are expected to increase as a result of increased battery production.

The fundamental source for sulfur is the lithosphere (Table 6.13), the earth's rocky crust, where sulfur exists primarily in its reduced state. Sulfur is released from rocks by weathering and, to a lesser extent, volcanism.[91-93] In the process of weathering, sulfide metal ores are oxidized, and in most soils sulfur occurs as sulfates in the S^{6+} valence state. Runoff of sulfates into the hydrosphere can be a major pathway within the sulfur cycle. On the global scale, some 30% to 70% of sulfur input to the pedosphere has been estimated to be lost through runoff.[93] In local watersheds, runoff losses of sulfur can approach and exceed rates of sulfur inputs to the system.[94-96] Sedimentation from the hydrosphere returns sulfur to the lithosphere.

Table 6.13. Representative Natural Concentrations of Sulfur in Components of the Biogeochemical Cycle

Component	*Concentration*
Lithosphere, μg/g	260 - 2400
Atmosphere, μg/m^3	3 - 50
Hydrosphere	
Seawater, μg/g	885
Freshwater, μg/g	∿4
Pedosphere, μg/g	700
Biosphere	
Marine plants, μg/g	12,000
Marine animals, μg/g	5000 - 19,000
Land plants, μg/g	3400
Land animals, μg/g	5000

Source: H.J.M. Bowen, "Trace Elements in Biochemistry," Academic Press, N.Y., 1966.

Sulfur, taken up by plants in the form of sulfate, is reduced during the synthesis of organo-sulfur compounds; in addition, plants may absorb sulfur dioxide (SO_2) directly from the atmosphere. Estimates suggest that gaseous uptake by plants could account for as much as one-third of the total biological uptake of sulfur in the environment.[95,96]

Most sulfur in the soils is bound in the organic component, the saprosphere, where the sulfur content is maintained at a fairly constant ratio of sulfur to total organic matter and to other essential elements, such as nitrogen.[52] Maintenance of these ratios results from biogeochemical equilibria among the pathways connecting the biosphere-saprosphere network with the pedosphere or hydrosphere. Mineralization of the reduced organo-sulfur compounds is carried out by a number of soil bacteria. Under aerobic conditions, oxidized sulfur is returned to the mineral soil as sulfate and is again available for uptake by plants.

Most of the sulfur emitted to the atmosphere under natural conditions is in the form of hydrogen sulfide (H_2S). The major source of H_2S is the activity of sulfur-reducing bacteria in anoxic environments containing abundant organic materials.[93] In the atmosphere, H_2S is rapidly oxidized to SO_2. On a global scale, the geothermal and volcanic production of SO_2 contribute only a small proportion to the total input of sulfur to the atmosphere, but these sources may be important on a local scale. Sulfate (SO_4^{2-}) aerosols from ocean spray are additional sources of natural atmospheric sulfur.

Currently, human activities are major sources of atmospheric sulfur. Combustion of fossil fuels such as coal and processing of metal-sulfide ores release SO_2 to the atmosphere. Two nickel smelters in Sudbury, Ontario, are estimated to contribute up to 10% of the total anthropogenic SO_2 produced in North America.[57] Atmospheric concentrations of SO_2 vary widely, from near zero to values in excess of 9 mg/m^3.[93] The higher values are associated with industrial urban areas where anthropogenic production of SO_2 is high; Kellogg et al. have estimated that anthropogenic sources of sulfur contribute about half as much as natural sources.[97] By the end of the century anthropogenic releases are expected to equal natural releases. Most global anthropogenic sulfur release occurs in the Northern Hemisphere; only 6% to 7% of the production is estimated as attributable to the Southern Hemisphere.[97] Accelerated metal ore processing in North America to meet predicted requirements for battery production would result in an increase in anthropogenic sulfur in the Northern Hemisphere.

Excess sulfur may accumulate in soils or be leached to groundwater or carried away in runoff into surface waters. The relatively unpolluted Walker Branch Watershed in Tennessee accumulated sulfur at a total rate of 6.6 (kg/ha)/yr ($\sim$ 33% of annual input); 65% of this accumulation occurred in the mineral soils.[96] These mineral soils accumulated sulfur, while sulfur content declined in the organic litter on the forest floor. In contrast, Likens et al. estimated that only about 6% of annual sulfur input was retained in a northern hardwood forest system. Most of this accumulation occurred in the organic components of the ecosystem; the balance of sulfur inputs to the forested ecosystems were removed through runoff.[95] The greater rainfall in the northern forest may explain why little sulfur was retained in the soils of that site. Schindler et al. indicated that a large proportion of sulfur inputs to watersheds in Ontario (80% to 160%) are lost as runoff.[94] This suggests that the main sink for excess sulfur is in the hydrosphere and associated sediments, rather than in the organic or soil components of a system.

Few data are available from which to analyze the concentration ratios for sulfur in the food chain (see Table 6.14). Biota tend to concentrate sulfur some 2 to 20 times above ambient levels; however, concentration ratios for sulfur are not as high as they are for many heavy metals.[24] Some sulfur accumulation would be expected in biota growing in an area of high sulfur emissions, e.g., in the vicinity of a smelter.

McIlveen and Balsillie, examining the sulfur content of soils and plant tissues in the region surrounding the smelters at Sudbury, Ontario, found some evidence of increased sulfur in soils subjected to the highest amounts of SO_2 fumigation; however, accumulation was only 1 to 4 times the content of control soils.[98] Vegetation near SO_2 sources also exhibited higher (threefold to fourfold) foliar concentrations of sulfur than found in the control areas. The higher ratios of exposed to control concentrations of sulfur in foliage compared to concentrations in

Table 6.14. Concentration Ratios for Sulfur in Ecosystems[a]

Taxon	*Habitat*	*Concentration Factor*
Plankton	Marine	2 - 7
Invertebrates	Marine	5 - 20
Fish	Marine	∿ 8
Vascular Plants	Northern deciduous forest	3 - 12

[a] $[S]_{biota}/[S]_{ambient}$.

Source: H.J.M. Bowen, "Trace Elements in Biochemistry," Academic Press, New York, 1966.

soils are probably due to direct absorption and adsorption of SO_4 from the atmosphere by vegetation. Much of this will be leached from the foliage in throughfall.[95,96]

Impacts on Terrestrial Ecosystems

Pollutant sulfur enters the soil in four manners: (1) as sulfuric acid (H_2SO_4) or sulfate salts in precipitation; (2) as particulates containing sulfates (by dry deposition); (3) absorbed into the vegetative canopy as SO_2, leached from the foliage, and carried down in the throughfall; and (4) adsorbed directly onto the soils as SO_2.[52] The major effect of sulfur compounds is acidification of the soil. Adsorbed SO_2 is readily oxidized to SO_4^{2-} in most soils, releasing hydrogen ion. The sulfate ion is readily leached, carrying with it basic cations such as Ca^{2+}, Mg^{2+}, and Na^{+}, thus leading to a buildup of hydrogen ion concentration and a consequent depression in pH.[99] The extent of acidification depends upon the buffering capacity of the impacted soils. Hutchinson and Whitby reported pH levels of less than 3 at distances up to 7.4 km from one smelter in the Sudbury region.[84]

Acidification of soils affects a number of soil properties.[52,100,101] Cation exchange capacity in most soils decreases with acidification as hydrogen ions displace basic cations that are less tightly bound to the exchange sites. Acidification increases the mobility of many cations in the soil, which can result in increased availability of heavy metals. Plant intolerance for acidic conditions has often been attributed to Al^{3+} toxicity because the aluminum ion is available at low pH.[52] Plant uptake of iron, zinc and copper (emissions from the primary refining of materials for battery production) is also increased at low pH. Calcium, magnesium and phosphorus deficiencies also can occur in acid soils because these elements become less available to plants as pH decreases below about 6.

Acidity modifies the rates at which soil organic material is decomposed. Phosphorus and sulfur mineralization are depressed under acid conditions. Nitrification and denitrification occur less rapidly under acid conditions than more neutral conditions.[52,102] Nitrogen fixation by bacteria living symbiotically with legumes is inhibited by low pH. In addition, nodule formation by nitrogen-fixing bacteria in the roots of legumes is retarded by high concentrations of hydrogen ions in the soil. In general, increasing the hydrogen ion content above slightly

acidic levels decreases the diversity and abundance of soil microbes, thereby depressing rates of biologically mediated decomposition.[96]

Several studies have examined the effects of large-scale fumigation of natural ecosystems by SO_2. Many of these cases involve emissions from the processing of metal sulfide ores. Because particulate emissions containing heavy metals have also added to the stresses on these ecosystems, it is not clear how much damage can be attributed directly to SO_2 in these cases.

Two of the most striking examples of SO_2 emission impacts have occurred in Ontario, Canada. In the Sudbury region, nickel and copper smelters generated about 5×10^6 kg SO_2/day prior to 1972.[103] An area of about 1900 km^2 extending northeastward from Sudbury exhibited extensive tree mortality. Species composition of the forest was modified as those species sensitive to SO_2 (e.g., eastern white pine) were eliminated.[103,104] Dead wood in the area deteriorates slowly, and there is some indication that decomposer populations in the soils have decreased in areas near the sulfur source. In areas of high mortality, heavy soil erosion has exposed the bedrock.

In the vicinity of Wawa, Ontario, an iron-sintering plant generated an estimated 3.5×10^5 to 6.0×10^6 kg SO_2/day between 1961 and the end of 1977. Daily emissions at Wawa were markedly less than at Sudbury, but the effects have been as dramatic. In 1970, the area of nearly 100% tree mortality covered about 110 km^2, with a total of over 780 km^2 of forest being damaged to a lesser degree.[53,103] As one approaches the iron-sintering plant, the forest becomes more depauperate, and the composition of the plant community is altered.[58] In the heavily impacted areas, erosion is extensive.

Similar damage has been noted near other ore-processing and sulfuric-acid-manufacturing plants that release sulfur dioxide.[57,105,106] In general, the following changes in ecosystems have been attributed to exposure to numerous fumigations with SO_2: decreased population abundance, decreased species diversity, depressed growth and reproduction, increased sulfur concentrations in soils and foliage, and depressed rates of organic decomposition. Effects on fauna have not been examined in these studies, but similar impacts are expected as the habitat becomes degraded. These conditions arise from both the direct effects of SO_2 toxicity and indirect effects due to soil acidification.

It is difficult to attribute the levels of damage to levels of SO_2 emissions and concentrations. Most studies do not directly relate these parameters, but rather use indirect estimates of SO_2 exposures, such as distance from the source. In the Wawa area, where over 6×10^5 kg SO_2/day were emitted in 1976 and 1977, near-source average monthly SO_2 concentrations were 3 to 125 $\mu g/m^3$, with hourly peaks exceeding 2600 $\mu g/m^3$ and 24-hour maxima exceeding 260 $\mu g/m^3$.[53] The provincial criterion of 660 $\mu g/m^3$ hourly averages were exceeded 188 times in the two years; the 24-hour criterion of 260 $\mu g/m^3$ was exceeded 18 times. At the edges of the moderately damaged area (35 km northeast of the source) monthly averages ranged from 10 to 130 $\mu g/m^3$, with hourly maxima exceeding 1000 $\mu g/m^3$ and 24-hour maxima exceeding 260 $\mu g/m^3$. Hourly averages (660 $\mu g/m^3$) at the distant point were exceeded 88 times and 24-hour averages (260 $\mu g/m^3$) eight times. The development of the damage to biotic communities observed in the Wawa area probably occurred in response to levels of SO_2 fumigation similar to those measured in 1976 and 1977.

Impacts on Aquatic Ecosystems

The primary impact of sulfur emissions on aquatic systems is acidification of the waters.[107,108] In the Sudbury region of Ontario, lakes within 4600 km^2 of smelter operations were found to have pH values of 5.5 or less.[109] It is believed that the main cause of the depressed pH is acidic precipitation and direct deposition of SO_2 emissions from the smelting operations.

In acidified waters, Al^{3+} concentrations can reach toxic levels.[108] In combination with deposition of excess sulfur, there is increased leaching of basic cations from soils into the waters. Increasing input of hydrogen ion decreases bicarbonate levels while inputs of such cations as calcium and magnesium increase and sulfates replace bicarbonates as major anions in the system. In aerobic waters, cations may be precipitated out as sulfate salts. Sulfur is also removed from the aqueous phase by sedimentation with organic particulates.

A major effect of lake acidification is the reduction in the number of aquatic species.[108-111] Diatoms, blue-green algae and green algae are the phytoplankters affected most by acidification; zooplankton and other aquatic fauna respond in a similar manner. Species diversity and abundance decline, and biotic composition is altered as sensitive species disappear and tolerant species increase. Plankton biomass and production may actually increase as acid-tolerant species replace less tolerant species and nutrients are mobilized from the sediment. However, with increasing acidity there is depressed recycling of nutrients, productivity may decline, and oligotrophication may begin, resulting in an overall decrease in biotic diversity and abundance.[112,113]

Pathways to Humans

Sulfur enters the human body through inhalation from the atmosphere or ingestion of food and drink. Inhalation is the most likely pathway to lead to toxic effects; it is unlikely that sulfur accumulation in human food as a result of ore processing would cause direct, toxic effects to humans. Although concentrations of sulfur have been shown to be higher in the foliage of plants exposed to SO_2 fumigation, it does not appear to be bioaccumulated to toxic levels. Indirect impacts are more likely from crop damage due to excessive exposure. Many crop species are considered to be sensitive to SO_2 exposures as low as 100 $\mu g/m^3$ for 8 hours of exposure.[114] This concentration is not uncommon in the vicinity of the Sudbury smelters.[89]

6.1.8 Zinc

Biogeochemical Cycle

Zinc is an essential trace element for all biota.[78] The zinc cycle is not as well documented as cycles of other elements; however, the pathways are similar to those of the sulfur cycle described above, although rates of transfer differ. The fundamental source of zinc is the lithosphere, where it occurs primarily as sphalerite (ZnS) and smithsonite ($ZnCO_3$).[24] Weathering transfers zinc into the pedosphere and hydrosphere.

In the pedosphere zinc (Zn^{2+}) concentrations can range from 10 to 2000 $\mu g/g$ (Table 6.15). In terrestrial ecosystems, the mineral soil is the major zinc reservoir. In a southeastern

Table 6.15. Representative Natural Concentrations of Zinc in Components of the Biogeochemical Cycle

Component	*Concentration*
Lithosphere, μg/g	16 - 95
Atmosphere, μg/m^3	<0.07
Hydrosphere	
Seawater, μg/g	0.002 - 0.01
Freshwater, μg/g	0.015 - 130
Pedosphere, μg/g	105 - 2000
Saprosphere, μg/g	60 - 130
Biosphere	
Marine plants, μg/g	150
Marine animals, μg/g	6 - 500
Freshwater plants, μg/g	30
Freshwater animals, μg/g	20 - 500
Land plants, μg/g	10 - 200
Land animals, μg/g	160

Sources: H.H.M. Bowen, "Trace Elements in Biochemistry, Academic Press," New York, 1966.

H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment," Oak Ridge National Laboratory, Tennessee, ORNL-5361, 1978.

R.I. Van Hook, W.F. Harris and G.S. Henderson, "Cadmium, Lead, and Zinc Distributions and Cycling in a Mixed Deciduous Forest," Ambio. 6: 281-286, 1977.

G.P. Parker, W.W. McFee and J.M. Kelly, "Metal Distribution In Forested Ecosystems in Urban and Rural Northwestern Indiana," J. Environ. Qual. 7: 337-342, 1978.

R.V. Anderson, W.S. Vinikour and J.E. Brower, "The Distribution of Cd, Cu, Pb, and Zn in the Biota of Two Freshwater Sites with Different Trace Metal Inputs," Holarctic Ecol. 1: 377-384, 1978.

mixed deciduous forest system, 99% of the zinc standing crop was bound in the mineral soil.[115] A recalculation of the data of Westman has indicated that soils are also the major sinks in three western coastal coniferous forests, holding over 95% of the zinc in each system.[116] Parker et al. has shown similar distributions in forested ecosystems in northern Indiana.[117]

Divalent zinc is taken up by plants, primarily through the root system.[115] Some zinc may be absorbed from the atmosphere, but Jordan has suggested that zinc-containing particles remain biologically inert.[118] The most rapid pathways in the zinc cycle involve the biosphere and saprosphere components. Van Hook et al. found turnover rates in the organic pools to be much more rapid than in soil.[115] Zinc was effectively retained by the system; only about 25% of the annual input was lost via stream runoff.

In aquatic systems, the primary sink for zinc is in the sediments, where it recycles rapidly through the biological components.[119-121] The hydrosphere does not play a large role in storing zinc although nonrooted biota take up zinc from the aqueous medium.

Under natural conditions, the atmosphere does not play a major role in zinc cycle, and contains less than 0.07 $\mu g/m^3$ of zinc (Table 6.15). Wind erosion and saltwater spray are the major natural contributors of zinc to the atmosphere. In areas of anthropogenic emissions, zinc concentrations can rise dramatically; some urban areas show measured concentrations as high as 17 $\mu g/m^3$.[24] Emissions from fossil fuel and smelting facilities are usually in the form of ZnO.

In terrestrial systems, bioaccumulation does not appear to occur to a major extent (Table 6.16). Vegetation growing in soils contaminated by a zinc smelter had foliar:soil Zn ratios of about 0.2 to 0.6, indicating biodilution.[118,122] Other biota in zinc-contaminated habitats showed similar results.[117,123,124] Zinc in aquatic systems is much more prone to be bioaccumulated in nonrooted, aquatic biota; concentration ratios range to over 500,000 (Table 6.17). Studies of several aquatic systems indicate that Zn accumulates in the higher trophic levels of the food chain.[125,126] However, in an estuarine system, Baptist and Lewis found that no biomagnification of Zn occurred above the primary producer level, although all biota had concentrations considerably higher than concentrations in the water.[127]

Impacts on Terrestrial Ecosystems

Buchauer noted that the soil pH near a zinc smelter was about 2 units higher than distant sites, despite the concurrent addition of SO_2 and H_2SO_4 which tend to acidify soils.[122] This elevation was attributed to deposition of ZnO in these soils.

Zinc (Zn^{2+}) tends to be relatively immobile in soils, although it can be transported readily under some conditions.[24,82] Divalent zinc is held in the soils by organic colloids, clay minerals, and hydrous oxides of manganese and iron; its mobility and availability to plants is highly dependent on pH. Compounds of zinc with common anions, such as chloride and sulfate, are readily soluble in acid, and hence move readily under neutral to acid conditons. Solubility is correlated inversely with pH. Thus, in areas of combined zinc and heavy SO_2 fumigation, zinc toxicity in soils could be severe.

The primary impact of zinc emissions in soils is on the biotic decomposition processes. In the vicinity of a zinc smelter, researchers have found a depression in soil microfloral abundance and depressed rates of decomposition in contaminated soils.[128,129] Laboratory experiments have implicated zinc accumulation as the major cause of altered soil microbial funcion.[130,131] An urban site in northern Indiana was found to have lower decomposition rates for litter than a rural site.[132] High levels of zinc and other metals were associated with the decreased decomposition.

In the investigations made of zinc-emission impacts on terrestrial ecosystems, it has been difficult to separate the effects of zinc from those of gaseous pollutants or other heavy metals. A coordinated series of studies near a zinc smelter in Pennsylvania has implicated Zn^{2+} as the major phytotoxic pollutant.[133] In a chestnut-oak forest, oak-chestnut prior to onset of chestnut blight, decomposition and soil microbial and macrofaunal activity were reduced in areas where soil zinc concentrations exceeded about 25 g/kg. There was markedly less lichen cover in the contaminated area than in control areas. The composition and abundance of the tree and shrub species differed significantly between pollutant and control areas. On the polluted areas, regeneration by sexual reproduction (seeds) was inhibited, and seedlings were abundant only for species that reproduced vegetatively. Inhibition of seed germination and seedling development

Table 6.16. Concentration Ratios for Zinc in Terrestrial Systems[a]

Taxon	*Habitat*	*Concentration Ratio*
Trees[b]	Oak forest	0.03 - 0.25
Sorghum[c]	Micrososm	2 - 10
Vascular plants[d]	--	1
Vascular plants[e]	Pasture	1 - 5
Vascular plants[f]	--	0.6
Vascular plants[g]	Pasture	0.05 - 0.08
Trees[h]	Deciduous forest	0.5 - 1.0
Vascular plants[i]	Black Oak savannah	0.5 - 1.0
Corn[j]	Cropland	0.2 - 4.0
Earthworm[k]	Mine tailings	0.02 - 0.12
Voles[k]	Mine tailings	0.01 - 0.15
Shrew[k]	Mine tailings	0.01 - 0.15
Cow[e]	Pasture	8 - 16

[a] $[Zn]_{biota}/[Zn]_{soil}$.

[b] *H.M. Braunstein et al., "Environmental and Health Aspects of Disposal of Solid Wastes from Coal Conversion: An Information Assessment, Oak Ridge National Laboratory," Tennessee, ORNL-5361, 1978.*

[c] *P.Y. Lu et al., "Model Ecosystem Studies of Lead and Cadmium and of Urban/Sewage Sludge Containing These Elements," J. Environ. Qual. 4: 505-509, 1975.*

[d] *D.E. Reichle, P.B. Dunaway and D.J. Nelson, "Turnover and Concentration of Radionuclides in Food Chains," Nucl. Safety 11: 43-55, 1970.*

[e] *C.R. Dorn et al., "Environmental Contamination by Lead, Cadmium, Zinc, and Copper in a New Lead-Producing Area," Environ. Res. 9: 159-172, 1975.*

[f] *J K. Hodgson, "Chemistry of Trace Elements in Soils with Reference to Trace Element Concentrations in Plants," pp. 45-58 in Proceedings of the Third Annual Conference on Trace Substances in Environmental Health, D.D. Hemphill, ed., University of Missouri Press, Columbia, 1970.*

[g] *N.I. Ward, R.R. Brooks and E. Roberts, "Contamination of a Pasture by a New Zealand Base-Metal Mine," New Zealand J. Sci 20: 413-419, 1977.*

[h] *R.I. Van Hook, W.F. Harris and G.S. Henderson, "Cadmium, Lead, and Zinc Distributions and Cycling in a Mixed Deciduous Forest," Ambio. 6: 281-286, 1977.*

[i] *G.P. Parker, W.W. McFee and J.M. Kelly, "Metal Distribution in Forested Escosystems in Urban and Rural Northwestern Indiana," J. Environ. Qual. 7: 337-342, 1978.*

[j] *R.I. Pietz et al., "Zinc and Cadmium Contents of Agricultural Soils and Corn in Northwestern Indiana," J. Environ. Qual. 7: 381-385, 1978.*

[k] *R.D. Roberts and M.S. Johnson, "Dispersal of Heavy Metals from Abandoned Mine Workings and Their Transference through Terrestrial Food Chains," Environ. Pol. 16: 293-310, 1978.*

Table 6.17. Concentration Ratios for Zinc in Aquatic Systems[a]

Taxon	*Habitat*	*Concentration Ratio*
Algae and Vascular Plants[b]		140 - 33,500
Phytoplankton[c]	Riverine	>30,000
Phytoplankton[d]	Micrososm	300
Phytoplankton[e]	Microscosm	2,700 - 9,500
Vascular Plants[f]	Ash pond drainage	12 - 15
Herbivorous Invertebrates[b]		150
Herbivirous Invertebrates[c]	Riverine	16,000 - 536,000
Omnivorous Invertebrates[c]	Riverine	78,000 - 360,000
Carnivorous Invertebrates[c]	Riverine	140,000 - 180,000
Invertebrates[d]	Microcosm	150
Snail[e]	Microcosm	12,000 - 22,000
Invertegrates[f]	Ash pond drainage	5 - 38
Carnivorous Fish[b]		4 - 40
Carnivorous Fish[c]	Riverine	103,000 - 170,000
Omnivorous Fish[c]	Riverine	106,000 - 196,000
Mumichog[d]	Microcosm	10
Mosquitofish[e]	Microcosm	21,000 - 67,000
Mosquitofish and Tadpoles[f]	Ash pond drainage	20 - 30

[a] $[Zn]_{biota}/[Zn]_{H_2O}$.

[b] *D.E. Reichle, P.B. Dunaway and D.J. Nelson, "Turnover and Concentration of Radionuclides in Food Chains," Nucl. Safety 11: 43-55, 1970.*

[c] *R.V. Anderson, W.S. Vinikour and J.E. Brower, "The Distribution of Cd, Cu, Pb and Zn in the Biota of Two Freshwater Sites with Different Trace Metal Inputs," Holarctic Ecol. 1: 377-384, 1978.*

[d] *J.P. Baptist and C.W. Lewis, "Transfer of Zn-65 and Cr-51 Through an Estuarine Food Chain," pp. 420-430 in Symposium on Radioecology, D.J. Nelson and F.C. Evans, eds., Division of Biology and Medicine, U.S. Atomic Energy Commission, CONF-670503, 1969.*

[e] *P.Y. Lu et al., "Model Ecosystem Studies of Lead and Cadmium and of Urban/Sewage Sludge Containing These Elements," J. Environ. Qual. 4: 505-509, 1975.*

[f] *D.S. Cherry and R.K. Guthrie, "Toxic Metals in Surface Waters from Coal Ash," Water Res. Bull. 13: 1227-1236, 1977.*

apparently have altered the pattern of reinvasion of burned areas, burned areas on the polluted site contained more open ground than those on the control site, which led to increased soil erosion, exacerbating the impacts on the biotic community.

Impacts on Aquatic Ecosystems

In general, pollutants entering a more acid system are more likely to lead to toxic effects because of the higher solubility of some metals, including zinc. Because water normally has low zinc concentrations ($\sim$ 0.01 mg/L), zinc must be bioaccumulated in aquatic biota; this can lead quickly to toxicity if zinc concentrations increase markedly. Toxic levels of zinc would lead to modification of species composition and community structure as sensitive species were eliminated; disruption of decomposition and nutrient recycling would also be expected. Aquatic organisms

have been affected by exposure to concentrations as low as ~ 0.1 mg/L.[114] Other metals and pollutants associated with zinc emissions may exacerbate the effects of zinc.

Pathways to Humans

A few studies have traced the movement of zinc through human food chains. Although zinc does not tend to be bioaccumulated in terrestrial food chains, Lu et al.[134] found increased zinc levels in sorghum growing in a model ecosystem amended with sewage sludge. Corn growing on soils with elevated zinc concentrations in northern Indiana showed no significant difference in zinc levels from corn growing in control sites.[135] In another area, contaminated by lead mining and smelting operations, soil concentrations of zinc were significantly raised above control values, but this difference was not found in the tissues or milk of dairy cattle grazing in the experimental and control sites.[136] In both these studies, zinc concentrations in biota were 2- to 10-fold higher than in soils. Roberts and Johnson found that zinc concentrations in small mammals and herbivorous invertebrates were about the same or less than the concentrations in their diets near abandoned mine tailings and at control sites.[124] However, total body burdens of zinc did increase with increasing zinc in the diet.

In aquatic systems, zinc tends to be bioaccumulated above levels in the ambient water. Anderson et al. reported biotic concentrations ranging from 10,000 to 35,000 times water concentrations;[126] Lu et al. found similar results in a model ecosystem.[134] In the former study, zinc concentrations increased from the producer to the carnivore trophic levels, but in the model ecosystem zinc concentrations went down as the trophic level increased. Because aquatic biota tend to concentrate zinc at several orders of magnitude higher than ambient levels, high concentrations could develop in aquatic food sources such as fish or shellfish as a result of zinc emissions.[125,137]

6.2 ECOLOGICAL IMPACTS FOR THE BATTERY DEVELOPMENT SCENARIOS

In this report quantification of impacts from the battery cycles suffers from a paucity of information relating environmental responses to emission levels as well as from the inherent imprecision of our knowledge of emission levels because of uncertainties regarding future control technologies and regulations, future battery technologies, and actual levels of emissions. Thus, the assessments presented in this section cannot give a precise prediction of expected impacts. Quantification of the estimates serves primarily to identify those aspects of the battery cycle likely to lead to environmental problems.

The following discussions revolve around the year of peak emissions in the scenarios--the year 2000. By that time, control technologies are likely to be more advanced and new technologies may provide methods for mitigating emissions levels below the values used for this assessment.

Estimated permissible ambient concentrations (EPC) of emissions that will sustain public health (EPC_H) and provide environmental protection (EPC_E) are shown in Table 6.18. The data are derived principally from a review by Cleland and Kingsbury for the EPA.[138] Values for the protection of man health are derived from primary EPA standards, recommended criteria, or minimum dose levels known to be toxic to man or laboratory animals; those for environmental

Table 6.18. Estimated Permissible Ambient Concentrations (EPC) of Emissions from the Battery Cycles for Protection of Health and the Environment[a]

	EPC_H[b]			EPC_E[c]		
Emissions	*Air, µg/m³*	*Water, µg/L*	*Land, µg/g*	*Air, µg/m³*	*Water, µg/L*	*Land, µg/g*
Gaseous						
Cl_2	10[d]	40,000[d]	-	150[d]	2[e]	-
CO	10,000	522	-	10,000	30	-
Propylene	20,000	-	-	17,000	>50,000	>1,000
SO_2	365[f]	-	-	160[g]	-	-
Particulate						
Total Partic.	260[f]	-	-	150[h]	-	-
As	0.005	50	0.1	-	10	0.02
Cd	0.12	10	0.02	-	0.4	0.0004
Co	0.12	0.7	0.001	-	50	0.1
Cr	0.12	50	0.1	-	50	0.1
Cu	0.5	1,000	2	-	10	0.2
Fe	16,000[i]	300[e]	0.6[e]	-	1,000[e]	2[e]
Hg	0.1	2	0.004	1	50	0.1
KOH	5	75	0.15	-	-	-
Li	0.05	0.3	0.0006	-	75	0.15
Mg	14	83	0.2	-	43,000	87
Mn	12	50	0.1	-	20	0.04
Ni	0.24	1.4	0.003	-	2	0.004
Pb	0.36	50	0.1	-	10	0.02
Sb	1.2	7	0.014	-	40	0.08
Zn	9.5	5,000	10	-	20	0.04

[a] *Primary data source: J.G. Cleland and G.L. Kingsbury, "Multimedia Environmental Goals for Environmental Assessment, Vol. II, MEG Charts and Background Information," U.S. Environmental Protection Agency, Washington, D.C., EPA 600/7-77-136b, 1977.*

[b] *Based on health effects.*

[c] *Based on ecological effects.*

[d] *Based on data from "Chlorine and Hydrogen Chloride," National Academy of Sciences, Washington, D.C., 1976.*

[e] *From "Quality Criteria for Water," EPA 440/9-76-023, U.S. Environmental Protection Agency, Washington, D.C., 1976.*

[f] *U.S. Environmental Protection Agency 24-h primary standard.*

[g] *U.S. Environmental Protection Agency 3-h secondary standard normalized to 24-h.*

[h] *U.S. Environmental Protection Agency 24-h secondary standard.*

[i] *Based on data from Subcommittee on Iron, Committee on Medical and Biological Effects of Environmental Pollutants, "Iron" National Research Council, University Park Press, Baltimore, Maryland, 1979.*

protection are derived from EPA secondary standards, recommended criteria, or minimum levels known to be toxic to vegetation. The EPC values are lower than threshold values required to produce toxic effects. Because the effects of long-term exposures of organisms in nonlaboratory situations are poorly understood, safety factors have been applied to toxicity data in the derivation of the EPC values. The EPCs for soils are for plant-available concentrations and are, in general, much lower than the threshold levels for toxic effects to plants. In part, this is because of uncertainties as to how pollutants in soils will actually interact under the myriad of different permutations of ambient conditions that may exist. As a result, if a given pollutant concentration exceeds the stated EPC, an adverse impact will not necessarily occur; rather, a high potential for deleterious effect from this pollutant is indicated, which requires further scrutiny.

Atmospheric, soil, and runoff concentrations of emissions from battery-related facilities were estimated assuming a fixed wind direction and other worst-case conditions (Sec. 5.1). Soil and runoff concentrations could be one to two orders of magnitude lower than the predicted concentrations, depending on local conditions.

In our evaluation of atmospheric discharges, the concentrations of potentially hazardous constituents in the ambient atmosphere, soil, runoff, and lakewater are assumed to be zero, even though potentially toxic materials probably are already present. Interactions (antagonistic, additive or synergistic) between emission constituents have not been considered, because few data exist on these complex relationships. These interactions could result in toxic effects even though EPCs for individual constituents are not exceeded.

Assuming that wastewaters are discharged into streams, the following relationship is used to predict the receiving-stream flow rates required to achieve acceptable stream EPC_H and EPC_E values (Table 6.18) for potentially toxic discharge constituents with no sedimentation after complete mixing:

$$EPC = \frac{C_r D_r + C_e D_e}{D_r + D_e},$$

where EPC is the estimated permissible concentration of a given parameter in the receiving stream after compete mixing; C_r is the ambient receiving-stream concentration of a given parameter before effluent addition, assumed to be zero; C_e is the effluent concentration of a given parameter; D_r is the receiving stream flow rate; and D_e is the effluent flow rate. Ambient receiving stream concentrations of potentially toxic constituents are assumed to be zero, even though potentially toxic constituents probably are present. The presence of potentially toxic materials in receiving streams would necessitate additional dilution to achieve acceptable stream EPC values. Because complex interactions can occur between discharge constituents and receiving stream biota, an additive relationship was assumed; therefore, receiving-stream flow rates for each constituent were summed. The degree of environmental degradation from battery-related industries will depend on the quantity and quality of discharge, and receiving stream flow rates.

Soils and sediments serve as sinks for many constituents released from battery-related industries. Although incremental increases of constituents in soils and sediments may be small,

these concentrations will increase with time and the likelihood of adverse environmental impacts will increase with continued deposition. Our analysis emphasizes the annual increment of increase in metal concentration. Accumulation will occur over the lifetime of the facility. The magnitude of the impacts will vary from site to site and depends on prior rates of metal deposition, soil and water-body properties, background concentrations of metals in the environment, and leaching and sedimentation rates. Therefore, we have not tried to assess the cumulative impacts. Where annual incremental additions to the soils approach EPCs, we can expect cumulative effects of metals, particularly where leaching and mobilization rates are low.

Solid wastes from battery-related industries could contain potentially hazardous constituents, and adverse impacts could result from the toxic leaching of these wastes into soils, groundwater, and surface water. Leaching of potentially toxic constitutents from settling ponds and other treatment ponds into ground and surface waters could have adverse impacts. Leaching rates are dependent on a host of environmental variables; however, proper management of waste materials in accordance with the Resource Conservation and Recovery Act of 1976 (P.L. 94-580) should mitigate adverse impacts.[139]

Groundwater supplies could be adversely impacted, both quantitatively and qualitatively, by intersecting aquifers during mining operations. Physical surface disturbances, resulting in habitat destruction, will also occur as a result of establishing battery-related industries, especially mines and mills, and solid-waste disposal (mining overburden, mill tailings, sludge from treatment processes, and slag). However, land reclamation should mitigate effects associated with surface disturbances. Long-term land allocation will be required for many battery-related industries.

Direct environmental effects from battery use are expected to be immeasurable because of the essentially emission-free operation of the battery system. Indirect effects may be attributed to facilities generating electricity for recharging batteries. Hatcher summarizes the potential environmental effects for various alternative electricity-generating methodologies.[140] A worst-case situation would occur if all the electricity was generated from coal combustion steam generators. Environmental effects of the coal fuel cycle have been reviewed in a number of documents.[141-143] We have presented methods for estimating the amount of ambient particulate and SO_2 concentrations which might be attributed to the generation of electricity from coal in order to recharge 10,000 electric vehicles per power plant (Appendix D). The highest value for particulates estimated was 2 $\mu g/m^3$, two orders of magnitude below the EPC_E for air. Increasing the number of vehicles serviced per power plant to 100,000 still leaves a large margin between the EPC of 150 $\mu g/m^3$ and the concentrations attributable to recharging (20 $\mu g/m^3$). Thus, the impact of battery recharging on ambient particulate concentrations would only be a problem in those areas already exceeding or approaching EPA standards. To recharge 100,000 vehicles per plant, the estimated maximum emissions of SO_2 due to recharging are such as to yield ambient concentrations in excess of the EPC_E for aerial SO_2 (160 $\mu g/m^3$, Table 6.18). Thus, SO_2 emissions due to the battery recharging could pose a threat to the environment. However, it must be remembered that these are worst-case estimates representing stable atmospheric conditions near the power plant and serve only as indicators of where potential problems may arise.

6.2.1 Lead/Acid Battery Cycle

Primary Production of Input Materials

Mining and Milling. In the year 2000 antimony requirements will be several times total domestic mining output, which currently provides only 9% of the feed for domestic antimony smelters (see Sec. 2). Domestic environmental impacts have been discounted because foreign supplies are expected to fill the void. Environmental impacts due to arsenic mining have been discounted because the quantity required for battery use is small and because arsenic is a by-product of the lead industry. Thus, arsenic residuals have been incorporated with lead production residuals. Environmental impacts from sulfuric acid production have also been discounted; much of the needed acid can be produced as a by-product from primary lead smelting operations and the remaining deficit is small compared with present production levels. If domestic lead sources are to fill the requirements for lead/acid batteries, U.S. mine-mill production will have to increase by approximately 66% or six new mine-mill complexes by the year 2000.

Increased mining and milling of lead will result in increased fugitive dust emissions, mill wastewater, mill tailings, and loss of biotic habitat. Total atmospheric emissions from a single model lead mine-mill complex and the resulting pollutant concentrations in air, soil, and runoff are presented in Table 5.1. These are maximum concentrations. Actual concentrations could be one to two orders of magnitude lower than the values presented here, depending on the patterns of pollutant dispersal.

Projected ground-level atmospheric emission constituents could exceed EPC_H values (Table 6.18) 1 km from the source. The only projected ground-level atmospheric concentration exceeding EPC_H values at 50 km from the complex is that for lead. However, concentrations of several constituents are projected to be excessive at several kilometers from the complex. Thus, a large area downwind could be adversely impacted.

Incremental maximal concentrations of many emission constituents in the soil (Table 5.1) could exceed EPC_H and EPC_E values (Table 6.18) by several orders of magnitude 1 to 2 km from the model complex. This could lead to high concentrations in the immediate vicinity of mine-mill operations, particularly if soils already contain these constituents. Constituent concentrations in soils 30 to 50 km from the complex are projected to exceed EPC_H and EPC_E values for several constituents, i.e., lead, copper, zinc, iron, cobalt, nickel, cadmium, and manganese. Environmental biogeochemistries of major emission constituents are reviewed in Section 6.1. As noted in Section 6.1, high concentrations of metals can retard germination and inhibit nutrient recycling, resulting in disruption and possible elimination of ecosystem structure and function.

Projected runoff concentrations of many emission constituents (Table 5.1) could exceed EPC_H and EPC_E values (Table 6.18) by several orders of magnitude. Constituent concentrations in runoff 30 to 50 km from the complex are projected to exceed EPC_H and EPC_E values. The impact of runoff to receiving waters will be dependent on receiving stream flow rates or receiving lake volumes.

A moderate receiving stream flow rate could be required for dilution of wastewater discharges from the model lead mine-mill complex (Table 5.18) to achieve acceptable ambient EPC_H and EPC_E values (5 and 83 m^3/sec, respectively), assuming six complexes in the year 2000

(Table 6.19). Additionally, sedimentation of constituents may pose environmental problems in receiving systems.

Table 6.19. Receiving Water Flow Rates Required for Dilution of Lead/Acid Battery-Related Industry Discharges to Achieve Acceptable Stream Concentrations during the Year 2000 Assuming 3×10^6 Lead/Acid Electric Vehicles

	Health Effects	*Ecological Effects*	*Health Effects*	*Ecological Effects*
	$(m^3/s)/MWh$		m^3/s	
Mining and Milling of Lead	5×10^{-1}	7×10^{-3}	30	500
Primary Lead Subcategory	3×10^{-5}	2×10^{-4}	2	20
Battery Manufacturing				
Dry process and $Ca(OH)_2$ treatment	9×10^{-5}	4×10^{-4}	7	30
Dry process and NaOH treatment	9×10^{-4}	3×10^{-4}	70	20
Wet process and $Ca(OH)_2$ treatment	8×10^{-5}	3×10^{-4}	6	20
Wet process and NaOH treatment	3×10^{-4}	3×10^{-4}	30	30
Battery-Breaking Operations	2×10^{-5}	1×10^{-5}	2	0.8

Mill tailings containing potentially hazardous constituents from processing lead ore in the battery scenario may reach 17,000 MT/day in the year 2000 (see Sec. 2). Proper tailings management in accordance with the Resource Conservation and Recovery Act (RCRA) of 1976 should mitigate impacts of tailings leachate.

Smelting. Emission data are not available for air or water emissions from domestic antimony smelting, and arsenic is recovered as a by-product of lead smelting. Thus such matters are not discussed here. If domestic lead smelters are to fill the requirements for lead/acid batteries, U.S. production will have to increase by approximately 55% or four new complexes by the year 2000 (see Sec. 2).

Increased smelting of lead will result in increased stack emissions, fugitive dust emissions, wastewater, and solid wastes (sludges from treatment processes and slag materials). Total atmospheric emissions from a single model lead smelting complex and the resulting pollutant concentrations in air, soil, and runoff are presented in Table 5.2 These are maximum concentrations. Actual concentrations could be 1 to 2 orders of magnitude lower than the values presented here, depending on the patterns of pollutant dispersal.

Projected ground-level atmospheric lead concentrations (Table 5.2) could exceed the EPC_H value (3.6×10^{-1} µg/m^3) (Table 6.18) at 1 km. Projected ground-level atmospheric lead concentrations are not projected to be excessive at 50 km from the complex. However, concentrations could be excessive at several kilometers from the complex. Thus, a large area downwind could be adversely impacted. The environmental biogeochemistry of lead is outlined in Section 6.1.

Incremental maximal concentrations of lead in soil (Table 5.2) could exceed EPC_H and EPC_E values (0.1 and 0.02 µg/g, respectively) (Table 6.18) 30 to 50 km from the model complex. This This could lead to high concentrations in the vicinity of smelter operations, particularly if soils already contain lead.

Projected lead concentrations in runoff could exceed EPC_H and EPC_E values (50 and 10 µg/L, respectively) (Table 6.18) 30 to 50 km from the model complex. Impacts of runoff to receiving waters will be dependent on receiving stream flow rates or receiving lake volumes. However, excessive lead could pose a threat to aquatic biota.

A small receiving stream flow rate is projected to be required for dilution of wastewater discharges from the model smelting complex (Table 5.19) to achieve acceptable ambient EPC_H and EPC_E values, 5 m^3/sec, assuming four complexes in the year 2000 (Table 6.19).

Solid wastes from lead smelting for the battery commercialization scenario may reach 567 MT/day in the year 2000 (see Sec. 2). Proper management in accordance with RCRA (1976) should mitigate impacts of solid wastes leachate.

Manufacturing Process

Battery. For the projected scenario of 3×10^6 lead/acid battery-powered EVs, the existing U.S. lead/acid battery manufacturing industry will have to increase by approximately 94% of its existing capacity, or by 23 new plants by the year 2000 (see Sec. 2). Total atmospheric emissions from a single model battery manufacturing plant and the resulting air, soil, and runoff constituent concentrations are presented in Table 5.4. These are maximum concentrations. Actual concentrations could be 1 to 2 orders of magnitude less, depending upon the patterns of pollutant dispersal.

Environmental levels of antimony and arsenic could approach but are not projected to exceed EPC_H and EPC_E values (Table 6.18) 1 to 2 km from the plant. Environmental lead concentrations could approach or exceed EPC_H and EPC_E values for ground-level atmospheric and resulting soil and runoff concentrations up to 50 km from the plant. Ecosystem structure and function could be disrupted by elevated lead concentrations downwind from the model plant, particularly if the soils already contain high lead concentrations.

Little dilution of wastewater discharges from the model battery manufacturing industry (Tables 5.20 and 5.21) should be required to achieve acceptable ambient EPC_H and EPC_E values for various processes assuming 23 new complexes in the year 2000 (Table 6.19).

Solid wastes containing potentially hazardous constituents from battery manufacturing could vary from 1.5 to 1700 MT/day in the year 2000 depending on wastewater treatment processes (see Sec. 2). Proper management in accordance with RCRA (1976) should mitigate impacts of solid waste leachate.

Case and Cover. Polypropylene requirements for battery cases and covers will be a small fraction of the existing polypropylene industry (see Sec. 2.4.2). Model manufacturing industry atmospheric emissions, and resulting soil and runoff constituent concentrations for the year 2000 are presented in Table 5.5. EPC_H and EPC_E values are not expected to be exceeded by the discharge constituents identified.

Polypropylene industry wastewaters have been poorly characterized and quantified; it is known, however, that wastewaters contain suspended solids, vanadium, titanium, aluminum, solvents and inhibitors in unknown quantities. These constituents have the potential to cause adverse impacts when released into the environment (see Sec. 2.4.2).

Proper management of solid wastes containing potentially hazardous constituents in accordance with RCRA (1976) should mitigate impacts of solid wastes leachate.

Battery Use

The major effects from projected EV battery use will be indirect, from the generation of electricity for recharging (see Sec. 6.2.1). Arsine and stibine production during battery charging probably is the most serious direct environmental problem associated with lead/acid EVs. These gases are extremely toxic and decompose rapidly to oxide dusts (see Sec. 2.5); both gases and oxides could collect in closed garages and present human health problems, but are not expected to pose major environmental problems. Another direct effect is the production of potentially explosive hydrogen and oxygen gases during charging. Again, these are not expected to pose major environmental problems. Ozone is produced in small quantities during EV operation, but is unlikely to cause environmental problems.

Battery Recycling

Battery Breaking. U.S. lead/acid battery breaking operations, by the year 2000, will have to increase by approximately 78%, or by 13 new battery breaking facilities. Information concerning atmospheric discharges from battery breaking is lacking, but the potential quantities to be emitted are considered small (see Sec. 2).

Little dilution of wastewater discharges (Table 5.22) is projected to be required to achieve acceptable EPC_H and EPC_E values (Table 6.19).

Proper management of solid wastes, 9.3 MT/day in the year 2000, containing potentially hazardous constituents in accordance with RCRA (1976) should mitigate impacts of solid wastes leachate.

Secondary Smelting. U.S. secondary lead smelting will have to increase by 44% or 13 new smelters by the year 2000 to meet EV battery industry needs (see Sec. 2). Increased secondary lead smelting will result in increased stack emissions and solid wastes. No wastewater discharges are expected. Total atmospheric emissions from a single model secondary smelter and the resulting air, soil, and runoff constituent concentrations are presented in Table 5.3. These are maximum concentrations. The actual concentrations could be 1 to 2 orders of magnitude lower than the values presented here, depending on the patterns of pollutant dispersal.

Projected ground-level atmospheric concentrations of lead could exceed the EPC_H for lead (3.6×10^{-1} $\mu g/m^3$) at 1 km from the smelter. Lead concentrations are not projected to be excessive at 50 km. However, they could be excessive several kilometers from the smelter.

Incremental maximal concentrations of lead and antimony in the soil from stack emissions are projected to exceed EPC_H and EPC_E values (1×10^{-1} and 1.4×10^{-2} $\mu g/g$, respectively). The

environmental biogeochemistries of lead and antimony are outlined in Section 6.1. Ecosystem structure and function could be disrupted downwind from the model smelter.

Projected lead and antimony concentrations in runoff could exceed EPC_H and EPC_E values (Table 6.18) 1 to 2 km from the smelter. Lead concentrations are projected to be excessive 30 to 50 km from the smelter. Impacts to receiving waters will be dependent on receiving stream flow rates and receiving lake volumes. However, excessive lead and antimony concentrations could pose a threat to aquatic biota.

Solid waste containing potentially hazardous constituents from secondary lead smelting may reach 570 MT/day in the year 2000 (see Sec. 2). Proper management in accordance with RCRA (1976) should mitigate impacts of solid wastes leachate.

6.2.2 Ni/Zn Battery Cycle

Primary Production of Input Materials

Mining and Milling. In the Ni/Zn battery scenario, increased mining and milling of zinc and nickel will result in increased fugitive dust emissions, mill wastewater, mill tailings, and loss of biotic habitat (Sec. 3.3). Habitat loss from surface mining and preemption of land for tailings storage has not been quantified because the area involved varies substantially with the nature of the ore being mined, its depth, and the width of the ore seam. This impact is expected to be greatest for the nickel mining industry, which is predicted to increase to about 150% of current production by the year 2000 (see Table 3.9). Cobalt production will also increase by about the same percentage over that time period, but is expected to total only about 5% of nickel production. Mining effects can be mitigated by land reclamation, although in many areas it may be several decades before the original vegetation structure of the habitat is restored.

Approximately 5 new model zinc mine-mill complexes will be required by the year 2000. In general, total fugitive dust emissions are not expected to exceed the estimated permissible concentration (EPC) of 150 $\mu g/m^3$ for protection of the environment in the area of a model zinc mining and milling operation (see Table 5.6). Lead is the only metal likely to pose human health problems. Maximum aerial lead concentrations could exceed the EPC_H (0.36 $\mu g/m^3$) to a distance of over 1 km downwind from the mine-mill. Thus, a large area downwind is unlikely to be adversely impacted by aerial exposure to lead.

All values given for incremental maximal concentrations of metals in the soil (see Table 5.6) surpass or approach the EPCs for protection of the environment. This could lead to serious problems in the immediate vicinity of mine-mill operations, particularly if the soils are already high in metals content. As noted in Section 6.1, high concentrations of metals can retard germination and inhibit nutrient recycling, resulting in the disruption and possible elimination of ecosystem structure and function. Depending on patterns of pollutant dispersal, impacts could extend to 50 km from the site.

Some of the metals that are deposited will be removed in runoff. The amount of this runoff will depend on the amount of local rainfall and local soil properties. Lead and zinc are at high enough concentrations in runoff near the mine that the metals in the runoff would raise

the concentrations of lake or pond receiving waters near or above environmental EPCs for water bodies (Table 5.6). Runoff into fast-moving streams or rivers may not be a severe problem if the streams are moving fast enough to dilute the incoming metals below critical levels.

The zinc mine-mill complexes are estimated to discharge wastewater at a rate of 400 L/day/MWh, reaching a maximum discharge rate in the year 2000 of 8×10^7 L/day (Table 5.23). By the year 2000, the discharge into riverine systems would require receiving-water flow rates of 220 m^3/sec in order to dilute the effluent from the total industry to permissible levels for protection of the aquatic environment (Table 6.20). However, five new complexes are expected to meet the demands and, therefore, five receiving streams flowing 44 m^3/sec would be required for adequate dilution of mine-mill discharges.

Table 6.20. Receiving-Water Flow Rates Required for Dilution of Ni/Zn Battery-Related Industry Discharges to Achieve Acceptable Stream Concentrations during the Year 2000, Assuming 8×10^6 Ni/Zn Electric Vehicles

	Health Effects	*Ecological Effects*	*Health Effects*	*Ecological Effects*
	$(m^3/s)/MWh$		m^3/s	
Model Zinc Mine-Mill Complex	4×10^{-5}	1×10^{-3}	1×10^{1}	2×10^{2}
Model Canadian Nickel Operation	5×10^{-3}	4×10^{-3}	1×10^{3}	1×10^{3}
Model Primary Zinc Operation	1×10^{-4}	6×10^{-4}	2.5×10^{1}	1×10^{2}
Model Caustic Potash Operation	8×10^{-9}	4×10^{-8}	1.5×10^{-3}	1×10^{-2}
Model Nickel-Zinc Battery Manufacturing Plant	2×10^{-1}	8×10^{-1}	3×10^{4}	1.5×10^{5}

Mill tailings from processing zinc ore in the battery scenario may reach 3.2×10^7 kg/day in the year 2000. If we assume that tailings composition is similar to that of the typical ores discussed in Section 3, zinc and lead are the most likely sources of potential adverse effects. Most problems with metal toxicity will arise from leaching from the tailings into the surrounding soils, groundwater and surface water. The rates of leaching will depend on the amount of local rainfall, the chemical nature of the tailings, the type of impoundment or landfill containing the tailings, and the nature of the surrounding soils. Proper tailings management in accordance with the Resource Conservation and Recovery Act of 1976 should mitigate to a major extent the impacts from the leachate. Heavy-metal content may make reclamation difficult, but metalliferous tailings have been successfully reclaimed.[144]

Emission values for nickel mining and milling are not as well defined as for zinc production (see Sec. 3.3.2). It appears that total wastewater effluent will be three to four times higher for nickel than for zinc (Table 5.24); thus, the potential for degradation of receiving waters is higher for nickel mining and milling operations. Solid waste production is estimated to be seven to eight times higher for nickel than for zinc. The nature of the nickel tailings is not quantified, but it is expected that other heavy metals in addition to nickel, such as copper, lead, and cadmium, will be the major potential pollutants. It is likely, however, that

increased demand for nickel will require opening several new mines and dividing the total expected waste effluents among several different sites. Thus, the impacts may be about the same as those described for a single zinc mine-mill complex.

A number of factors may mitigate or exacerbate these impacts. If zinc mining-milling operations are in areas of high metal concentrations in soils and aquatic systems, then additional metal inputs have a high potential for inducing adverse effects on local biota. In addition, the acidity of the soil or water, human sensitivities to the metals, abilities of biota to bioaccumulate or biomagnify metals, and availability of the metals to biota, all will play a roll in determining the magnitude of the impact on the surrounding ecosystems, as discussed in Section 6.1.

Smelting. Smelting and refining of the metal ores are major sources of aerial pollutants from the battery cycle. Zinc smelting is expected to release gaseous and particulate pollutants at a rate of about 0.1 (kg/day)/MWh (see Table 3.5); and nickel smelting is expected to release about 3 (kg/day)/MWh of aerial pollutants (Table 3.8). Over 95% by weight of these emissions are expected to be gaseous SO_2.

Maximum ground-level aerial concentrations of metals near a model zinc smelter are not expected to approach EPCs for the protection of health (Table 5.7). It is expected that at least two new smelters will be required to meet the zinc demand for battery production in 2000.

Emissions from increased nickel smelting are expected to increase by nearly 50% of current levels by the year 2000 (see Table 3.9). Concentrating this increase into a single, model smelter could result in aerial concentrations of most metals that exceed EPCs for health, especially if the prevailing winds or topography restrict the area of distribution, as in the Sudbury region of Ontario, Canada. Excessive concentrations could extend several kilometers from the smelter. However, it is unlikely that all the increased production can be handled by a single plant; production may be spread over two or more operations. If concentrations are diluted by a factor of two, potentially toxic levels of metals will remain, although the areal extent of the effects around each plant will be reduced (Table 5.8). Nickel still may exceed EPCs up to 50 km from the facility.

The SO_2 emissions from model smelters of both nickel and zinc are expected to exceed EPCs (160 $\mu g/m^3$) for the environment for some distance around the plants (Tables 5.7 and 5.8). A model zinc plant could have potential impacts to over six kilometers, even if prevailing winds do not concentrate the stack plume. If nickel production were spread over two model plants, SO_2 could exceed EPCs for environmental protection for over 50 km from the plant. Thus, it appears that SO_2 emissions are the major potential sources of impact on air quality in the battery cycle. Because control technology is expected to improve, we do not expect impacts due to increased smelting to approach the magnitude of those described in Section 6.1 for operating zinc and nickel smelters. However, the expected impacts will be similar in quality.

The maximum rates of accumulation of metals in the soils exceeds the EPC for environmental protection around both zinc and nickel model plants (Tables 5.7 and 5.8). If the zinc production is distributed between two new plants, zinc (land EPC = 0.04 $\mu g/g$) and cadmium (EPC = 0.0004 $\mu g/g$) remain potential hazards at distances over 50 km. Were the nickel production in the year 2000 distributed over two model plants, nickel (EPC = 0.004 $\mu g/g$), lead (EPC = 0.02 $\mu g/g$), and cadmium annual increases in soil could still be major potential problems to over 50 km from the plant.

The major effects of accumulating sulfur in the soil will be an increase in acidity (see Sec. 6.1.2). It is difficult to quantify the degree to which the acidity will be increased, but deposition rates near the model plants are quite high, even if production is distributed over several plants. Maximum deposition rates within 10 to 15 km of the model nickel plants exceed or equal deposition rates measured near the Coniston smelter in Ontario.[84] Vegetation and soil impacts are noted at up to ∿ 20 km from that smelter. This suggests that the annual rates of SO_2 deposition predicted from the scenario do pose potential threats to ecosystems near smelters affected by the nickel/zinc battery scenario.

By the year 2000, runoff concentrations of metals from soils near nickel smelters are expected to exceed EPCs for the environment even if production is distributed over two plants (Table 5.8). Maximum nickel concentrations (water EPC_E = 2 μg/L) and lead concentrations (water EPC_E = 10 μg/L) are expected to be higher than the EPC_E over more than 50 km downwind. This could result in impacts when runoff enters lakes, ponds, or slow-flowing bodies of water where concentrations cannot be readily diluted. Zinc smelter operations pose a similar environmental threat to a smaller but measurable extent.

Interpretation of the estimates of SO_2 deposition and runoff is again difficult. As in soils, the main impact is acidification (Sec. 6.1.2). Annual increases of sulfur in a large area around each of two model nickel smelters would approach or exceed concentrations found in acidified lakes in the Sudbury region.[109] The potential for impacts from acidification of aquatic systems near smelters appears quite high under the Ni/Zn battery scenario.

Assuming that two zinc and two nickel smelters will share the production called for under the Ni/Zn battery scenario, approximately 50 and 500 m^3/s, respectively, of receiving water flow per plant will be needed to dilute effluent input from each smelter (Table 6.20). Thus, it appears that rivers of modest to large flow will be able to handle wastewater effluents from the smelter operations.

We have been unable to quantify solid wastes from nickel smelting, but solid wastes from zinc smelters are predicted to be about 2.4×10^4 kg/day in 2000 (from Table 3.3-4). Leaching of zinc, lead, and cadmium from disposal of solids is likely to pose the greatest threat of impact to biota. Disposal and management of the sludge under proper conditions will reduce the potential for leaching. There have been moderately successful attempts at reclaiming and stabilizing solid wastes ponds for nickel/copper smelters in the Sudbury region of Ontario, Canada.

Caustic Potash Production. Approximately two new caustic potash producing complexes will be required by the year 2000. The major air emissions from caustic potash production are the gases chlorine (air EPC_E = 150 μg/m^3) and carbon monoxide (EPC_E = 10,000 μg/m^3) (Table 3.10). The predicted ground-level atmospheric concentrations for these gases near a model production plant are several orders of magnitude below the EPCs for environmental protection (Table 5.9).

The major toxic element in wastewater effluent is lead. Assuming that emissions rates meet EPA water effluent standards (Table 3.10), a relatively low flow is required for the environmental protection of receiving waters (Table 6.20). Solid waste production is also expected to be low (Table 3.10), reaching about 2000 kg/day by the year 2000. The major elemental species are magnesium and calcium which are relatively non-toxic. Proper management of the wastes will alleviate potential problems from leaching of toxic elements from the waste disposal sites.

Manufacturing Process

Battery. Approximately 50 new nickel/zinc battery manufacturing plants will be required by the year 2000. The major emissions from a battery manufacturing plant are fugitive dust and wastewater effluent (Table 3.14). Aerial concentrations of nickel (air EPC_H = 0.24 μg/m^3) and cobalt (EPC_H = 0.12 μg/m^3) could exceed health EPCs for much of the area around the plant (Table 5.10). Electrolyte mist (KOH) is unlikely to be a problem except perhaps in the immediate work area.

Accumulation of zinc (soil EPC_E = 0.04 μg/g), nickel (EPC_E = 0.004 μg/g), and cobalt (EPC_E = 0.1 μg/g) in soils poses a potential threat to the environment around battery manufacturing plants (Table 5.10). Accumulation rates of KOH in the soil also exceed EPCs (0.15 μg/g) within a few kilometers of the model plant. In the region of battery manufacturing plants, there appears to be a high potential for deleterious effects upon soils and biota similar to impacts discussed in Section 6.1.

Runoff in the vicinity of a model plant is also expected to bear high concentrations of metals and electrolyte (Table 5.10), even with distribution of production over 50 plants. Maximum nickel concentrations in runoff exceed or approach water EPCs. Deleterious impacts are likely in receiving waters with low rates of mixing. Annual accumulation of pollutants in the model lake predicted for the year 2000 also approach or exceed the nickel EPC for environmental protection up to about 10 km from each of 50 model plants. In order to meet acceptable stream concentrations of wastewater effluent, each of 50 model plants would have to discharge into receiving waters flowing at 3000 m^3/s or more (Table 6.20). Thus, except for very large rivers, the flow of pollutants from battery manufacturing into aquatic systems has a high potential for inducing deleterious effects upon the biota. Future regulations may restrict the amount of this effluent that can be discharged and require alternative treatment.

Case and Cover. Emissions from this stage of the battery cycle include fugitive dust and propylene (Table 3.12). We have assumed that a single model plant is the source of these emissions because, by the year 2000, one plant can handle the required production (Table 3.13). Aerial concentrations of propylene around the model plant are not expected to exceed EPC (17,000 μg/m^3) in the immediate vicinity of the plant. Emissions from case and cover manufacture do not appear to be major air quality problems.

Particulate fallout near the model plant in the year 2000 is expected to result in a maximum rate of increase in soil concentration of about 580 μg/g·yr (Table 5.11). It is difficult to assess the impact of this increase without greater knowledge of the composition of the particulate emissions. Maximum rates of increasing soil propylene concentration may exceed the environmental EPC (> 100 μg/g) in the immediate vicinity of the plant. Effects are not expected to be marked or widespread.

Deposition of propylene into water or inclusion of propylene in runoff is not expected to result in concentrations which pose an environmental threat (Table 5.11). The maximum concentration in runoff expected in the year 2000 is at most about one-tenth of the EPC (> 50,000 μg/L) for environmental protection. Impacts of particulates from deposition, runoff, and wastewater effluent cannot be quantitatively addressed. The effects are dependent upon the chemical composition of the particulates and natural level of suspended solids in the receiving water body.

Solid emissions are expected to be on the order of 400 to 4000 kg/day by the year 2000. Proper management and disposal of these materials should reduce the potential for impacts from toxic leachates. Without knowledge of the composition of the sludges, we cannot readily assess the potential for impacts.

Battery Use

Direct environmental effects from battery use are expected to be immeasurable because of the essentially emission-free operation of the battery system. Indirect effects may be attributed to facilities generating electricity for recharging batteries as discussed above.

Battery Recycling

Section 3 presents a characterization of the emissions from materials recovery of Ni/Zn batteries. It has not been possible to quantify these emissions due to the uncertainties surrounding the technologies that will be used. Predominant air emissions will be fugitive dusts containing zinc, nickel and other metals. These probably would not be spread over a large area because the batteries must be kept wet to reduce the chances of zinc combustion. Dust containing asbestos from separators could result from the dismantling operation, but it appears unlikely that toxic concentrations of asbestos would occur except, perhaps, in the immediate work area. Electrolyte (KOH) mists may be induced during emptying of the electrolyte but would probably pose little environmental threat. Misting is not likely to be higher than during battery manufacture (see above).

The major environmental problems could be associated with liquid and solid wastes associated with recovery. Heavy metals (Ni, Zn, Co, Cd) are expected to be the major factors posing an environmental threat. The wastes can be either highly acidic or highly alkaline, depending upon the process involved. Wastewater from the front-end operations will be highly alkaline, because of the disposal of the electrolyte (KOH). Generally, effluent from the hydrometallurgic processes will be highly acidic.

6.2.3 Ni/Fe Battery Cycle

Primary Production of Impact Materials

Mining and Milling. As with the other battery cycles discussed in the preceding pages, mining and milling of nickel produces fugitive particulates, tailings, wastewater from milling, and loss of habitat. The nickel/iron battery scenario calls for about 60% of the production of nickel in the nickel/zinc scenario (Table 4.6). The impacts from mining and milling in the Ni/Fe scenario are expected to be proportionally lower than those discussed in Section 6.2.2. We have not quantified mining and milling impacts from the iron industry because the required material can be readily obtained from scrap and is insignificant in comparison to the total industry (Sec. 4.3.1).

Processing and Smelting. Maximum ground-level atmospheric concentrations of metals and total particulates do not approach or exceed the health EPCs (Table 5.12). Because the maximum required steel production in the scenario is only about 1% of current production (Table 4.4), it

is unlikely that impacts due to battery production increases will be measurably higher than current impacts.

The maximum annual rates of metal accumulation in the soil near the model steel plant exceeds the environmental EPCs for most of the major metal emissions (Table 5.12). However, these rates are again unlikely to be measurable above accumulation due to current steel production. The same is true for runoff concentrations and deposition into lake systems (Table 5.12). Wastewater effluent due to the Ni/Fe battery scenario is not expected to require a large flow rate in the receiving waters in order to achieve acceptable levels of dilution (Table 6.21). Impacts from nickel processing would be about 60% of the amount described in Section 6.2.2 (Table 5.14).

Table 6.21. Receiving-Water Flow Rates Required for Dilution of Ni/Fe Battery-Related Industry Discharges to Achieve Acceptable Stream Concentrations During the Year 2000, Assuming 8×10^6 Ni/Fe Electric Vehicles

	Health Effects	*Ecological Effects*	*Health Effects*	*Ecological Effects*
	$(m^3/s)/MWh$		m^3/s	
Model Canadian Nickel Operation	3×10^{-3}	3×10^{-3}	1×10^{3}	8×10^{2}
Model Lithium Mine-Mill Complex	0	0	0	0
Model Iron and Steel Factory	0	2×10^{-6}	0	7×10^{-1}
Model Caustic Potash Operation	7×10^{-9}	4×10^{-8}	2×10^{-3}	1×10^{-2}
Model Nickel-Iron Battery Manufacturing Plant	2×10^{-2}	1×10^{-2}	5×10^{3}	4×10^{3}

Production of Lithium Hydroxide. Approximately five new model LiOH facilities will be required in the year 2000. We have been unable to quantify the composition of the particulate emissions from LiOH production. Maximum expected aerial concentrations of particulates are 1 to 2 orders of magnitude below the EPC for protection of the environment (Table 5.13). Impacts of accumulation of particulates in soils and water cannot be assessed further without knowledge of the particulates composition, nor can the impacts of solid waste be quantified. Proper management of the latter should mitigate most problems with leachate from wastes. The production of KOH electrolyte is not expected to have any measurable adverse environmental impacts (Table 5.15).

Manufacturing Process

Battery. Approximately 50 new model Ni/Fe battery manufacturing plants will be required in the year 2000. The aerial emissions from Ni/Fe battery manufacture include iron and steel dusts and electrolyte mists (Table 4.13). Concentrations of electrolyte immediately adjacent to the plant are not expected to exceed EPC (5 $\mu g/m^3$) (Table 5.16). Steel and iron dust concentrations in the year 2000 are not likely to exceed EPC (150 $\mu g/m^3$) for total particulates.

For several kilometers, maximum annual rates of accumulation of iron and steel in the soil will exceed land EPC (2 μg/g) for iron by the year 2000 (Table 5.16). Thus, there is potential for deleterious effects to vegetation surrounding the plant. Maximum rates of accumulation of KOH electrolyte also exceed the EPC (0.15 μg/g) for protection of health, suggesting possible harmful environmental effects may be associated with deposition of electrolyte mist.

Maximum runoff concentrations of iron (water EPC_E = 2 μg/L) exceed EPCs for environment (Table 5.16). This suggests that slow-flowing receiving waters may be adversely impacted by battery manufacturing. Impacts from wastewater effluent are expected to be similar to the Ni/Zn battery scenario (Table 4.13). Nickel effluents may pose a problem for biota in the receiving waters.

Cover and Case. Impacts from the manufacture of covers and cases are expected to be approximately 1.5 times the effects described in Section 6.2.3 (Table 5.17).

Battery Use

As with the other battery systems, direct effects from use of the Ni/Fe battery are expected to be negligible. The major effects will be indirect from the generation of electricity for recharging (as described above).

Battery Recycling

As with the Ni/Zn battery system, it is impossible at this time to present a specific assessment of emissions from Ni/Fe battery recovery. In general, the impacts from these emissions should be similar to those associated with the other battery systems. Iron would replace zinc as a major component of the emissions, but iron is generally considered less toxic (Table 6.18). Lithium would also be a major component of emissions. Effects from electrolyte misting and disposal are expected to be of lesser magnitude than with the Ni/Zn system. We anticipate that most of the electrolyte will be discarded prior to transport to the recovery operation and impacts would be mitigated by dispersing the recycling operations over a greater number of sites.

6.3 SUMMARY

1. The elements emitted to the environment from the battery cycles are cycled among biota and their physical environments to some degree.

2. Bioaccumulation of these elements above ambient levels is likely in aquatic ecosystems but not in terrestrial ecosystems. In a terrestrial ecosystem higher-than-normal tissue concentrations occur in polluted areas containing these elements.

3. Biomagnification of elements along the food chain is not likely to be important for the major constituents of the emissions from the battery cycle.

4. The lead/acid battery-related aerial emissions and wastewater constituents could cause adverse human health and ecological impacts within several kilometers of the operations (Sec. 6.2.1). Lead, arsenic, and antimony appear to be hazardous emission constituents and exceed estimated permissible concentrations for the protection of health (EPC_H) and for the protection of ecology (EPC_E) (Table 6.18) in one or more of the battery-related industries (Tables 5.1-5.5 and 5.18-5.22).

5. Zinc, nickel, lead, cadmium, cobalt, and sulfur dioxide appear to be the major hazardous emission constituents for the nickel/zinc battery cycle exceeding EPC_H and EPC_E values within several kilometers of the facilities (Sec. 6.2.2 and Table 6.18) in one or more of the battery-related industries (Tables 5.6-5.11 and 5.23-5.27).

6. Nickel, cobalt, lead, total particulates and sulfur dioxide appear to be major hazardous emissions constituents for the nickel/iron battery cycle exceeding EPC_H and EPC_E values within several kilometers of the facilities (Sec. 6.2.3 Table 6.18) in one or more of the battery-related industries (Tables 5.12-5.17 and 5.28-5.32).

7. Battery use is not expected to have measurable direct adverse environmental impacts. Indirect effects due to electricity generation for battery charging are addressed in Section 7.

8. Disposal of solid wastes in accordance with the Resource Conservation and Recovery Act (1976) in all battery-related industries should have minimal impact on the environment.

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7. BIOMEDICAL AND HEALTH EFFECTS OF EMISSIONS FROM THE BATTERY CYCLES

ABSTRACT

The biological and medical effects of the toxic agents identified in Sections 2 through 5 as being of concern to human health in an expanded production of the three near-term EV battery systems--lead/acid, Ni/Zn, and Ni/Fe--are discussed in this section. In addition, known effects of these agents on human health are identified. For each toxic agent, the following information is reviewed and evaluated: (1) pathways for tissue uptake, retention, and excretion; (2) toxic responses; and (3) means for protection from exposure, remediation of toxic responses, and diagnosis of the extent of exposure. The focus is on the chemical forms of these toxic agents, the modes of human exposure, and the levels of exposure anticipated as being relevant to battery production for EV use.

For the lead/acid battery, the toxic agents reviewed are lead (Pb), stibine (SbH_3), arsenic (As), arsine (AsH_3), and antimony trioxide (Sb_2O_3). For the Ni/Fe and Ni/Zn systems, nickel, cobalt, and cadmium are reviewed for their potential impact on human health from occupational and environmental exposures. An assessment is also presented of the human health impact that may result from central power station emissions required to charge 10,000 battery-powered EVs for civilian use. A demographic projection analysis is used to evaluate the impact of various vehicle and battery configurations in terms of their impacts on life shortening in a general population.

Research recommendations are outlined in Appendix G based on an analysis of the metabolic and toxicological data presented. Issues of concern to human exposure and also unique to the EV battery systems are: (1) a need for better measurements of ambient stibine levels produced during charging of lead/acid EV batteries containing antimony; (2) potential toxic responses to low levels of stibine or antimony trioxide in the home garage; (3) potential toxic responses to nickel compounds involved in battery manufacture and those industries involved in domestic recycling of nickel.

7.1 ANTIMONY AND THE GAS STIBINE

Stibine (SbH_3), the volatile hydride of antimony (Sb), is a colorless, water-soluble, inflammable gas, denser than air, with a strong reducing potential.[1] It is unstable in air, with a 6- to 12-minute half-life at room temperature, decomposing to antimony trioxide (Sb_2O_3).[2] It is rapidly decomposed at 150°C.[1] Stibine is produced by (1) contact between nascent hydrogen and metallic or soluble antimony, (2) the action of dilute acid or water on certain binary metallic compounds of antimony, or (3) charging of batteries that contain antimony as a component.

Stibine generation during normal charging and equalization charging of lead/acid batteries has been identified in Section 2 as a potential concern to human exposure. Table 7.1 shows the amount of antimony expected to be produced as stibine during one charging cycle and over one year's use of an EV. According to these values, if a single EV were charged in a 108 m^3, two-car

Table 7.1. Antimony Produced from Stibine Generation during Charging of a Lead/Acid EV Battery

Conditions	One Cycle,[a] mg	One Year,[b] g
Regular Charge	68	8.3
Equalization Charge		
at 2.45 V	41	1.2
at 2.65 V	354	10.6
	Total	9.5 - 18.9

[a] *Data from Section 2: 1 mg SbH_3 ≡ 0.94 mg Sb.*

[b] *Assumes one charge every 2.4 days, with one equalization charge after every four regular charges (see Sec. 2).*

garage (6m × 6m × 3m), the following concentrations of stibine might be produced by the end of charging:

	One Regular Charge	One Equalization Charge	
		2.45 V	2.65 V
SbH_3(mg/m^3)	0.67	0.40	3.47

The values above indicate that levels from 1 to 7 times the threshold limit value (TLV) for stibine of 0.5 mg/m^3 might be produced at the end of one charge cycle; these are estimates of maximum possible levels, however, which would occur only if all the stibine formed inside the battery were released completely and uniformly into the garage without decomposition.[3] Actual levels anticipated are lower, as it is likely that the stibine produced will be partially released and will decompose to Sb_2O_3. A review of current information on the toxicity of stibine is nevertheless warranted. Information on its toxicity at low levels should be evaluated and the basis for setting the current TLV scrutinized.

Because stibine has a low stability, decomposing to Sb_2O_3, it is appropriate to analyze the hazards of exposure to Sb_2O_3 (see also Sec. 7.1.2). During one year, approximately 10 to 20 g of antimony as Sb_2O_3 would be formed during charging, depending on the voltage used for equalization charging (Table 7.1). The actual amount of Sb_2O_3 released into the garage would depend on the amount of stibine released from the battery. If all the stibine formed were released, uniform dispersal of one year's accumulation of Sb_2O_3 into the air of a 108 m^3, two-car garage, e.g., when dust is stirred up by thorough cleaning of the garage, would result in 100 to 200 mg Sb/m^3 as Sb_2O_3. More realistically, if only about 5% of the stibine formed during charging were released, uniform dispersal of the dust after one year of charging would result in 5 to 10 mg Sb/m^3 as Sb_2O_3. This level is 10 to 20 times the TLV for use of Sb_2O_3 (0.5 mg/m^3). Dispersal in the garage of the settled dust from one year of charging would not be expected to occur often, however, and exposure to this level would thus be sporadic. More than one year's accumulation of dust would result in proportionally higher levels of Sb_2O_3 on dispersal. In addition, antimony would be in the air at the end of any single cycle. If only 5% of the stibine formed were released and decomposed, levels of from 0.04 to 6.2 mg Sb/m^3 as Sb_2O_3

would be present at the end of one charging. Combining the amount of antimony released from the battery during charging every 2.5 days with the stirring up of antimony dust accumulated from long-term charging of an electric vehicle, it is reasonable to project that eventually levels of Sb_2O_3 in the garage would be maintained which are at or near the TLV for use of Sb_2O_3 (0.5 mg/m^3), even if only 5% of the stibine formed in the battery were released and accumulated as Sb_2O_3. The hazard associated with long-term exposure to Sb_2O_3 at levels close to the TLV for use of Sb_2O_3 should thus be evaluated.

In addition to the exposure of the general public to Sb_2O_3, occupational exposure would occur during the smelting of antimony ore (15,000 MT in 1974) and preparation of the grids during battery manufacture.

7.1.1 Metabolic Uptake, Retention and Excretion

Stibine

Stibine is a potent hemolytic agent; however, the literature on stibine contains little information on cases of human exposure. Only one incident was found--that of exposure to a mixture of arsine, stibine and hydrogen sulfide.[4,5]

Existing information on the uptake, retention, and excretion of antimony when administered as stibine comes from the work of Smith et al.,[6] who were investigating the potential use of stibine in the treatment of tropical diseases. They administered Sb-122, Sb-124 labelled stibine to chickens and guinea pigs via inhalation at a concentration of approximately 25 ppm for 50 minutes. The results showed that stibine was concentrated in the red blood cells and produced a significant degree of hemolysis. At 15 minutes after exposure of guinea pigs to stibine, the ratio of antimony concentration in red blood cells to that in plasma was approximately 150. The liver quickly concentrated and retained antimony at levels higher than other tissues analyzed. At eight hours after exposure of chicks to stibine, antimony concentrations in liver, spleen, heart, kidney, lung, blood and brain were 24, 5, 4, 3, 3, 3 and 0.1 µg/g, respectively. By eight hours, tissue antimony concentrations were relatively stable, and were maintained to 24 hours.

A species difference in the rate of metabolism of stibine was observed. The initial concentration of antimony in the blood of chicks was about 10 times that of guinea pigs; the initial rate of disappearance of antimony from chick blood was about one-half that from guinea pig blood. Levels of antimony in tissues other than blood were also considerably higher in the chick than the guinea pig; relative distributions in the various organs were similar, however.

Excretion of antimony into bile and urine was measured. In the guinea pig the maximum antimony concentration in liver was found at one half hour after exposure and the maximum concentration in bile at two hours. Urinary concentrations reached a peak at two hours also. Calculations of total antimony excretion provided a theoretical biological half-life of approximately 45 minutes when antimony was administered as stibine to the guinea pig. Chicks required two to five hours to reach the same degree of elimination.[6]

Antimony Trioxide

Antimony trioxide is a white crystalline powder, insoluble in water. Review of the literature revealed only four articles on the metabolism of antimony oxides.[7-10] The most complete information on antimony metabolism following exposure by inhalation to an insoluble oxide comes from two articles reporting experiments using Sb-124.[7,8] In these experiments, mice and dogs were exposed to aerosols of Sb-124-tartrate heated to 1100°C prior to exposure. (Oxidative degradation of the Sb-124-tartrate complex at this temperature might be expected to produce an aerosol of Sb_2O_3, as the heating of antimony metal above 700°C in the presence of large quantities of air produces Sb_2O_3.[11]) The aerosol had a particle size of 0.3 ± 1.3 μm activity mean aerodynamic diameter (AMAD; ± geometric standard deviation). Mice were exposed to the aerosol for 10 minutes.[7] Whole-body retention and organ distributions of Sb-124 were determined from 0 to 50 days after exposure. Following inhalation exposure, antimony was retained in the mouse with a half-life of 39 days. The skeleton, lung and liver contained the greatest percent of the initial body burden (adjusted by subtraction of the antimony in the pelt and GI tract from whole-body antimony). These organs accounted for approximately 40%, 25%, and 20%, respectively, of the adjusted body burden at zero time. The lung and skeleton lost antimony at a rate similar to that of the whole body (half-life of 39 days), maintaining 40% to 50% and 20% to 30% of the residual body burden, respectively, to 50 days after exposure. The liver antimony content decreased faster than that of the whole body, and by 50 days contained only 2% to 3% of the residual body burden. These data indicate that, in the mouse, substantial absorption and translocation of antimony from the lungs to internal organs takes place, with liver and skeleton accounting for most of the translocated material.

In a similar set of experiments with dogs,[8] three beagles were exposed to an Sb-124 aerosol heated to 1000°C, and having an AMAD the same as that described above for the mice. At zero time, 70% of the initial body burden was estimated to be in the lung, with 3% remaining there at 32 days. The Sb-124 was eliminated from the dogs with a 45-day half-life. At 32 days, the pelt, lung, liver, skeleton and thyroid of one of the dogs contained 8.6, 3.2, 1.3, 1.1, and 0.3%, respectively, of the initial body burden. The thyroid and lung had by far the highest concentration of antimony, with 0.5% and 0.3% of the initial body burden per gram. The above organs all lost antimony with time, contributing to the 45-day half-life observed for the whole body. Excretion of antimony in these dogs was close to equal in urine and feces; a mean urine-to-feces ratio of 0.81 was observed from days 2 through 32 after exposure.

In another study[9] of Sb_2O_3 metabolism, rats were fed a diet containing 2% Sb_2O_3 for 8 months and then shifted to an Sb_2O_3-free diet. Animals were killed immediately or at 40 days after the diet switch, during which time excretion of antimony into urine and feces was measured. Excretion into feces was high just after the switch, but levels in the feces fell rapidly with time compared to those in urine. After 20 days after the diet switch, excretion of antimony into urine was greater than that in feces, and the ratio of antimony in urine to that in feces continued to increase with time. The order of antimony concentrations in tissues were thyroid > liver > spleen > kidney > heart > lungs > bone > muscle. In addition, after 40 days, the concentration in thyroid and liver were 48% and 60% of the initial levels, respectively. All tissues lost antimony with time at a similar rate except muscle, in which the concentration increased slightly by 40 days. Liver and bone contained the greatest percent of the body burden, as had been found in the inhalation exposure experiments presented above.

In summary, antimony trioxide appears to be eliminated from mice and dogs with a fairly short half-life (40-45 days) following inhalation exposure. It is translocated from the lung, principally to liver and skeleton. In addition, it appears in high concentrations in the thyroid. Excretion occurs through both urine and feces, with the urinary route predominating after termination of exposure to dietary Sb_2O_3.

7.1.2 Toxic Responses and Human Health Implications

Stibine

The early work of Stock and Guttman[12] demonstrated the acute toxicity of stibine in mice. In humans, death can occur within a few hours at stibine levels of 100 ppm in air; rapid death follows exposure to 10,000 ppm. Because of contamination by arsine, hydrogen sulfide, and other toxic agents, stibine has not been conclusively proven to be the toxic agent in any of the reported poisonings.[13] The TLV for stibine is 0.1 ppm, compared to 0.05 ppm for arsine.[14] Reports of low-level exposure to stibine at concentrations approaching the TLV were not found in the literature, rather, only three very early reports on toxic responses in animals to higher levels of exposure.[3,6,15] More information is available on arsine toxicity, and the TLV for stibine[14] has apparently been set based on analogy with arsine. (Arsine is discussed in Sec. 7.2.)

Blood. Stibine, like arsine, is a strong hemolytic agent. Exposure to stibine at 25 ppm for approximately 50 minutes reduced the hematocrit of guinea pigs from approximately 50% to 20% in the first hour after exposure.[6] In a second study, guinea pigs exposed to 65 ppm for 1 hour showed changes in the morphology of their erythrocytes that preceded hemolysis.[3] Spherical erythrocytes having tiny spicules extending outward on the surface of the cell appeared within minutes after exposure. Webster[1] named these cells "spine cells." At this exposure level hemoglobinuria was seen within 10 to 20 hours. The urine became clear within the next few days. At the same time, anemia developed. Red blood cell counts dropped to $1.7 \times 10^6/mm^3$ (normal range 4.5 to $6.8 \times 10^6/mm^3$) before starting an upward trend.

Renal System. Hemoglobin casts and crystalline material were found to precipitate in the kidneys of guinea pigs exposed to sublethal doses of stibine.[1] Frequently oliguria or anuria were seen to occur also. These observed effects of stibine on the kidney are similar to the effects of arsine and suggest that acute kidney damage is also a sequel to exposure to toxic levels of stibine.

Respiratory System. Exposure of cats and dogs to stibine at 40 to 45 ppm for one hour showed that pulmonary congestion and edema were visible in acutely poisoned animals.[6] The histopathology of the lung was not discussed in this investigation.

Other Systems. Jaundice, fatty metamorphosis of the liver, and splenomegaly were reported to occur in response to stibine exposure in guinea pigs and rabbits.[1] Further details regarding these responses were not given.

In summary, very little detailed information is available on toxic responses to stibine. Analogy to arsine must be relied upon as a means of estimating toxic responses anticipated following exposure to stibine.

Antimony Trioxide

Bradley and Frederick[16] in 1941 established in rats and guinea pigs the median lethal intraperitoneal doses for various antimony compounds. As a cause of death, antimony metal was ten times more toxic than antimony trisulfide (Sb_2S_3), and Sb_2S_3 was three times more toxic than Sb_2O_3. Death was in all cases attributed to myocardial failure. Degenerative changes in the liver and kidney were also observed.

In other animal experiments with inhalation as the exposure mode, responses were measured in rats, rabbits and guinea pigs exposed to Sb_2O_3 at levels from 45 to 125 mg/m^3.[11,17] Changes in lung, blood cells, liver and spleen were observed[11] in guinea pigs exposed to Sb_2O_3 at 45 mg/m^3 for two to three hours daily for 8 to 265 days. Subpleural petechial hemorrhages of the lungs were seen in all animals exposed for more than 30 days. Pneumonitis was more extensive in exposed guinea pigs, and pneumonia was observed in treated but not in control animals. Of those animals exposed for more than 45 days, 73% showed fatty degeneration of the liver. Fifty percent of the exposed animals showed hyperplasia of the lymph follicles of the spleen with a decrease in polymorphonuclear leucocytes. Blood cell changes included decreases in white blood cells, polymorphonuclear leucocytes, and eosinophils, with increases in lymphocytes. No microscopic pathological changes in heart or kidney were observed; EKGs run on three treated pigs were normal. Gross et al.[17] exposed rats and rabbits to Sb_2O_3 at 90 to 125 mg/m^3 for three hours per day for various times to look for lung changes. Chalk white foci which stained with the lipid stain Sudan IV were seen in the lungs starting at the 5th month of exposure in rabbits and 9th month in rats. In both species, pneumonitis secondary to lipid accumulation was the salient feature observed.

Exposure of animals to lower levels of Sb_2O_3 by inhalation have not been reported. Brieger et al.,[18] however, saw changes in the EKGs and degenerative changes in the heart of rats and rabbits exposed for only 6 weeks to the more toxic Sb_2S_3 at 3 to 5 mg/m^3. In summary, toxic responses in animals following inhalation exposure to high levels of Sb_2O_3 include changes in lungs, liver, spleen and blood cells. The levels studied are 4 to 10 times those estimated to exist in a garage during cleaning operations at the end of one year of charging, and are 100 to 200 times the TLV for use of Sb_2O_3 (0.5 mg/m^3).

Additional information on the toxicity of Sb_2O_3 must be obtained from studies of persons occupationally exposed to antimony. In general, responses to antimony among typesetters,[19] textile workers,[20] antimony smelter workers,[21,22] and antimony oxide sackers[23] include irritation of the skin, producing a pustular dermatitis; of the gastrointestinal tract, resulting in vomiting and diarrhea; and of the respiratory tract, producing nose bleeds, laryngitis, rhinitis, tracheitis, and pneumoconiosis. Blood cell changes, such as anemia and decreased leucocyte counts, have also been observed. Among persons industrially exposed to Sb_2S_3, changes in EKGs have been seen in 49% of those studied, and heart disease was suspected as the cause of death in the case of eight persons exposed to Sb_2S_3.[18] Air levels of Sb reported

in the occupational settings include 5 to 10 mg/m^3 in the breathing zone of antimony smelter workers,[22] 138 mg/m^3 at the site of an antimony bagging operation,[24] and 0.58 to 5.5 mg/m^3 for the exposure to Sb_2S_3 reported above.[18]

Considerable information is available on antimony exposure levels and related pneumoconiosis. At a French antimony smelter pneumoconiosis with and without associated pulmonary dysfunction was experienced among 15 to 17% of the workers whose duration of exposure ranged from 6 to 40 years.[10] The antimony ore also contained 1% to 20% silica. In that area of the plant where the oxide was produced, the antimony concentration averaged 0.30 mg/m^3, with many particles $\leq$ 3 µm. When the ventilation system was not working, antimony levels in the roasting furnace area ranged from 3.4 to 14.7 mg/m^3. Antimony was considered to be the principal causative agent, but sulfur dioxide and silica dust exposure could not be disassociated from the effects.

British workers in a plant processing antimony oxide and antimony metal from sulfide ore had a pneumoconiosis incidence of 9%.[10] Antimony levels in the urine of three people who were examined ranged from 425 to 680 µg/L. The pneumoconiosis was symptomless, but skin eruptions typical of antimony exposure did occur during warm weather. Atmospheric antimony levels in work areas ranged from 0.53 to 5.34 mg/m^3, and most of the ten sample sites had concentrations greater than 2 mg/m^3. One short-duration operation had concentrations up to 36.7 mg/m^3. When this plant was reexamined six years later, the pneumoconiosis incidence had increased to 18%.

Antimony-induced pneumoconiosis was found in 3 of 28 American workers, and suspicious chest x-rays were found in an additional five in one smelter.[24] The duration of exposure ranged from 1 to 15 years. Antimony concentrations throughout the smelter plant ranged from 0.081 to 138 mg/m^3. Urinary antimony was measured six times over a period of five years and found to range from 0 to 1020 µg/L. Among the current eight workers, urinary antimony ranged from 0 to 735 µg/L, averaging 202 µg/L. Urinary antimony as high as 1 mg/L was considered safe because of the insolubility of the dust and oxide that were the forms of antimony exposure in this plant.

When x-ray spectrophotometry was used on 113 men exposed to antimony, the amount found in the lungs ranged from a trace to 11 mg/cm^2.[25] Further, there was a significant relationship between the lung antimony concentration and length of employment, especially when the first 20 years of employment were considered ($r = 0.703$).

Workers in a Yugoslavian plant exposed to antimony oxides (Sb_2O_3 and Sb_2O_5) developed a slower onset pneumoconiosis with different symptoms from those previously described.[10] Many complained of difficult breathing, mylagia, cough and dyspeptic discomfort. Of 62 examined, 22 had emphysema with bronchitis, eight were less than 40 years old. The workplace dust antimony concentration ranged from 16 to 248 mg/m^3, with most particles < 0.5 µm.

Without a more definitive measure of antimony exposure, it is difficult to reach a conclusion as to the toxicity of inhaled antimony. Air levels as low as an average 0.3 mg/m^3 (in the presence of silica dust) have resulted in health problems, while others as high as 138 mg/m^3 (range: 0.08-138 mg/m^3) appear to have been better tolerated. Regardless of workroom air levels, past experience with antimony smelter workers has been a pneumoconiosis incidence rate of about 10-15%. Certainly particle size has an important influence on the delivery of antimony to the deep lung, as indicated by the Yugoslavian experience. Here the emphysema rate was about

35% and the recorded particle size the smallest, most particles being less than 0.5 μm. Given past experience, one could expect pneumoconiosis rates to at least continue at present (10-15%) levels. However, more definitive exposure and dose data are needed to better assess the effects of antimony exposure. In addition, the long-term health consequences of antimony pneumoconiosis need to be determined.

The effect of Sb_2O_3 exposure in the garage due to charging of lead/acid EV batteries needs to be considered in light of the metabolic and toxicity information presented. Sporadic exposure (at garage cleaning time) to levels of Sb_2O_3 of 5 to 10 mg Sb/m^3 following uniform dispersal of settled dust from one year of charging were estimated in the introduction. In addition, garage levels maintained close to the TLV for Sb_2O_3 use (0.5 mg/m^3) might be reasonable due to buildup of dust from more than one year of charging along with the continuous emissions from charging every 2.4 days. If the lead/acid battery with an antimony electrode additive is commercialized for EV use, clearly better measures of antimony emissions during charging will have to be obtained. In addition, information on the effects of daily exposure of animals of various ages to levels of Sb_2O_3 near the TLV should be examined. Concentration of antimony in the thyroid of young children following exposure to Sb_2O_3 might be a point of concern requiring further investigation.

Estimation of the current impact on the occupational sector is difficult because there is no current information on in-plant antimony levels or on the size of the worker population involved. However, based on previous industry experiences, one could expect an incidence of benign pneumoconiosis approaching 15%, even with relatively low work-area antimony concentrations (0.5 to 5 mg/m^3).

The primary rate of antimony exposure for the general public is via food. In regards to the EV battery cycle, contamination of food and drinking water by dust, process waste waters or slag pile or sludge pond leaching into groundwater could be possible in local areas. By the year 2000, these slag piles could contain about 300 MT of antimony as slag and about 9 MT of antimony as sludge, based on a production of 75,000 MWh by 2000. The possible effect of this additional antimony in local foods and water is unknown as there is a dearth of information on current intake levels. One estimate is that daily food intake of antimony varies from 250 to 1250 μg/day.[26] The antimony content of drinking water varies widely from place to place. The U.S. Environmental Protection Agency has not set a drinking water standard for antimony but others have recommended a limit of 0.05 ppm.[10] Thus, without any baseline information on the effects of low-level antimony exposure via ingestion, it is not possible at this time to speculate as to the health effects of any additional antimony in local area food and water.

7.1.3 Protection, Remediation, and Diagnostic Techniques

Stibine

Knowledge of those conditions likely to produce toxic levels of stibine should help identify and prevent accidental exposures to this toxic gas. Wherever possible, adequate ventilation of areas where stibine will or can be produced should reduce the probability of an accidental exposure. Short and Wheatley[27] designed and built a portable prototype instrument for the detection of stibine in air. The use of this or a similar monitoring instrument in areas where batteries are being charged would be valuable in identifying potential stibine sources.

Because of the similar actions (hemolysis and acute renal damage) of the two gases, therapy for an acute exposure to stibine would most likely be the same as that for arsine, i.e., (1) exchange transfusions using whole blood and (2) hemodialysis and/or peritoneal dialysis if renal shutdown develops.

Antimony Trioxide

Adequate ventilation of garage areas during charging could prevent buildup of Sb_2O_3 levels. In addition, design of the lead/acid battery to provide for containment of emission gases or elimination of antimony from the electrodes could end the problem of antimony release.

Treatment for the occupationally exposed has involved removal from exposure, treatment of pneumonitis with penicillin aerosols, and use of penicillin preparations for relief from laryngitis.[22] Diagnosis of the extent of occupational exposure has been provided by measurements of urinary and fecal antimony levels,[18,22,24] with levels of 1 mg/L urine suggested as a safe level.[18]

7.2 ARSENIC AND THE GAS ARSINE

Arsenic is a contaminant of both lead and zinc ores, the primary metals used in the lead/acid and Ni/Zn batteries. Arsenic is also used as an alloying agent in the lead/acid battery grid. Arsenic air emissions from lead smelting have been reported to be 0.8 lb As/ton Pb (0.4 kg/MT) and 1.3 lb As/ton of zinc smelted (0.65 kg/MT).[28] These values can be used to estimate the arsenic emissions anticipated from primary lead and zinc smelting, as outlined in Appendices E and F. By a conservative estimate, the arsenic emissions related to zinc smelting for EV batteries could increase 20-fold over the 1977 levels by the year 2000 (Table 7.2). Arsenic emission rates for the primary lead industry could increase nearly seven-fold. Secondary lead smelting would contribute an additional 112 to 2660 kg of arsenic as air emissions based on data shown in Table 2.22 and the total MWh output for EVs produced by 2000. There are six zinc and lead smelters in the United States, thus localizing the arsenic deposition. In addition to smelter workers, workers in lead/acid battery manufacturing industries could be exposed to 0.0085 kg arsenic per day per megawatt hour of batteries produced, or a total of 11 MT of arsenic by the year 2000. In a large battery manufacturing plant producing 6500 batteries per day, the total arsenic handled per day would be about 1.4 kg in the year 2000.

Secondly, low-level ingestion of arsenic over a period of many years could be a health concern. Based on the available data (Table 7.3) it is estimated that by the year 2000, lead/acid battery manufacture for EVs (total process cycle) will contribute about 1.05×10^{-5} kg As/day per MWh in liquid wastes and 5.08×10^{-6} kg As/day per MWh in solid wastes such as sludges and slurries. The total for the year 2000 would be on the order of 287 kg of arsenic in liquid wastes (1.05×10^{-5} kg/day per MWh × 365 days/yr × 75,000 MWh in the year 2000). The total arsenic in slurries and sludges would be about 139.1 kg in the year 2000. Zinc smelting would add another 139 kg (Table 3.4; 1.9×10^{-6} kg/day per MWh × 365 days/yr × 200,000 MWh in the year 2000). Public exposure could result from contaminated air and water near mining and smelting sites and the decomposition of arsine to As_2O_3 during battery charging.

Because maximum levels of stibine anticipated in a garage after charging range from 1 to 7 times the TLV for stibine (see Sec. 7.1), an analysis of the data available on arsine toxicity,

Table 7.2. Projected Atmospheric Arsenic Associated with Primary Smelting of Metals for EV Batteries

	Arsenic, kg/yr[a]	
Year	Zn	Pb[b]
1977[c]	733	600
1985[d]	<1-134	N.A.-106
1990[d]	3-645	N.A.-362
2000[d]	52-12,600	N.A.-4340

[a] *Based on Secton 2.2, Tables 3.5 and 3.7, and Appendix F.*

[b] *Insufficient data for derivation of lower estimate.*

[c] *Total U.S. smelter production for 1978 was 1128.4 MT Zn and 1503.7 MT Pb (Statistical Abstracts of the United States, 1978, Tables 1344, 1349). Arsenic emission factors were 0.65 mg/MT Zn and 0.40 kg/MT Pb ("Arsenic," Committee on Medical and Biological Effects of Environmental Pollutants, National Academy of Sciences, Washington, D.C. 1977, p. 73).*

[d] *Cumulative total emissions.*

Table 7.3. Predicted Arsenic Content in Liquid and Solid Wastes (kg/day)/MWh from Various Stages of the Lead/Acid Battery Cycle

	(kg As/day)/MWh[a]	
Component	Liquid Wastes	Solid Wastes
Primary Lead Smelting	1.8×10^{-7}	N.A.
Secondary Lead Smelting	U	6.1×10^{7}
Battery Manufacture	1.0×10^{-7}	$1.9 \times 10^{-6} - 3.5 \times 10^{-6}$
Battery Breaking	$9.6 \times 10^{-8} - 4.8 \times 10^{-6}$	$6.2 \times 10^{-6} - 1.1 \times 10^{-5}$
Antimony Smelting	N.A.	1.8×10^{-7}
TOTAL (conservative)	1.05×10^{-5}	5.08×10^{-6}

[a] *For source of values shown, see Tables 2.18, 2.11, 2.24, 2.26, and 2.28.*

which by analogy form the basis for the TLV for stibine, is also relevant. Maximum levels of arsine anticipated in a garage at the end of a single charge cycle would not exceed one-tenth the present TLV[4] for arsine of 0.2 mg/m^3 (0.05 ppm).

For the occupationally exposed, direct contact and inhalation are the primary routes of arsenic uptake. Ingestion is the major route of uptake for the general public.

7.2.1 Metabolic Uptake, Retention and Excretion

Arsenic

Absorption Through Inhalation. Arsenic exposure through inhalation usually involves arsenic oxide, particulary arsenic trioxide (As_2O_3), among workers in the smelting industries. Deposition in the respiratory tract would be expected to range from 30% to 60% depending on particle size, rate of respiration, and other factors already discussed with respect to inhalation of lead particles (Sec. 7.5.1).

Absorption Through Ingestion. Absorption of arsenic through food ingestion is apparently influenced by the chemical form of the arsenical involved and by the dietary composition. Coulson et al.[29] fed rats either As_2O_3 or arsenic bound to shrimp tissue. About 18% of the administered As_2O_3 was retained by the rats during the first three months of feeding, indicating substantial gastrointestinal absorption. Only 0.7% of the arsenic bound to shrimp tissue was retained during the same period. Morgareidge[30] found that approximately 15% of the arsenic fed to rats in the form of either As_2O_3, or an organoarsenical in turkey livers, was retained. Tamura[31] reported that a powdered milk diet reduced the absorption and retention of arsenite by rats to zero as compared with animals on a cereal diet in which at least 50% of the arsenic fed to the animals was absorbed.

Tissue Distribution. After absorption by the lungs or by the gastrointestinal tract, arsenic is transported by way of the blood to other parts of the body. Hunter et al.[32] found arsenic in the liver, kidney, lungs, spleen and skin during the first 24 hours after oral or parenteral administration of As-74 potassium arsenite to various species including rats, rabbits and guinea pigs. The highest concentrations were found in liver, spleen, kidney and bone marrow. Bone and muscle concentrations were low; because of their large mass, however, these tissues, along with skin, contained most of the arsenic in the body. The rat differed from other species with respect to distribution of As-76-sodium arsenite in the blood.[33,34] The rat stored 79% of the administered arsenic in the hemoglobin of the red blood cells after 48 hours, while less than 0.27% was stored in the organs studied in the dog, cat, chick, guinea pig and rabbit. From the work of Peoples[35] on the distribution of arsenic in rats, guinea pigs, rabbits and hamsters fed arsenic trioxide, it appears that the hamster or rabbit is a better animal-model for man than the rat.

Excretion. Arsenic is excreted mainly in the urine. Excretion was nearly complete by six days after injection of As-74-potassium arsenite into various species other than rat.[32] Only a trace appeared in the feces. Excretion of arsenite in rats was found to be much slower, most likely because of binding by the hemoglobin of the red blood cell. In man, 40% to 50% of an injected dose of As-74-arsenite was excreted in urine by 96 hours.[33] Studies with cows and dogs have shown that arsenates and arsenites are methylated prior to excretion in the urine.[36]

Arsine

Arsine is an extremely poisonous gas whose toxicity has been recognized since the early 1800s. Arsine is a moderately stable, colorless, water-soluble, inflammable gas, denser than

air, with a very strong reducing potential. Arsine is generated by (1) the reaction of water or acid on metallic arsenides, (2) the combination of nascent hydrogen with soluble arsenic, or (3) charging of lead/acid batteries containing arsenic as a component or impurity.

Existing data on the metabolic uptake, retention and excretion of arsine and its oxidation products are scarce. In one early investigation, mice and rabbits in carefully designed exposure chambers were exposed to arsine concentrations ranging from 25 to 2500 mg/m^3.[37] Animals receiving median lethal exposures (exposure times required to produce 50% mortality) deposited more arsenic in total when exposed to lower concentrations than to higher concentrations of arsine. Analysis of tissue revealed that, at the lowest exposure level (25 mg/m^3), the concentration of arsine resulting in 50% mortality was 14.8 mg AsH_3/kg body weight, while at the highest exposure level, this concentration was only 0.67 mg AsH_3. The animals thus had a way of handling exposure to 25 mg AsH_3/m^3, making it less toxic than 2500 mg/m^3. The relevance of this finding to the toxicity of arsine at levels close to the TLV (0.2 mg/m^3) is not clear.

Measurements of whole-body arsenic levels in this study allowed calculation of the fraction of inhaled arsine that was retained by the body. The mean value obtained for the given range of arsine concentrations was 0.64. In addition, the elimination of arsine after exposure of mice to 180 mg AsH_3/m^3 for 20 minutes was such that, at 24 hours after exposure, 45% of the initially retained arsenic was still present[37] and the excretion rate of this retained fraction was slow.

Tissue retention of arsenic prior to hemolysis was also determined in rabbits following arsine exposure.[37] Tissues were washed in ice-cold saline after exsanguination of the rabbits to reduce the amount of arsenic contributed by blood. The relative concentrations in the organs analysed were: liver > kidney = heart > intestine > muscle > brain. The authors note[37] that the arsenic concentrations found in any one tissue did not parallel the blood content, indicating that the presence of arsenic was not due solely to arsenic associated with erythrocytes; rather, tissue arsenic levels correlated well with the product of gas concentration and the length of exposure.

Other data on the tissue retention of arsenic comes from the analysis of human tissues taken at autopsy of persons who died from exposure to an undetermined amount of arsine. For one autopsy[36] the organs showing the highest arsenic concentrations were liver > spleen > kidney > pancreas; in another, the lungs were also high in arsenic.

Excretion of arsenic into the urine can be used as an aid to diagnosis of arsine poisoning but does not provide a consistent indication of the degree of exposure[38] or the degree of hemolysis that follows.[39]

7.2.2 Toxic Responses and Human Health Implications

Arsenic

Toxic response to arsenic inhalation has been discussed in several review papers dealing with occupational exposure.[28,40,41] The symptoms of subacute and acute arsenic exposure are similar: dyspnea, headache, extreme general weakness, nausea and vomiting, diarrhea, body pains, skin and mucous membrane irritation.[28,41] Prolonged occupational exposure can result in brittle hair and loss of hair, eczema, hyperpigmentation, hyperkeratosis of the palms and soles,

mucous membrane lesions, and peripheral neuritis.[28,40,41] Circulatory effects, cirrhosis of the liver, lung and skin cancers[40] and cardiovascular disease[42] have also been reported, but in fewer cases.

Direct contact with arsenic trioxide (occupational exposure) has a local effect on skin, mucous membranes and conjuctiva where the dust settles. Hoarse voice, inflammation of the pharynx and larynx, a sore tongue, excessive salivation, and perforation of the nasal septum may occur.[43] The skin lesions can ulcerate or thicken and become pigmented, slowly developing into a cancerous lesion.[44] Protective clothing and face masks are the best means to prevent exposure.

Ingestion of arsenic can result in hyperkeratosis of the palms and soles, skin cancer and vascular disorders of the lower extremities, leading to gangrene.[41] Other symptoms include hyperpigmentation, gastrointestinal tract disorders, and neurologic symptoms (paresthesias). There is also evidence that arsenic depresses the immune system[28] and may be mutagenic.[28,45,46]

In the general environment, arsenic levels in ambient air in the early 1960s were <0.02 $\mu g/m^3$ in rural areas and about 0.1 $\mu g/m^3$ in larger cities. These values are put into perspective when compared with occupational levels: at 10 to 40 mg/m^3 only slight symptoms are usually seen after several hours' exposure, and a maximum of 25 to 125 mg As/m^3 can be tolerated for one hour by some without serious effects; however, this latter exposure level overlaps with the 50 to 250 mg/m^3 that has been associated with serious effects after one hour's exposure.[47]

Inhalation Exposure. Good human exposure-response data on inhaled arsenic are limited. Dermatitis was associated[40] with urinary arsenic levels of 0.82 mg/L in a group of gold smelter workers. Dermatitis was also seen in 80% of the workers in a copper smelter who had 1.0 to 2.0 mg As/L in their urine. All workers with urinary arsenic levels greater than 3.0 mg As/L urine had dermatitis.

The prevalence of hyperpigmentation[28] seems to increase monotonically with urinary arsenic levels. Of the chemical workers in a sheep-dip factory who were exposed to an average of 0.562 mg As/m^3 air, 90% showed hyperpigmentation.[40]

Arsenic has been suggested to have a role in the etiology of lung cancer. Smelter worker studies have found lung cancer and arsenic associations[48] and two[49,50] studies found respiratory cancer death rates to increase with length of exposure. Pinto and coworkers[49] were able to eliminate smoking habits as the cause of the respiratory cancer death differential. Mortality increased significantly with the calculated average intensity of arsenic exposure (measured as $\mu g/L$ urine) and duration of exposure--see Table 7.4. In addition, the number of person-years lived decreases with intensity of arsenic exposure. A threshold effect was suggested by the superior survival experience of men with low exposure levels and less than 25 years' exposure. Because other contaminants were present in the atmosphere of both study smelters, in neither study could it be certain that arsenic was the only agent responsible for the increased lung cancer mortality.

The evidence cited above suggests that airborne arsenic has a role in the development of human lung cancer. It must be noted, however, that this hypothesis has come under attack on several occasions, largely because there are no animal models for arsenic-induced lung cancer[51] and the presence of other contaminants.

Table 7.4. Standardized Mortality Ratios (SMR) for Respiratory Cancer Deaths in Copper Smelter Workers by Intensity and Duration of Exposure

Arsenic in Urine, µg/L	*Duration of Exposure*	
	< 25 yrs, SMR	*≥ 25 yrs, SMR*
50-199	95.2	277.8[a]
200-349	266.7	363.6[a]
350+	600.0[a]	833.3[a]

Source: S. V. Pinto, V. Henderson and P. E. Enterline, "Mortality Experience of Arsenic-Exposed Workers," Arch. Environ. Health 29: 325-331, 1978, Table 5. In control group, SMR = 100.

[a] *$p < 0.05$.*

Because of this latter problem, a recent study[52] of a Swedish copper smelter included other contaminants in the analysis. Using the case-control method, the authors found that with arsenic exposure, lung cancer mortality increased nearly five-fold, cardiovascular (CVD) mortality increased two-fold and the cerebrovascular (CBVD) mortality increased about 60% over controls. Further, the effect seemed to increase with the degree of arsenic exposure. Although the effects of sulfur dioxide, lead, copper, nickel, selenium, bismuth and antimony were also considered, the risks of mortality from lung cancer, CVD and CBVD were consistently greater with arsenic exposure. This study confirms the previously noted effect on cardiovascular disease mortality[50] and contains the strongest data in support of arsenic as an etiologic agent in lung cancer. The small size of the study (251 cases, 74 controls) precludes any development of a dose-response equation, but there is a definite suggestion of lung cancer and CVD mortality increasing monotonically with the degree of arsenic exposure.

A recent study[53] of Japanese copper refinery workers found subclinical responses to low-level, long-term arsenic exposure. Workers and controls were matched for age, sex and length of employment. The arsenic exposure ranged from 2.64 µg/m^3 to 12.22 µg/m^3, with an average exposure period of 20.6 years. Urinary arsenic levels in the two exposed groups averaged 82.6 ± 49.6 µg As/L and 40.6 ± 3.16 µg As/L. Compared with the control group, the group with the highest average arsenic exposure had significantly higher levels ($p < 0.05$) of the enzymes glutamic oxylacetic transaminase (GOT) and lactic dehydrogenase (LDH). These differences, though still within normal limits, seemed to indicate a slight change in liver function. LDH was also found to be elevated in 6.1% of employees in a U.S. arsenic smelter,[44] but was not correlated with elevated serum GOT levels. Average exposure and duration of exposure were not available for this group of workers; however, it was noted that in the United States, the average urinary arsenic level is 30 µg/L, with a range of 20 to 60 µg/L for persons with no exceptional exposure to arsenic. More research on the effects of low-level, long-term arsenic exposure appears to be necessary.

Related to this is the finding of decreased survival in retired chemical workers engaged at one time in the production of arsenicals. The retired worker group was similar to the controls

in median age at death and percent surviving past 70 years of age, but survival past 75 years of age was one-fourth to one-sixth that of controls.[54]

The importance of public exposure to arsenic from smelting and refining processes must not be overlooked. In a study of one- to five-year-old children living near primary copper smelters, Baker and coworkers[55] found elevated levels of arsenic in the hair and urine. Children in copper smelting towns had significantly higher hair arsenic levels than children in three comparison towns with no exposure. The average hair arsenic level in the exposed children was 2.60 μg/g; in unexposed children, 0.09 μg/g. Urine arsenic levels averaged 18.7 μg/L in the exposed and 5.8 μg/L in the unexposed children. In one town (Anaconda, Montana), the exposed children had urine arsenic levels 200-fold greater than the control children. In another smelter town hair levels of arsenic decreased with distance from the smelter. Differences in the amount of pica did not appear to be a factor; thus, inhalation was probably a major route of exposure. There were no clinical manifestations of arsenic poisoning, and the reported levels were below those normally associated with arsenic poisoning.

In a study of cancer by county, Blot and Fraumeni[56] suggested that arsenic exposure from copper, zinc and lead smelters was responsible for the significantly higher lung cancer death rates seen in counties having those industries. This pattern was apparent despite control for intervening socioeconomic factors, and occupational exposure alone could not explain the entire differential.

Ingestion Exposure. Endemic arsenic poisoning from contaminated groundwater in Taiwan was linked to skin cancer at levels near 0.05 mg/L water.[57] For over 45 years this area had used arsenic-contaminated wells, with the arsenic level ranging from 0.01 to 1.82 mg/L. In a survey of more than 40,000 people, arsenical skin cancer was found at the rate of 10.6 per 1000, with all victims over 20 years of age. The prevalence increased with age, except for women older than 60 years, and was higher among men at all ages. The incidence of skin cancer in both sexes at all ages increased monotonically with arsenic exposure.

Other symptoms of arsenic poisoning were also present in this population: hyperpigmentation, 18.4%; keratosis, 7.1%; and Blackfoot disease (gangrene of the lower extremities), 0.9%. As with the arsenical skin cancer incidence, the prevalence of these symptoms also increased with age, except for women older than 70 years. Of the surveyed population, the youngest person with arsenical skin cancer was 24 years old; with keratosis, 4 years; with hyperpigmentation, 3 years. Thus, very few years of exposure at these levels were necessary for symptoms of arsenic poisoning to occur.

Good correlations were found between arsenic in well water (224 μg/L, average; < 1.0 → 2450, range) in Fairbanks, Alaska, and the arsenic content of urine in those who drank the water.[58] Comparisons based on the EPA standard of 50 μg/L showed that as the water arsenic increased above the standard, the urinary levels also increased (correlation coefficient = 0.58). Age, sex and length of residence did not significantly influence urine, hair or nail arsenic levels. However, only 15% of the population had lived in the area for more than 10 years. No clinical symptoms of arsenic poisoning (hyperpigmentation or keratosis) were observed, even among those with the highest exposure ($\geq$ 100 μg As/L well water; urine arsenic averaging 17.83 μg/100 ml).

In comparing their study to the study in Taiwan by Tseng et al.,[57] Harrington and coworkers noted an important mitigating factor and one that may also increase the risk of some to arsenic poisoning. In contrast to the Taiwanese, the Alaskan population was well nourished and of high socioeconomic class. Calabrese[59] has noted that treatment with ascorbic acid (Vitamin C) prevents the toxic effects of arsenic given as Fowler's solution (potassium arsenite). Thus, persons with inadequate vitamin C intake could be more susceptible to arsenic poisoning. In addition, arsenic ingestion increases iodine requirements.[42] Without extra iodine in the presence of arsenic, increased incidence of goiter and cretinism can occur.[60]

In a study of a patient taking Fowler's solution (8.8 mg As_2O_3) daily for 28 months,[28] it was possible to follow the onset of symptoms. Within 13 months, darkening of the nipples, increased freckling and gastrointestinal symptoms occurred. Redness and puffiness around the eyes and hyperkeratosis appeared by 1.5 years. At the end of two years, neurologic symptoms (paresthesias and weakness) were present.

With the exception of local areas, dietary intake of arsenic will probably not be a problem. The present water quality standard is 0.05 mg/L. This would have to increase more than 11-fold or the average arsenic intake in food[41] would have to increase about 3-fold to produce a situation in the United States similar to that in Taiwan.

Arsine

Many cases of human exposure to arsine following accidental industrial exposure have been documented.[61] The insidious nature of the gas is due to its hemolytic properties and lack of symptoms immediately following exposure. Headache, malaise, abdominal pains, chills, weakness, nausea, vomiting and passage of blood-colored urine appear 0.5 to 6 hours after exposure, depending on exposure severity.[39,62,63] Exposure to 250 ppm for 0.5 hour is considered lethal in humans;[63,64] lower concentrations produce hemolysis, anemia, hematuria, oliguria, anuria, jaundice and acute renal damage.[5,38,39,61-70] In one case of industrial exposure in which four men out of 13 died, later measurements of arsine levels produced under similar conditions showed that those receiving the highest exposure were breathing air containing 70 to 300 ppm arsine.[38,67] In most cases, however, determinations of arsine concentrations in the exposure situation were not made, limiting the availability of dose-response information.

Blood. Hemolytic anemia is the most striking and consistent effect of arsine exposure. Intravascular lysis of erythrocytes occurs 1 to 12 hours after inhalation of an acutely toxic dose. Hemoglobin concentrations of less than 10 g/100 ml are not unusual. Plasma hemoglobin values of 2 g/100 ml or more have been reported.[61] The mechanism by which arsine lyses red blood cells has not yet been clearly demonstrated.[61]

Low levels of arsine have been reported[71] to reduce hemoglobin levels in exposed individuals. Following installation of a ventilation system in a zinc smelter, the hemoglobin level of plant workers rose from 85% to 90% of normal over a 40-week period. It was concluded that chronic exposure to arsine levels as low as 0.05 ppm (average length of exposure was five years) was sufficient to reduce hemoglobin levels. Further, return to normal hemoglobin levels is slow, even after removal of exposure conditions.[80]

Renal System. Arsine-induced hemolysis is accompanied by the release of large amounts of hemoglobin into the blood. The kidneys handle this assault with varying degrees of efficiency. Some arsine-poisoned subjects begin to excrete blood-colored urine one to two hours after exposure.[38,62,64,66,69] These subjects recovered, with minimal impairment of renal function, if exposure was not severe; however, for subjects showing extensive hemolysis, kidney function was impaired, resulting in oliguria or anuria.[39,63,65,67] In a comprehensive study by Muehreke and Pirani,[63] five serial renal biopsy specimens from a subject suffering from arsine-induced anuria were studied by light and electron microscopy. They found acute tubular necrosis followed by rapid regeneration of the tubular cells. However, the regenerated tubular cells were simplified, with no brush border and a reduced number of organelles. The slow differentiation and maturation of the regenerated tubular cells correlated well with the prolonged period of anuria. Although permanent renal damage has not been identified in arsine poisoning, impairment of renal function (reduced qlomerular filtration) has lasted as long as 10 months after exposure.[69] The mechanism of arsine-induced anuria has three possible explanations: (1) The reduction in the number of red blood cells could result in insufficient oxygen reaching the kidney cells; (2) The arsenic-hemoglobin complexes released after lysis of the erythrocytes and precipitated in the tubular lumen could cause damage to the nephron;[63] (3) *In vitro* studies on the incubation of kidney tissue with arsine solutions have shown decreased respiration of arsine-exposed kidney tissue,[72] and any uncomplexed arsine dissolved in the blood could have a direct toxic effect on the kidneys by decreasing respiration.

Nervous System. Effects on the nervous system seem to be related to the severity of exposure. Those subjects severely exposed show symptoms of arsine encephalopathy, whereas those having moderate exposure show only symptoms of peripheral neuropathy.[39,66,70] The symptoms of arsine encephalopathy include extreme restlessness, memory loss, agitation and disorientation. Arsine encephalopathy is usually a transient effect, disappearing as the condition of the patient improves. The symptoms of peripheral neuropathy include numbness of the hands and feet, extreme muscle weakness, hypo- and hyperesthesias, generalized burning sensations and photophobia.[39] Peripheral neuropathy has lasted as long as six months after severe exposure to arsine.[66,70]

Cardiovascular System. There is disparity in the literature with regard to the effects of arsine on the heart. One group of investigators[73] presented evidence of significant changes in the electrocardiograms (EKGs) of patients suffering from an acute exposure to arsine which they interpreted as a primary response of the heart to the insult of arsine exposure. In a different investigation[64] involving chronic low-level exposure to arsine among workers employed in the cyanide extraction of gold, electrocardiographic changes were also observed, but these authors did not conclude that the changes were a primary response to arsine exposure. Other accidental exposures to arsine have brought about suspected changes in the EKGs of those exposed, but the authors concluded that the changes were identical to those found in hyperkalaemia, a secondary response arising from the acute kidney damage.[68] In addition, the EKGs of three patients severely exposed to arsine were normal, even though two had renal failure.[70] There thus appears to be insufficient evidence to support the view that there are typical changes in the EKGs brought about by exposure to arsine.

Other Systems. Some of the severely exposed subjects who later died showed a marked stimulation of the reticuloendothelial system.[68] Another characteristic feature of arsine exposure is a transient (1 to 2 day) rise in the number of circulating leucocytes (leucocytosis).[38,62] Evidence of bone marrow depression has been observed as indicated by a reduction in the number of circulating reticulocytes.[39,70] Whether these effects are a primary or secondary response to arsine exposure remains to be answered.

Animal Toxicity Data. To study the effects of chronic low-level exposure to arsine, Nau[5] exposed guinea pigs for one to three hours daily for periods up to 144 hours and at concentrations ranging from 0.5 ppm to 2.0 ppm. He reported his findings in a generalized, descriptive way, and included very little quantitative information on the toxic responses described. He indicated that low-level exposure to arsine produced:

- A rapid decrease in the erythrocyte count to a level near 80% of the original value. This new lower level was maintained during exposure.
- A rapid decline in hemoglobin levels paralleling the decrease in the red blood cell count.
- An increased fragility of the red blood cell membrane.
- A rapid and marked decrease in the white blood cell count.
- An increase in the polymorphonuclear eosinophil, monocyte and basophil relative counts.
- A positive direct van den Bergh (indicates presence of free biliruben).

The author concluded that even at such low-level exposures, arsine had a destructive action upon the blood.

Levvy[37] exposed mice to arsine concentrations ranging from 25 to 2500 mg/m^3 (6.3 to 625 ppm) for different lengths of time. He determined median lethal exposure times (time required to produce 50% mortality) for the various concentrations of arsine to which the mice were exposed. These times ranged from 0.4 minutes for the highest level to 24 hours for the lowest. All levels studied were thus acutely toxic. A study of the values for total arsine exposure indicates that animals had to be exposed to more arsine at the lower concentrations studied to produce 50% mortality than at higher levels, i.e., the product of arsine concentration (in mg/L) times median lethal exposure time (in minutes) was 36 at the lowest arsine concentration compared to 1 at the highest concentration.

In another study, Kensler et al.[74] exposed rabbits, dogs and monkeys to an atmosphere containing 450 mg/m^3 of arsine (112 ppm). Of the 140 rabbits exposed to this arsine concentration for 30 minutes, 125 (89%) died. Two monkeys were exposed to the same arsine concentration for 15 minutes. One monkey died within 24 hours of exposure, showing marked intravascular hemolysis and hematuria. The surviving monkey became acutely ill and was ataxic from the second to seventh day. Again, the arsine concentrations studied were high compared to the recommended TLV (0.2 mg/m^3; 0.05 ppm).

Responses to long-term, low-level exposures have not as yet been rigorously studied. Slight decreases in hemoglobin levels in the absence of other symptoms have, however, been observed in men exposed for six days to an atmosphere containing small amounts of arsine.[64] The arsine concentration was such that men exposed to this same atmosphere for eight months were not acutely ill; reduced hemoglobin and RBC levels, however, prompted treatment with blood transfusions. In another case already mentioned, where chronic low-level exposure to arsine was

identified, hemoglobin levels of workers gradually rose from 85% to about 90% of normal over a period of 40 weeks following installation of positive exhaust ventilation.[80] An effect of arsine on hemoglobin concentrations in response to prolonged exposure to unknown low levels of arsine has thus been indicated.

7.2.3 Protection, Remediation and Diagnostic Techniques

Arsenic

Prevention of exposure by use of gloves and respirators, and by ventilation of work areas, is important to protect workers from exposure to arsenic compounds. BAL (2, 3-dimercaptopropanol) has been used for the treatment of chronic arsenic poisoning, especially for cases of dermatosis.[75]

Arsenic exposure is generally measured as arsenic in urine. Data from Wagner and Weswig[76] show that total urinary arsenic per 24 hours generally increases with duration of exposure to dimethylarsenic acid, an organic arsenic compound. Five forestry workers were exposed to an average of 8.7 grams (dry weight) dimethylarsenic acid for an average of 23.7 hours per week for eight weeks. Data on their average 24-hour total urinary arsenic excretion can best be fitted to a quadratic equation:

$$y = 32.750 + 26.917x - 1.083x^2,$$

where:

- y = average 24-hour total urinary arsenic for the group,
- x = weeks of exposure, and
- R^2 = 0.83.

All workers were asymptomatic. Thus, for organic arsenic compounds at least, a direct relationship between exposure duration and urinary arsenic on a group basis can be derived.

Arsenic smelter workers also showed a fair correlation ($r = 0.528$) between airborne levels and urinary arsenic.[77] For this group, the urinary level = 0.309 x (airborne level), standard error = 19.9 and significance = 0.01.

Exposure of the general public to airborne arsenic has been shown to decrease with distance from the source. Downwind of a copper smelter, urinary arsenic levels were seen to decline from 0.3 ppm close to the smelter to 0.02 ppm at 2.0 to 2.4 miles away.[40] A similar response in urine and hair arsenic was seen in a study of 10-year-old boys living near a power plant burning coal high in arsenic.[78]

Thus the relationship between arsenic intake and urinary arsenic has led to its use as an indicator of industrial and environmental exposure. Appearance of skin lesions or perforation of the nasal septum are additional diagnostic indications of exposure to arsenic.[79]

Arsine

The treatment for severe acute arsine poisoning consists of (1) exchange transfusions with whole blood to replace the patient's lysed red blood cells and to remove the arsenic-hemoglobin complexes and "ghost" red blood cells, (2) hemodialysis and/or peritoneal dialysis, necessary if renal shutdown occurs.[63,65,68-70] BAL therapy has been tried in many exposure cases but has met with little or no success.[38,61,63] Animal experimentation has shown that BAL[74] and other thiols[37] are effective only when administered very early after exposure. Because the symptoms of arsine exposure are usually delayed, these agents are ineffective.

The literature on arsine contains very little information on prevention of or protection from exposure. A thorough understanding of the conditions that produce arsine should help locate and prevent any potential exposure.[68,73] Adequate ventilation of areas where arsine is or can be produced will also be helpful in reducing the number of exposures. As noted earlier, the acute effects of arsine poisoning are unlikely to occur with normal lead/acid battery charging procedures in the average home garage.

Diagnosis of acute toxicity from arsine should include identification of the clinical features described under toxicity, along with appearance of arsenic in urine. According to a report[49] on the clinical manifestations of a severe poisoning incident, on the day following exposure, four of the nearly 50 exposed men had urine arsenic levels of 0.85 to 3.08 μg/100 ml urine. Two days after exposure, eight men had urine arsenic levels ranging from 0.14 to 2.04 mg/100 ml urine, and a week after exposure, seven men had urine arsenic levels of 0.04 to 1.02 mg/100 ml urine. All required hospitalization; most were treated for anemia, and four died.

Some indication of the relationship between arsine exposure levels and human response is given in Table 7.5. Symptoms appear in persons having urine arsenic concentrations ranging from 0.20 mg/L to 220 mg/L 24 hours after exposure. Rapid death from acute exposure occurred with concentrations of 0.26 mg As/L urine and 0.434 mg As/L blood. Other patients with blood arsenic levels of 2.2 mg/L survived. Even the lowest levels of urine and blood arsenic are accompanied by nausea, vomiting and apparent hemolysis (as indicated by dark or red urine). Urine levels of 11 exposed men[69] ranged from 0.6 to 5.8 mg As_2O_3/L during the first few days following exposure. Nearly all had red or brown urine and a combination of two or more of the following symptoms: jaundice, fever, chills, backache, oliguria, nausea-vomiting-malaise, diarrhea, headache, dizziness, convulsions, paraesthesias and abdominal cramps. As is evident from the range of apparent exposures and the similarity of responses shown in Table 7.5, urine and blood arsenic levels do not appear to be good measures of actual arsine exposure.

7.3 CADMIUM

Cadmium is a contaminant of zinc ores at concentrations varying from 0.1% to 1%.[81] Zinc smelting produces a distillate that may contain as much as 20% cadmium concentrated from an ore containing only 0.2%-0.5% cadmium.[82,83] Cadmium can also be found as a lead refining by-product.[81] Any increases in the use of zinc or lead resulting from the production of lead/acid or Ni/Zn batteries will increase man's exposure to cadmium. In addition, if cadmium is used in place of cobalt as a nickel electrode additive for the Ni/Zn EV battery, as indicated by one developer,

Table 7.5. Arsine Exposure Levels and Associated Symptoms

Symptoms	Arsenic Concentration in Blood	Arsenic Concentration in Urine	Period between Determination and Exposure
Severe abdominal pain, backache, pigmented skin, dark red urine[a]		220 mg/L	24 hr
Anorexia, nausea, red urine[b]		1.6 mg/L	24 hr
Jaundice, headache, vomiting, nausea, anorexia[a]		130 mg/L	24 hr
Vomiting, tingling in extremities, death[a]		175 mg/L	24 hr
Vomiting, hemolysis[c]	4334 µg/L	260 µg/L	80 min
Nausea, vomiting, abdominal cramping, neuralgia, anuria[c]	0.66 mg/L		24 hr
Nausea, vomiting, abdominal cramping, anesthesia and neuralgia still present in feet after six months[d]	2.2 mg/L		24 hr
Nausea, vomiting, jaundice, skin bronzing[d]	0.25 mg/L	0.43 mg/L	24 hr
Acute hemolysis, jaundice, polyuria, anemia, oliguria[c]	0.7 mg/L		∿ 30 hr
Fever, abdominal cramps, jaundice, acute hemolysis[e]		3.45 mg/L	24 hr

[a] *Source: P.R. Udall et al., "Renal Damage from Arsine Poisoning," Brit. J. Indust. Med. 27:372-377, 1970.*

[b] *Source: G.C. Jenkins et al., "Arsine Poisoning: Massive Haemolysis with Minimal Impairment of Renal Function," Brit. Med. J. 2:78-80, 1965.*

[c] *Source: D.T. Teitlbaum and L.C. Kier, "Arsine Poisoning," Arch. Environ. Health 19:133-143, 1969.*

[d] *Source: A.E. De Palma, "Arsine Intoxication in a Chemical Plant: Report of Three Cases," J. Occup. Med. 11:582 587, 1969.*

[e] *Source: B. Nielsen, "Arsine Poisoning in a Metal Refining Plant: Fourteen Simultaneous Cases," Acta Medica. Scand. (Suppl.) 496:1-31, 1968.*

inhalation exposure of workers to soluble salts of nickel and cadmium could occur during the impregnation step of battery manufacture. Cadmium is highly toxic to man.[84]

Because of its increasing presence in the environment and its toxicity, a good deal has been written on cadmium, and there are several thorough reviews on the topic.[83,85,86] This assessment will cover the important highlights of these and other pertinent papers.

7.3.1 Metabolic Uptake, Retention and Excretion

Absorption through Inhalation

Very few studies have identified the fate of inhaled cadmium. Harrison et al.[87] measured the accumulation of cadmium in the tissues of dogs exposed to cadmium chloride ($CdCl_2$) aerosols. Using these data, Friberg et al.[88] estimated that 40% of the dose was absorbed via the lungs of these dogs. Other experiments with mice,[89] rats,[90] and rabbits[91] have indicated a respiratory

absorption factor ranging from 10% to greater than 40%. The absorption efficiency of inhaled cadmium thus appears similar to that of lead particles, as described in Section 7.5.1.

For the occupationally exposed, workplace exposures are without doubt the major source of exposure. For those not exposed occupationally, contaminated food and cigarette smoking are major routes of exposure, and the latter can play an important role in cadmium accumulation. Cigarettes contain about 0.1 to 0.2 μg Cd/cigarette,[81] of which about 50% is deposited in the lung.[83] Over a 20-year period, an additional 15 mg of cadmium can be added to the body burden through use of cigarettes,[81] leading to a linear increase in the amount of cadmium in lung, liver and kidney.[85] More than half of the cadmium burden in cigarette smokers is due to cigarettes.[83] In studies of smokers versus nonsmokers, the former consistently have higher levels of cadmium. The differential can be quite large, as indicated by a recent best estimate[83] of daily cadmium absorption (μg/day) for the average American or western European:

	Cd Absorption, μg/day	
Residence	Nonsmoker	Smoker
Rural	0.36 to 5.78	2.28 to 7.70
Urban	0.37 to 7.88	2.29 to 9.8
Industrial	0.39 to 21.6	2.31 to 23.6

This makes the cigarette smoker highly susceptible to any increase in cadmium exposure from other media.

Absorption through Ingestion

Cadmium is poorly absorbed through the gastrointestinal tract. Most values determined experimentally for cadmium chloride ($CdCl_2$) absorption in mice,[91,92] rats,[93,94] monkeys,[85] and sheep[95] range from 1% to 3%. Cadmium absorption in animals on calcium-[96,97] or protein-deficient[96] diets has been shown to be enhanced.

Tissue Distribution

Cadmium accumulates in the body with time. A theoretical half-life for cadmium in humans has been calculated to be between 9 and 30 years.[85] The kidneys, liver, spleen, pancreas, and testes have been shown to contain the highest cadmium concentrations.[98] Following intravenous, intraperitoneal, or subcutaneous injection of $CdCl_2$, the liver and kidneys contain most of the Cd in the body, with the liver accumulating from 40% to 70% of the cadmium by several hours after its administration.[93,99,100] In man, cadmium accumulates in the liver and kidneys, and to a lesser extent in the pancreas, lungs, thyroid, and other soft tissues (Fig. 7.1). In the "standard" man weighing 70 kg with 30 kg muscle and 10 kg fat, the liver and kidneys contain 50% of the total body burden of cadmium. The kidneys not only accumulate about one-third of the total body burden but also have the highest concentration per gram wet weight.[85] Because of this, the kidneys have been designated the "critical organ" for cadmium toxicity. Further, it has been calculated (Ref. 85, pp. 113-114) that 200 μg Cd/kg, wet weight, is the critical limit in the kidney cortex. A decrease with time in hepatic cadmium levels has been observed to be accompanied by an increase in renal cadmium levels.[100-102] Transfer of cadmium, bound to the protein metallothionein, from the liver to the kidney by way of the bloodstream has been suggested as a mechanism for this redistribution with time.[102,103]

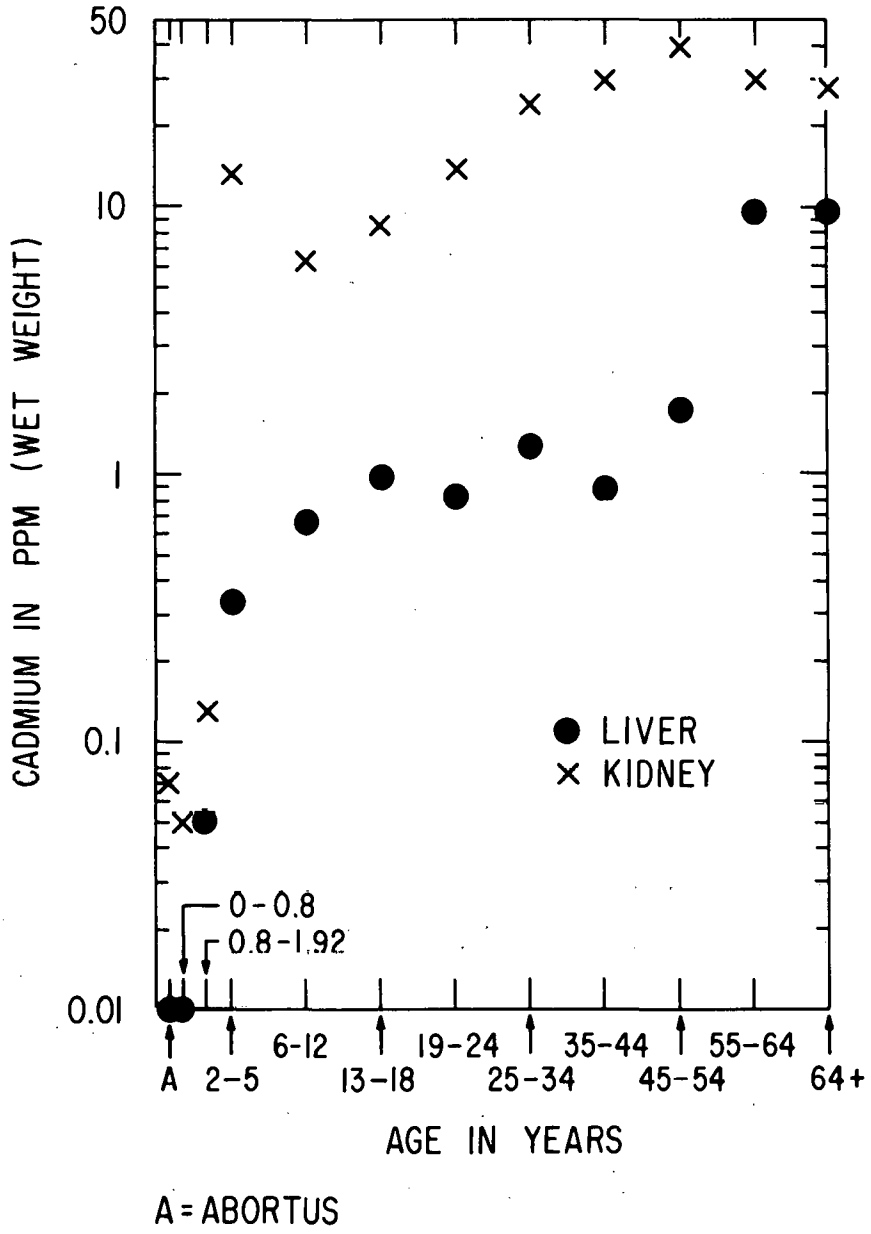

Fig. 7.1. Age Group Mean for Cadmium Content in Liver and Kidney. From: *Health Assessment Document for Cadmium*, U.S. Environmental Protection Agency, EPA-600/8-79-003, Research Triangle Park, N.C., 1979.

Autopsy studies have been used to determine both normal levels in persons not occupationally exposed to cadmium and in persons exposed to cadmium in the workplace. Tipton and Cook[144] used atomic absorption spectophotometric methods to determine the cadmium content of otherwise healthy persons who died in sudden accidents. Cadmium appeared in every kidney sample (median,

2900 μg/g dry ash; 80% range, 1500 to 5100 μg/g), most liver samples (median, 180 μg/g; range, 60 to 340 μg/g), most pancreas samples (median, 80 μg/g; 80% range, >50 to 200 μg/g), and much less often in other tissues. Swedish and Japanese autopsy studies confirm the increasing body burden with age to about 50 years, followed by a decline. A Danish autopsy study[104] showed renal cadmium levels as 82% higher among smokers than among nonsmokers, with the cigar and pipe smokers falling between the two. One review[86] has minimized the value of autopsy studies because they can be composed of biased samples. Differences in consent by social status, occupation and housing, factors associated with pollutant exposure, nutrition, and health, can result in biased samples for those autopsies not legally required. However, the true extent of this effect on autopsy samples was not evaluated, and it probably can be assumed that the composite picture presented by studies from various countries with their associated variances in exposure levels is as accurate as currently possible.

Excretion

Long biological half-lives for cadmium have been estimated for rodents (50 to 250 days), monkeys (>700 days), and man (9 to 30 years).[98] Therefore, only a small fraction of the body's cadmium is excreted from the body per day. Less than 0.2% of a single parenteral dose of cadmium was excreted in the urine of animals with normal renal function over a 4- to 10-day period.[98] Abrupt increases in urinary cadmium excretion have been observed to occur following development of renal dysfunction in response to cadmium administration.[105-109] Fecal excretion of a single parenteral dose of cadmium has also been observed to be low (1% to 2% per day).[100,107,110]

7.3.2 Toxic Responses and Human Health Implications

The primary symptoms of acute cadmium poisoning (Table 7.6) are upper respiratory tract irritation and gastrointestinal tract upset,[111] specifically, "shortness of breath, general weakness, fever, and in severe cases respiratory insufficiency with shock and death" (Ref. 81, p. 137). Pulmonary edema may occur with inhalation of cadmium fumes from smelting activities. Diagnosis is made difficult by a time lag of up to 24 hours in the appearance of symptoms.[86] A lung retention of 4 mg has been estimated to be fatal.[86]

Table 7.6. Number of Workers with Symptoms of Cadmium Intoxication by Blood Cadmium Level

	Number (%) with Sumptoms by Blood Level		
Symptom	*0 to 0.50 μg/100 ml (N = 19)*	*0.51 to 1.00 μg/100 ml (N = 8)*	*1.01 to 4.3 μg/100 ml (N = 8)*
Dyspnea	3 (16)	2 (25)	5 (62)
Chest pain	4 (21)	2 (25)	4 (50)
Dizziness	6 (32)	5 (62)	6 (75)
Dysuria	0	1 (12)	1 (25)

Source: E.L. Baker et al., "Subacute Cadmium Intoxication in Jewelry Workers: An Evaluation of Diagnostic Procedures," Arch. Environ. Health 34:173-173, 1979.

Chronic exposure to industrial levels of cadmium is associated with numerous effects. Exposure to cadmium dust has been associated with decreased lung function; exposure to cadmium fumes, with emphysema.[85] It has been estimated that lung impairment is likely with a chronic cadmium oxide fume exposure of < 0.1 mg/m^3.[86] The most characteristic effect of chronic exposure to cadmium at industrial levels is that on the kidney. The reabsorption function of the proximal tubules is affected, the symptom being the excretion of low-molecular-weight proteins (tubular proteinuria).[81,85] Cadmium oxide dust and fumes have been significantly correlated with low hemoglobin and below-normal haptoglobin levels, resulting in anemia.[85] There is strong suggestion that chronic cadmium exposure and prostatic cancer are related.[83,86] Hypertension has been associated with cadmium exposure, but it is the consensus that data are inconclusive for a cause and effect relationship.[81,86,112,113] Other chronic effects include impaired liver function,[81] possible olfactory impairments,[86] and bone damage (indirectly via renal damage).[81] There is inconclusive evidence linking cadmium exposure and genetic effects,[81,85] male fertility, human intrauterine growth retardation,[83] and teratogenic effects (in laboratory animals).[81]

Ingested cadmium can induce nausea, vomiting, abdominal cramps, and headache.[81] In severe cases diarrhea may occur, followed by shock. Symptoms generally occur within minutes after ingestion of the contaminated food or drink. If the food has no protein, larger concentrations of cadmium can be taken without the induction of vomiting. It has been estimated that cadmium at the level of 15 mg/L can induce vomiting. Violent nausea was experienced by children who ingested food contaminated with cadmium at the level of 13 to 15 mg/L or a total of about 1.3 to 3.0 mg ingested cadmium.[114]

Exposure-Effect Relationships

Information on exposure and response is available from the occupational experience. A lethal dose is 5 mg CdO fumes/m^3 for 8 hours; 1 mg CdO fumes/m^3 for 8 hours is considered immediately dangerous.[83] Dusts are considered less toxic than fumes, and it has been estimated that occupational exposures of > 20 µg/m^3 (8-hr day) would produce only slight lung function changes in susceptible persons in less than 20 years. Extrapolation of this estimate to continuous nonoccupational exposures results in levels of 2 µg Cd/m^3 over a 24-hour day, 70-year exposure period as the probable "no-effect" level for respirable CdO dusts. Another estimate for the no-effect level for an 8-hour workday of exposure is < 1 mg/m^3 of CdO fume and < 3 mg/m^3 of respirable dust.[83] However, exposures of < 0.1 mg/m^3 CdO fumes have also been estimated[85] to result in lung impairment (emphysema) with chronic exposure. This wide range of estimates points to the need for better exposure-response information on inhaled cadmium.

Among workers exposed to cadmium, proteinuria incidence increased with the number of years of exposure.[115] Quantitatively, the extent of proteinuria also increased with exposure in this group except for an unexplained decrease in prevelance and severity among those with 21 to 25 years of experience (Fig. 7.2). Limited air sampling indicated levels of 3 to 15 mg CdO dust/m^3 before installation of a ventilating system and 0.4 to 1.0 mg CdO dust/m^3 after installation. The major symptom among these workers was emphysema. Cadmium stearate dust at levels of 0.021 to 0.69 mg Cd/m^3 produced proteinuria in about 50% of exposed vinyl chloride workers in the very short average exposure time of 3.3 years. Twenty percent of the workers in a plate-making factory exposed to about 500 µg Cd dust/m^3 developed proteinuria in five years. The differentials in incidence of proteinuria and cadmium exposure levels in these two worker studies is significant. One can only guess that the combined exposure to other pollutants may have increased

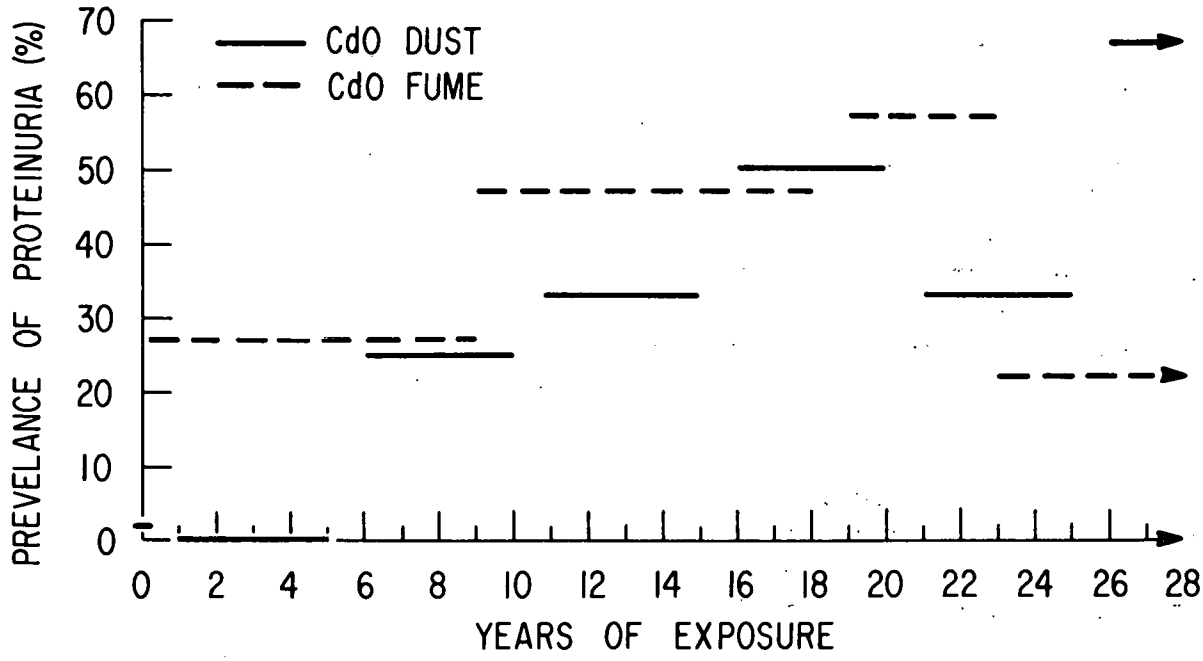

Fig. 7.2. Prevalance of Proteinuria in Workers Exposed to CdO Dust and Fume. From T. Kjellstrom, *Epidemiological Evaluation of Proteinuria in Long-term Cadmium Exposure with a Discussion of Dose-Response Relationships*, pp. 309-330 in *Effects and Dose-Response Relationships of Toxic Metals*, G.F. Nordberg, ed., Elsevier Scientific Publishing Co., Amsterdam, 1976.

susceptibility to cadmium. In contrast, it has been noted from Japanese data that with cadmium fume concentrations of about 50 $\mu g/m^3$, 50% of workers will develop proteinuria. Recent analyses predict that proteinuria can develop in a few years with cadmium dust levels of 0.02 to 0.7 mg/m^3 or fumes of 0.07 to 0.2 mg/m^3. The differential is due to the amount of each absorbed. Kjellström warns[115] that to maintain the incidence of cadmium-induced proteinuria at less than 10% among workers, work-area concentrations must be kept at less than 50 $\mu g\ Cd/m^3$.

Recent studies[116] of workers in a cadmium-nickel battery factor indicate that dust contamination of hands and other body surfaces and then into the mouth is a major source of exposure. The dust is so fine as to be nearly invisible, and personal cleanliness among the workers is variable. Smokers ingest more cadmium than nonsmokers because of the contamination of their cigarettes, in addition to the cadmium contained in the tobacco smoke. Company rules for preventing such exposure are not well followed.

It is interesting to note that olfactory impairments (anosmia) correlated quite well with proteinuria among battery workers exposed to CdO dust and nickel dust. Workers exposed for

10 to 29 years to CdO dust experienced anosmia at a prevalence of 53% to 69%, while those with 30 or more years of exposure had an incidence of 91%. The exposure levels were apparently very high but were not given.[86] Friberg had seen a similar effect in his early studies[117] of cadmium battery workers.

Significant increases in total cancer, and in respiratory system and prostatic cancers have been observed among workers in cadmium smelters.[118] Cadmium exposure levels were generally around 1 mg/m^3 and infrequently rose to 24 mg/m^3. However, respirator use reduced the actual dose by a factor of about 10. In one department a concentration of 1105 μg Cd/m^3 occasionally was sampled. Arsenic also was present in this and another department. Among the 292 white males with at least two years of employment at the smelter, 27 cancer deaths were observed, whereas only 17.6 would be expected statistically ($p < 0.05$). There was a deficit of heart disease and total mortality, with the greatest risk of death 30 or more years after initial employment. For prostatic cancer, there were four observed deaths (1.15 expected) with a latency of more than 20 years. However, this study omitted data on the smoking habits of the workers.

In a 1979 health document on cadmium, the EPA noted[86] that although the occupational studies of various smelter workers have found what seems to be a real excess in total, respiratory, and prostatic cancer rates, the weaknesses of the methodologies used in the studies are sufficient to render the data inconclusive.

Because cadmium accumulates in the body throughout the lifetime of an individual, exposure levels have often been evaluated on the basis of preventing at age 50 a kidney-cortex level greater than the critical limit. The half-life of cadmium in the body is also an important factor, although this value is not known exactly. For example, it has been determined that if the biological half-life of cadmium is 19 years and pulmonary absorption is 25%, then 13 μg Cd/m^3 over an 8-hour workday and 225 workdays per year would result in the critical level being reached in 25 years.[81] If the biological half-life is assumed to be 38 years, then a workplace level of only 11 μg Cd/m^3 would be needed to reach the critical level. These levels would have to be much lower to protect the cigarette smoker.

It is difficult to estimate future trends regarding cadmium levels in the workplace and difficult to postulate, on the basis of past experience, a "safe" level of cadmium in an occupational setting. The current TLV for cadmium is 0.05 mg/m^3 (time-weighted average) for both dust and fume. According to Kjellström[115] this level will keep the incidence of proteinuria in the worker population at less than 10%. However, the value of this level in protecting these workers from emphysema, which can occur with chronic exposure to less than 0.1 mg/m^3 in the workplace,[85] remains to be seen.

Whether or not airborne cadmium will be a health hazard to the population living near a smelter or battery manufacturing plant cannot be determined at this time. Children living and attending school less than 1 km from a Belgian lead smelter[120] had significantly elevated blood and urine cadmium levels ($p < 0.01$ and $p < 0.001$, respectively) in comparison to a similar group in a rural area. There was no difference between blood cadmium levels in children living 2.5 km from the plant and those in the rural area, but a very significant difference ($p < 0.001$) in urine cadmium levels was seen. This was thought to indicate a higher body burden in the group

living 2.5 km from the smelter. An American survey[121] found no significant differences between blood and urine cadmium levels in persons living within 1 km of a cadmium smelter and those living 13 km away.

Based on accidental poisonings, the following rough scale for oral toxicity (single dose) has been proposed:[03] 3 to 90 mg--emetic threshold, nonfatal results; 15 mg--experimentally induced vomiting; 13 to 326 mg--not fatal, but results in severe toxic symptoms; 350 to 3500 mg--estimated lethal dose; 1530 to 8900 mg--reported lethal doses. The upper limit of the single-dose, no-effect level is probably 3 mg.

Japan is the only nation in which the environmental level of cadmium has been so high as to produce cadmium poisoning. As a result, nearly all the information on general population exposure to cadmium derives from studies of the Japanese experience. Briefly, the situation is as follows.[85] Toyama prefecture has had a mining and smelting industry (primarily zinc) for many years. Wastes from this industry have been dumped into the Jintsu River, and the water used by farmers downstream to irrigate rice paddies. Since about the end of World War II, there has been an increased incidence of a debilitating and painful bone disease (Itai-itai, or ouch-ouch, disease) among the inhabitants, particularly postmenopausal women who had borne several (average of six) children.

Over the years, the Western assessment of the situation has changed. In 1974 Friberg and coworkers[85] discussed the Japanese data at great length, but admitted that the conclusions to be gained were relatively small despite the large amount of data. There was no doubt that Itai-itai disease is an expression of cadmium poisoning, but the poor diet, lacking calcium and vitamin D, was considered an important contributory factor. Proteinuria and glucosuria were known to be present in the endemic areas. The Japanese had found the prevalence of proteinuria and glucosuria correlated well (Fig. 7.3) with cadmium levels in the soil ($r = 0.918$ and $r = 0.874$, respectively). These relationships may be expressed by the following:

$$y_p = 9.23x + 1.1$$

$$y_g = 7.06x + 11.2,$$

where

y_p = incidence of proteinuria,

y_g = incidence of glucosuria,

x = soil cadmium concentration.

Although similar relationships were found for lead and zinc (Fig. 7.3), these were thought to reflect the intercorrelation between metal exposure levels. However, these relationships are only approximations, because of the considerable error in the estimation of the dose based on levels of cadmium in rice or water. There were different problems with each study by the Japanese, including poor controls or no controls, a lack of standard methods from study to study, laboratory analyses that were not blind, and poorly characterized exposure levels. Nevertheless, in the most polluted areas it was estimated that the daily cadmium intake was > 300 μg. In somewhat less polluted areas this figure was < 200-300 μg/day, while in generally unpolluted areas the average cadmium intake was < 60 μg/day.

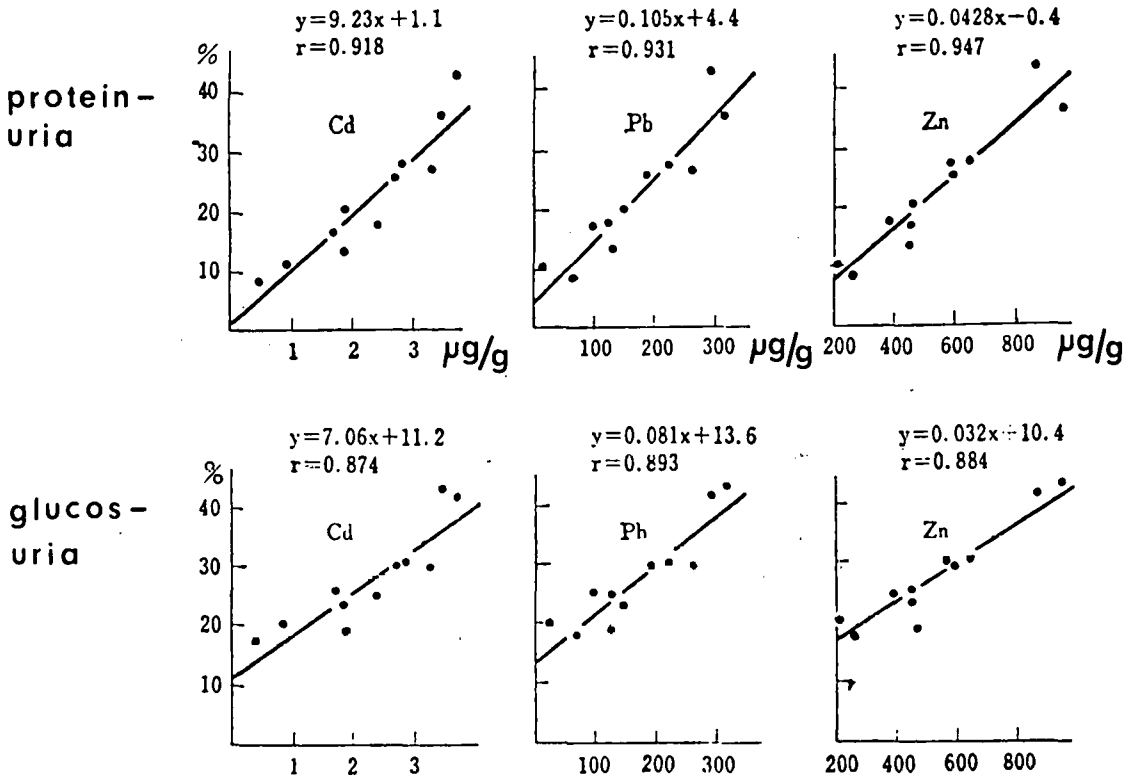

Fig. 7.3. Village Averages of Proteinuria and Glucosuria in Relation to Concentration of Heavy Metals in Paddy Soil. Regression Equations and Correlation Coefficients. From Y. Fukuyama and K. Kubota, Med. Biol. 85, 103, 1972. Reprinted in L. Friberg et al., eds., *Cadmium in the Environment*, 2nd ed., CRC Press, Inc., Cleveland, Ohio, 1974.

Two years later Kjellström[115] reevaluated the situation. He noted that proteinuria was present in 82% to 100% of Itai-itai patients, as well as being common among nonpatients in contaminated areas. The rate of proteinuria was strongly related to age (Fig. 7.4) and nearly four times higher among those 70 years of age and older as compared to those 35. Glucosuria showed the same trend, but there was only a two-fold differential between those 70 and those 35 years old. Differences of a similar order of magnitude were found in several studies of proteinuria and glucosuria incidence. These could not be directly compared for dose-response because of differences in methods of measuring proteinuria and estimating the cadmium dose; however, it was tentatively estimated that consumption of rice with cadmium concentrations of 0.4 to 0.6 μg/g over a period of decades can lead to an increased prevalence of tubular proteinuria in contaminated areas as compared with control areas. At this concentration in rice, the daily intake was about 270 μg of cadmium.

Despite past problems in dose determinations, a recent Japanese report[122] supports the use of average cadmium content of village rice as a good measure of exposure. In a reanalysis of data from three previous studies, an increase in the prevalences of both proteinuria and proteinuria with glucosuria was indicated with increases in the cadmium concentration of village

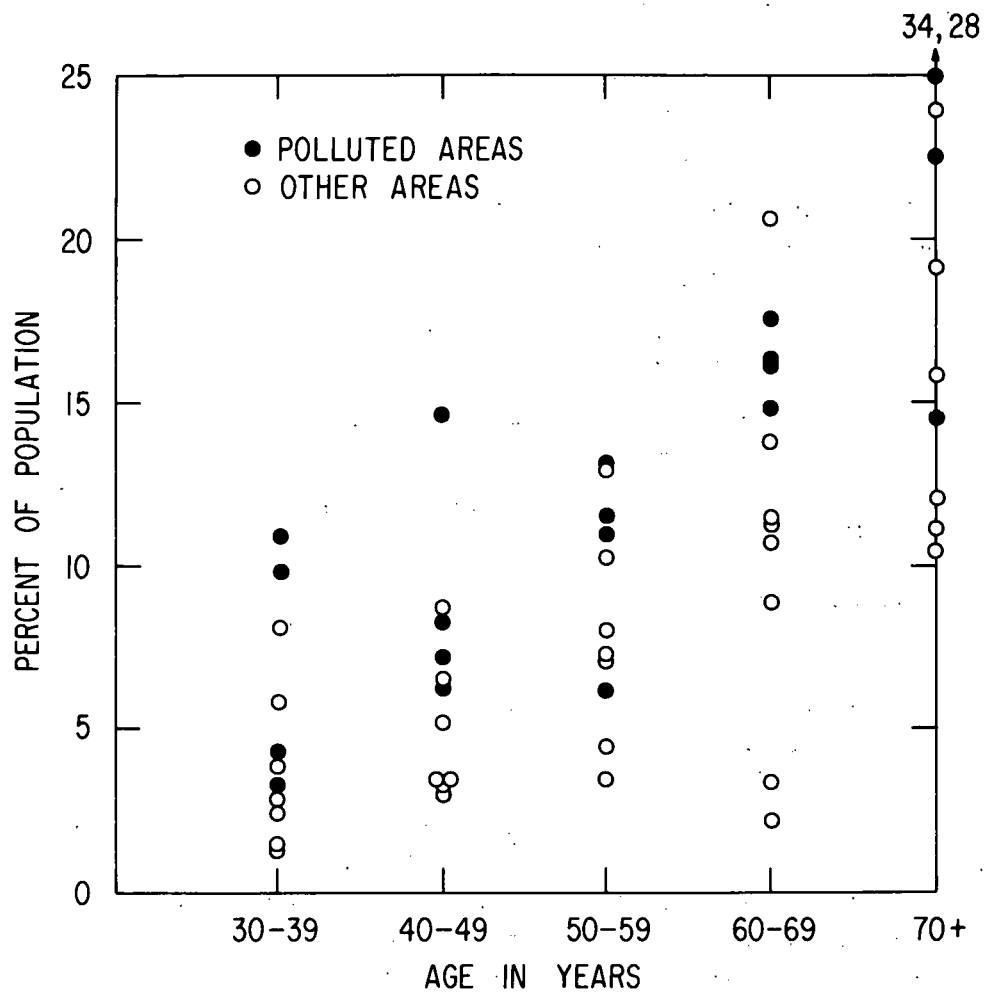

Fig. 7.4. Prevalance of Proteinuria by Age in Cadmium Polluted and Unpolluted Areas of Japan. From L. Friberg et al., eds., *Cadmium in the Environment*, 2nd ed., CRC Press, Cleveland, Ohio, 1974.

rice, residence time in the village, and age. Based on the assumption that the cadmium concentration in rice did not vary significantly over the last several decades, it was calculated that the daily cadmium intake at a concentration of 0.30 to 0.49 μg Cd/g rice was 201 to 287 μg. This value is very close to what Kjellström calculated could result in a critical kidney cortex concentration at age 50.[115]

As a result of a three-phase screening procedure for cadmium poisioning, Nogawa and coworkers[123] found a linear relationship between the log dose of cadmium as μg/g creatinine and the incidence of male and female proteinuria, male and female proteinuria with glucosuria, male and female serum alkaline phosphatase activity, and female hypocalcemia and hypophosphatemia. Although the selection method for the 406 adults (> 30 years of age) is not clear, it seems to have been based on the presence of protein and/or glucose in the urine. The two sexes were

divided in five groups based on the μg Cd/g creatinine: 0 to 9.9 (median, 5.8), 10 to 19.9 (15.0), 20 to 29.9 (25.0), 30.0 to 39.9 (33.8), and 40.0+ (47.1). A probit analysis was used to determine the linear relationship and goodness of fit for the following:

Proteinuria (> 100 mg urinary protein/L)

males $Y = 4.737 + 2.179\ (x-1.179)$

females $Y = 5.122 + 1.835\ (x-1.287)$

Proteinuria with glucosuria (positive for Benedict's reaction)

males $Y = 4.683 + 2.872\ (x-1.278)$

females $Y = 4.830 + 1.928\ (x-1.332)$

Hypocalcemia (< 8.8 mg calcium/dL serum)

females $Y = 5.119 + 0.332\ (x-1.279)$

Hypophosphatemia (< 3.0 mg inorganic phosphorus/dL serum)

females $Y = 4.775 + 1.674\ (x-1.321)$

Increased activity of serum alkaline phosphatase (2.9+ Bessey-Lowry units of serum alkaline phosphatase)

males $Y = 4.349 + 1.158\ (x-1.205)$

females $Y = 5.186 + 1.370\ (x-1.275)$,

where

Y = incidence rate of specific condition

x = μg Cd/g creatinine.

None of these relationships existed for cadmium as μg/L urine. The authors felt the use of cadmium as μg/g creatinine was a useful measure for estimation of health effects, especially if their hypothesis proves correct that Itai-itai disease results form a long-term upset of calcium and phosphorus metabolism in the presence of cadmium. Unfortunately, the authors used a biased sample composed only of persons showing some effect of cadmium poisoning.

When the general population was studied,[124] the criteria for evidence of cadmium effect were altered. The sample consisted of all persons 20 years and older (542) and the criteria definitions were as follows: proteinuria = > 50 mg/liter urine; glucosuria = > 200 mg/liter urine; tubular proteinuria = > 5 mg β_2-microglobulin and > 4 mg retinol binding protein/liter; aminoaciduria = > 10 mg proline/gram creatinine. The prevalence of each in unexposed areas of Japan was less than 1%. The exposed population was divided into eight groups on the basis of micrograms cadmium per gram creatinine. As was the case with their previous study of a highly selected subset of the population, the incidence rate of the conditions generally increased with the level of cadmium in the urine, measured as μg Cd/g creatinine.

Outside of Japan there has been much less work done on cadmium poisoning. In the United States, Berg and Burbank[124] correlated information on the trace-element content of river basins with the cancer mortality rates, by race and sex, for those states entirely within a single river basin. Many positive associations were evident; however, this single-variable approach is deficient in many ways, including a lack of consideration for the socioeconomic differentials of the sampled areas and the assumption that river samples reflect human cadmium intake (which they may or may not). The authors note that cadmium and cancers related to cigarette smoking have

the highest correlations in those states where smoking is also high. Such studies merely point to the need for more research.

Some cadmium ingestion may occur among the occupationally exposed as a result of poor hygiene and smoking on the job. However, the major concern is for the general public, as ingestion of cadmium through contaminated food is the most important medium for public exposure.[81] One group at especially high risk is cigarette smokers residing close to lead and zinc mines or smelters. Near smelters, short-term levels as high as nearly 0.5 μg Cd/m^3 have been recorded.[83] The reality of this concern is supported by the elevated concentrations of cadmium in soil and garden vegetables in an old zinc-mining area of England.[119]

As was the case with inhaled cadmium, a good deal of work has been done on modeling ingested cadmium intake and time necessary to reach the critical level. Kjellström[115] described biological half-time in the kidney cortex as an important factor in individual sensitivity when the exposure time is quite long. Essentially, the more one excretes each day the larger the daily exposure level which can be handled without reaching the critical limit. In one such calculation,[115] Kjellström made some estimations based on a 50-year exposure time, the variations in amounts eaten at various stages of life, and the changing size of the kidney with age. If the body excretes 0.02% of the body burden daily, then 616 μg of cadmium is the daily intake necessary to reach the critical level in the kidney cortex at age 50. However, if only 0.005% of the body burden is excreted daily, and this is according to Friberg the more reasonable excretion rate, then only 248 μg of cadmium ingested per day is required to reach the critical limit at age 50.

Kjellström (as cited in Ref. 186) took these estimations a step further and derived approximations of the proportion of the population with renal tubular damage given a variety of average daily cadmium intake levels. At 32 μg Cd/day, 0.1% of the American population at 50 years of age could be expected to have renal tubular damage, whereas with 100 μg Cd/day 5% of the population could be affected, and with 440 μg Cd/day, 50% of the population could have renal tubular damage. These estimations must be viewed with caution as they are based on a number of assumptions. The model fits the observed rates best for exposures greater than 80 to 100 μg Cd/day. At present, estimations of average American cadmium intake through food vary from 4 μg to 70 μg/day,[81,83,86] with an estimated absorption rate of < 4%[125] to 7%. Friberg et al.[85] have estimated that with 4.5% retention and 0% excretion per day, 164 μg Cd/day can be consumed by an average adult and result in the critical kidney concentration by age 50. If 0.005% is excreted daily (a more reasonable assumption), then 248 μg cadmium would have to be consumed daily to reach the critical concentration at age 50. To put this in perspective, let us assume that as a result of activities related to battery manufacture, members of the general public near a zinc smelter (currently each consuming 70 μg Cd/day in food) experience a 10% increase in cadmium intake in their food. Each person would then be consuming 77 μg Cd/day. This is only one-third of that calculated by Friberg as necessary to produce proteinuria after 50 years of exposure. If, as a recent paper by Ellis and coworkers[125] suggests, the amount absorbed is less than 4%, then more than 50 years' exposure to this level of cadmium would be necessary to produce proteinuria.

The significance of any change in the cadmium concentration of food is modified by several factors. It has been calculated[112] that over a 50-year exposure period and with an assumed biological half-life of 38 years, a total daily retention of 10 μg cadmium will result in renal dysfunction. According to Ellis et al.,[125] a person who smokes two packs of cigarettes per day

already is consuming about 3.8 µg (1.9 µg Cd/pack) of this daily limit. A food exposure of 77 µg Cd/day with 4% retention brings this total to nearly 8 µg Cd/day. Thus, for the cigarette smoker, increases in food cadmium become more significant by virtue of the smoking habit (Table 7.7).

Other dietary factors may also influence the significance of an increase in cadmium through the food pathway. It has been shown with rats[126] and is also suggested by the Japanese experience that a low-calcium diet can increase cadmium absorption in the gastrointestinal tract. Cadmium in the body interacts with several other elements to produce effects other than proteinuria. One of these elements is zinc, which may be redistributed in the body as a result of the binding of cadmium to zinc-dependent enzymes. This can affect essential functions[81] such as increasing blood pressure and/or atherosclerosis in humans.[126] Cadmium and zinc can interact with copper in the arterial wall, thereby interfering with "copper-mediated cross-linking of elastin and collagen."[126] In rats, the interaction of these three metals can affect liver activity.[126] On the other hand, dietary factors may diminish the toxicity of cadmium. The inactivation of cadmium by selenium has been hypothesized to instill a protective effect against hypertension,[104] deleterious effects on the reproductive system, and teratogenic effects on the fetus of laboratory animals.[127]

At present, one can not project the effect of a dietary increase in cadmium on the general population. According to the Kjellström model,[86] between 0.1% and 2.5% of the population 50 years of age and older should show proteinuria at present levels of dietary cadmium. The World Health Organization recommends[83] that average daily intake not exceed 57 to 71 µg Cd, about the present upper limit of American dietary intake. The actual incidence of tubular proteinuria needs to be determined both in the general population and the high-risk group of cigarette smokers living near sources of cadmium pollution.

From the foregoing discussion it is apparent that a major problem with cadmium toxicity studies is the determination of the amount in various vital organs and the associated effects. A recently reported method may offer a solution to this problem, although its economic usefulness has not been determined. Ellis and coworkers[125] used a partial body neutron activation technique to determine *in vivo* the amount of cadmium in the liver and left kidney of 20 healthy male volunteers. Blood and urine cadmium levels were determined by atomic absorption spectrophotometry after wet ashing. These men were not occupationally exposed to cadmium, but 12 were cigarette smokers. A comparison thus was made between smokers and nonsmokers. Blood and urine cadmium concentrations and blood and urine β_2-microglobulin (a low-molecular-weight protein) concentration did not significantly differ between the two groups. However, kidney and liver cadmium levels were different at the $p = 0.05$ level of significance and total body burden different at the $p = 0.01$ level of significance. Smokers had about twice as much liver, kidney and total body cadmium as did nonsmokers. Based on the body burden of 19.3 mg Cd for nonsmokers (average age 52 yrs) and 35.5 mg Cd for cigarette smokers (average age 50 yrs), the calculated biological half-time was 15.7 years, with a range of 10 to 33 years. The smokers had smoked an average of 1.4 packs per day for an average of 27 years (38.7 pack-years). Daily retention was calculated to be about 2.7 µg Cd; with a dietary intake of 75 to 100 µg Cd/day, the absorption rate would be < 4%. (Friberg has estimated 5% absorption.) As a group, smokers had an additional 16.2 mg Cd body burden, suggesting an inhalation dose of 1.9 µg Cd per pack (Friberg has estimated 2 to 4 µg Cd/pack). Thus, the inhalation absorption rate is 48 to 95%, a value also in agreement with that calculated by Friberg and coworkers (50%).[85] These data confirm earlier

Table 7.7. Estimated Relative Contribution of Dietary Intake, Cigarette Smoking and Ambient Air Cadmium Levels to Total Daily Cadmium Retention from All Sources[a]

Dietary Intake (net retention)	*Smoking Status/Day (net retention µg/day)*	*Air Cadmium Levels (net retention)*				
		0.0 ng/m³ (0.0 µg/day)	*1.0 ng/m³ (0.0005 µg/day)*	*10 ng/m³ 0.005 µg/day)*	*100 ng/m³ (0.05 µg/day)*	*1000 ng/m³ (0.5 µg/day)*
Food Level A	Nonsmoker (0.00)	1.590 (0;0)[b]	1.591 (0;0)[b]	1.595 (0;0)[b]	1.630 (0;3)[b]	2.090 (0;24)[b]
25 µg/day	0.5 pack (0.70)	2.290 (31:0)	2.291 (31;0)	2.295 (31;0)	2.330 (30;2)	2.790 (25;18)
(1.50 µg/day)	1 pack (1.41)	3.000 (47;0)	3.001 (47;0	3.005 (47;0)	3.050 (46;1)	3.500 (40;14)
	2 pack (2.82)	4.410 (64;0)	4.411 (64;0)	4.415 (64;0)	4.460 (63;1)	4.910 (57;10)
	3 pack (4.22)	5.810 (73;0)	5.811 (73;0)	5.815 (73;0)	5.860 (72;1)	6.310 (67;8)
Food Level B	Nonsmoker (0.00)	3.090 (0;0)	3.091 (0;0)	3.095 (0;0)	3.140 (0;2)	3.590 (0;14)
50 µg/day	0.5 pack (0.70)	3.790 (18;0)	3.791 (18;0)	3.795 (18;0)	3.840 (18;1)	4.290 (16;12)
(3.00 µg/day)	1 pack (1.41)	4.500 (31;0)	4.501 (31;0)	4.505 (31;0)	4.550 (31;1)	5.000 (28;10)
	2 pack (2.82)	5.910 (48;0)	5.911 (48;0)	5.915 (48;0)	5.960 (47;1)	6.410 (44;8)
	3 pack (4.22)	7.310 (58;0)	7.311 (58;0)	7.315 (58;0)	6.360 (57;1)	7.810 (54;6)
Food Level C	Nonsmoker (0.00)	4.590 (0;0)	4.591 (0;0)	4.575 (0;0)	4.640 (0;1)	5.090 (0;10)
75 µg/day	0.5 pack (0.70)	5.290 (13;0)	5.291 (13;0)	5.295 (13;0)	5.340 (12;1)	5.790 (12;9)
(4.50 µg/day)	1 pack (1.41)	6.000 (24;0)	6.001 (24;0)	6.005 (24;0)	6.050 (23;1)	6.500 (22;8)
	2 pack (2.82)	7.410 (38;0)	7.411 (38;0)	7.415 (38;0)	6.460 (38;1)	7.910 (36;6)
	3 pack (4.22)	8.810 (48;0)	8.811 (48;0)	8.815 (48;0)	8.860 (48;1)	9.310 (45;5)

Source: "Health Assessment Document for Cadmium," EPA-600/8-79-003, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, 1979.

[a] *Note that a total daily retention level of 10 µg/day would be expected to yield renal dysfunction over a 50-year exposure period.*

[b] *The first value signifies the total daily cadmium retention levels (in µg/day) from all sources, assuming constant daily retention from water of 0.09 µg/day derived from consumption of water with cadmium concentration of 1 ppb. The two values in parentheses following this value indicate the percentage of total daily cadmium retention attributed to cigarette smoking and to ambient air cadmium exposure, respectively, at a given ambient air cadmium level.*

warnings on the contribution of smoking to cadmium body burden and the inability to use plasma or urine levels as indicators of total body burden or single-organ levels of cadmium. They offer a promising technique for determining organ levels and total body burden on a group basis and may be very useful in the occupational setting.

7.3.3 Protection, Remediation and Diagnostic Techniques

Protection of the environment from exposure to cadmium-containing particulates generated during zinc smelting is effected by use of cyclone precipitators, wet scrubbers, and electrostatic precipitators.[98] Protection from cadmium exposures in the workplace is afforded by use of respirators combined with adequate ventilation of workroom areas.[118]

An effective means for therapeutic removal of cadmium from the body is not currently known. MacFarland[117] has urged the use of BAL for acute inhalation of cadmium oxide fume. BAL directs the cadmium to the kidney, where it can be excreted. The low solubility of the CdO fume would probably result in a kidney loading low enough so as not to be nephrotoxic following BAL therapy and yet enable BAL to remove the CdO from the lungs quickly. However, this therapy is not recommended for the more soluble forms of cadmium, as the loading mobilized to the kidney could result in severe or even fatal kidney damage.

Cadmium levels in hair, blood and urine are generally of limited value in evaluating an individual's organ level. Blood levels can indicate current exposure,[83,128] but generally not body burden.[83] Only after worker exposure has ceased for a considerable time are blood levels a reflection of organ levels.[85] More recently, however, a study[129] of 23 otherwise normal patients who had gallstone operations indicated that blood cadmium level is a fairly good indicator of liver cadmium level on a group basis (Fig. 7.5). Analysis of liver biopsies, blood and bile samples using atomic absorption spectrophotometry (AAS) techniques indicated that blood cadmium correlated well (correlation coefficient = 0.57, $p < 0.01$) with the cadmium concentration in liver (dry weight) on a group basis. There apparently is no correlation between cadmium in the blood and the exposure period or amount of proteinuria. Even though the blood cadmium may be low, the levels in organs are not necessarily low also.[85]

Also on a group basis and with low levels, there is a relationship between urine levels of cadmium and the total body burden.[85] Japanese data indicate that when the group average renal cortex level is 100 μg Cd/g, wet weight, the urine level is about 1.8 μg Cd/L. Assuming this relationship is valid at higher exposures, an excretion of about 5 μg Cd/day would correspond to approximately 200 μg Cd/g in the kidney cortex. Normal urinary excretion is about 1 to 2 μg/day,[85] with a concentration range of 0.2 to 3.1 μg/L.[83] This correlation between urine levels and body burden was reconfirmed in a recent three-country cooperative study of the general population.[130]

7.4 COBALT

Cobalt (Co) is essential in trace amounts to human health, and is present in vitamin B_{12} at about 4.34% by weight. As a metal, cobalt has a wide variety of uses--in hard metal alloy production, in oil and chemical industries, as a catalyst in glass and china manufacture and medicinally in the treatment of anemia.[132] Even though cobalt is a relatively rare element, human contact with it is already quite extensive.

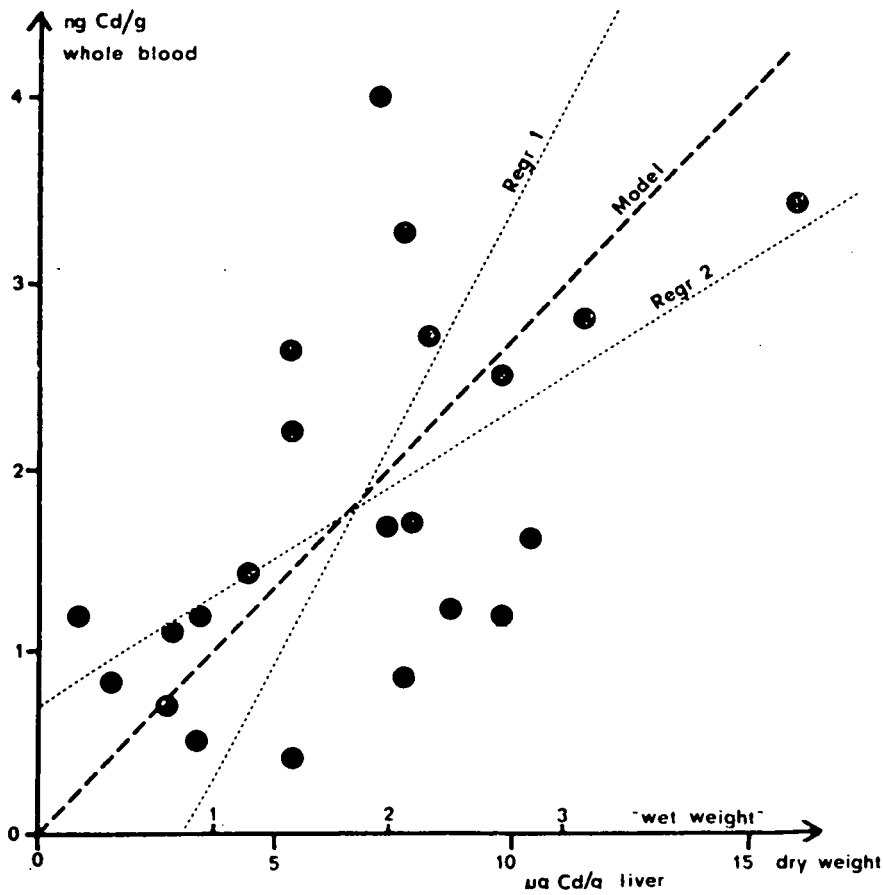

Fig. 7.5. Comparison between Cadmium in Liver Biopsy Material and Cadmium in Whole Blood of 22 Swedes who underwent Gallbladder Operations. Regression Lines and the Relationships Calculated from the Metabolic Model by Kjellstrom and Nordberg (1978) are included. From C. G. Elinder et al., *Cadmium Concentrations in Human Liver, Blood and Biles: Comparison with a Metabolic Model*, Environmental Res. 17: 236-241, 1978.

Cobalt is a component of the nickel electrode in the Ni/Zn and Ni/Fe battery systems. The use of cobalt in the United States should increase by 64% of 1973 world production levels for the Ni/Zn battery system and by 35% for the Ni/Fe system, by the year 2000, if EV production proceeds as estimated (see Tables 3.15 and 4.13). Reliance on foreign sources for this cobalt appears necessary (see Sec. 3.3.3). An insoluble form of cobalt is added as part of the nickel electrode used for Ni/Fe battery manufacture. In addition, cobalt nitrate [$Co(NO_3)_2$] is present as a component of the $Ni(NO_3)_2$ impregnation bath for the nickel electrode of the Ni/Zn battery and present in the $Ni(NO_3)_2$ electrolysis bath for electroprecipitation of active material on the nickel electrode of the Ni/Fe battery. As with nickel, cobalt will have to be recycled if battery systems containing cobalt are to be economically viable. The recycling process will introduce the potential for exposure to other forms of cobalt such as cobalt hydroxide [$Co(OH)_2$], cobalt sulfate ($CoSO_4$) and cobalt oxide (CoO). A review of pertinent literature on cobalt

metabolism and toxicity is presented here relating to exposure to these compounds of cobalt. Metabolic and toxic reactions resulting from exposure to cobalt compounds other than those listed above and by exposure modes other than inhalation or ingestion are also presented, to assure a thorough presentation of the implications of human exposure to increasing amounts of cobalt. Information pertinent to this assessment but not available in the literature relates to potential synergistic effects of exposure by the inhalation of vapors containing both $Ni(NO_3)_2$ and $Co(NO_3)_2$. Exposure to these vapors might occur during the impregnation or electroprecipitation steps of nickel electrode manufacture.

7.4.1 Metabolic Uptake, Retention and Excretion

The major route of cobalt exposure in humans is by way of food intake. Water is reported to contain very little cobalt,[143] and there are no water quality criteria for cobalt. A much smaller percentage of the population is exposed to cobalt either as dusts from hard metal alloy production or grinding, as salts (oral administration) in the treatment of anemia, or as particles from the physical wear on cobalt-chromium alloy joint prostheses.

Absorption through Inhalation

Wehner and Craig[133] investigated the deposition, absorption and retention of CoO after inhalation exposure of hamsters for 6 hr/day, 5 days/week over a period of three weeks. Deposition of 784 µg of CoO particles was observed in the lung following exposure to a cobalt aerosol at a mean concentration of 8.4 µg/L (8.4×10^3 µg/m^3) and having a MMAD in the range of 1.0 to 2.5 µm. The lung burden was cleared quite rapidly, with only 1.1% of the deposited CoO remaining in the lung three days after exposure. By six days after exposure practically all the CoO had been eliminated from the lungs. A fairly high percentage (60%) of the initial lung burden was measured in the gastrointesinal tract at 24 hours, reflecting clearance of the upper respiratory tract. The lack of any significant levels of CoO in the lung six days after exposure suggests that absorption of the CoO took place from the lung, followed by redistribution within the body. The observations of Wehner and Craig are in conflict with information presented by the ICRP Committee II Task Group on Lung Dynamics which classifies CoO under class "W", substances deposited in the lung having moderate retention, and having clearance rates on the order of weeks. In Syrian golden hamsters the clearance rates of inhaled CoO are on the order of days. Further support for the rapid clearance of inhaled CoO comes from the work of Barnes et al.[134] They exposed dogs to a well-characterized aerosol of ^{60}CoO and saw a similarly rapid clearance from the lungs. Eight days after exposure only about 10% of the initial lung burden remained in the lungs. Elevated levels of cobalt in the urine of the exposed dogs provides further evidence that a significant fraction of the CoO retained in the lungs was rapidly absorbed and excreted. It appears therefore that the insolubility of CoO in water is not reflected by a slow disappearance or dissolution of cobalt in the lungs after inhalation exposure of respirable-sized particles.

Absorption through Ingestion

In addition to their inhalation studies, Wehner and Craig[133] administered CoO to guinea pigs by gavage. The results showed CoO to be poorly absorbed from the gastrointestinal tract. Less than 0.5% of the 5-mg dose was absorbed and accounted for in the body tissues at 24 hours after exposure. Other investigators have seen much higher gastrointestinal absorption when soluble salts of cobalt were administered orally. Absorption of an oral dose of 20 µmoles of

radioactive cobalt chloride ($CoCl_2$) in normal human control subjects was 44%.[135] Weissbecker[136] administered cobalt orally to humans and found that 8% of a 25-mg dose (190 μmoles) was excreted into the urine within 48 hours. This is in agreement with the results of Paley et al.,[137] who found 4% to 8% of a 12.5-mg oral dose (95 μmoles) of $CoCl_2$ in the urine over a similar period. If one assumes negligible tissue retention of the administered cobalt, these levels of cobalt found in the urine approximate the amount absorbed after oral administration. It then appears that the fraction of cobalt absorbed by the gastrointestinal tract is variable and may depend in part on the amount administered. Additional evidence is provided which suggests that iron and cobalt may share a common pathway for absorption in the intestine.[138,139] The absorption of cobalt in human subjects with iron deficiency averaged 71%.[135] Schade also showed that an equimolar amount of cobalt decreased gastrointestinal absorption of iron in the rat.[138] One unexpected result in these investigations was the observation that conditions which reduced iron absorption (i.e., iron overload) did not diminish cobalt absorption. In a study by Valberg et al.,[135] five patients with exogenous iron overload showed decreased iron absorption (10% vs. 27% in normal subjects), while cobalt absorption was within the range seen in control subjects. This study's results are in agreement with Schade's experimental work in rats which also demonstrated[138] that cobalt absorption is not always decreased by conditions that reduce iron absorption.

Tissue Distribution

The exposure of hamsters to CoO by Wehner and Craig[133] showed only 3.25% of the initial lung burden to be present in the lungs at 24 hours after exposure. The liver and kidneys of these animals contained only 0.2% each of the initial lung deposition, with the carcass retaining approximately 23.3%. The tissue localization of the cobalt in the carcass was not determined. In another study, Berlin[140] administered multiple daily injections of 0.125 mg of radioactive $^{60}CoSO_4$ intraperitoneally to rats for 105 days and studied its localization in several tissues. Cobalt was not accumulated to any great extent with time in any of the tissues analyzed, indicative of its rapid excretion from the body. The levels of cobalt in liver, kidney and lung reached a plateau after several days and did not increase significantly, even by 105 days. A 2- to 4-fold increase in concentration in red blood cells, spleen and bone marrow was observed between the 1st and 15th week of cobalt administration. The latter increases were accompanied by a 73% increase in red blood cell count and a 38% increase in hemoglobin concentration induced by cobalt. The tissue concentration (μg/g) at 105 days was highest in liver (1.7), kidney (1.4), and bone marrow (1.7), with spleen (0.8), red blood cells (0.4), and lung (0.2) somewhat lower. Ulrich and Copp[141] administered 52 μg of $^{60}CoCl_2$ intraperitoneally to rats and studied its tissue distribution between 0.5 and 24 hours after injection. At 1 hour, the liver, carcass, pancreas, kidneys, and blood contained 34.7%, 31.6%, 8.9%, 4.2%, and 1.7% of the injected dose, respectively. At 24 hours these values fell to 6.3%, 8.3%, 0.5%, 0.8%, and 0.6% of the injected dose, respectively, with 65% of the administered cobalt accounted for in urine. A careful study of cobalt tissue distribution after either intravenous or oral administration of radioactive $CoCl_2$ to calves, swine and rabbits was reported by Comar and Davis.[142] Thirty to 60 μg of $CoCl_2$ was administered, and animals were killed at times varying from 17 hours to 11 days after cobalt. Cobalt was cleared very quickly from the body, confirming the findings of others. At all times studied, the livers contained a far greater percent of the administered dose than any other tissues analyzed. Other tissues containing significant amounts of the total tissue cobalt were kidney, bone, small intestine and lung. The concentration of cobalt (μg/100 g) in the tissues of a 6-month-old swine at 23.5 hours after intravenous injection were kidney (0.60), adrenals (0.38), liver (0.30), pancreas (0.22), spleen (0.13), thyroid (0.11), intestinal lymph glands

(0.10) and gastrointestinal mucosa (0.075-0.14), indicating a fairly high initial concentration in the glandular organs. Relative concentrations in various tissues were found to be similar for injected and orally administered cobalt.

Body Burden and Routes of Uptake

The human body burden of cobalt is uncertain, but is believed to be less than 1.1 mg.[131] Schroeder et al.[143] describe the tissue distribution on a wet-weight basis to be fairly evenly divided, except for a two-fold greater concentration in the liver, prostate, and heart and a four-fold greater concentration in the aorta. All these concentrations are less than 1 μg Co/g tissue. The greatest percentage of cobalt occurs in muscle, but the highest concentration is in the fatty tissue. This distribution pattern is only partly supported by atomic absorption spectrophotometry determinations on autopsy tissues.[144] The median level for all tissues tested as dry ash was < 2 μg Co/g, with the exception of the diaphragm, which had a median of < 5 μg/g. Tissues with the highest maximum concentrations were the adrenal, and parts of the intestine, liver, and skin. Differences in methodology between studies cannot be ruled out in the explanation of these variations. In any case, the average concentrations, like the body burden, are quite low.

Normal serum or plasma levels also show discrepancies from study to study. As Versieck and co-workers[145] have shown, the range of values is 0.02 to 71.0 ppb, and the averages range from 0.28 to 70 ppb. Using neutron-activation analysis on samples from 20 people to determine serum levels, they found an average serum cobalt level of 0.105 ± 0.058 ppb, with a range of 0.0384 to 0.264 ppb. Two apparently healthy people had serum cobalt levels of 0.228 and 0.264 ppb.[146]

Retention

The dietary intake and the fecal and urinary excretion of cobalt in humans have been measured.[143] Analysis of a two-day hospital diet and a one-day institutional diet showed daily cobalt intake to be between 166 and 436 μg/day. The daily dietary intake of cobalt measured using emission spectroscopy, for one man and one woman over a 30-day period and for two men measured for four periods of 60 to 61 days averaged 283 ± 66 μg/day (range: 160-580 μg/day). The fecal output was 39 ± 6.8 μg/day (13.4%) and the urinary output averaged 245 ± 31 μg/day (86.6%). In this study the balance between ingested and excreted cobalt was equal, indicating that cobalt was not being retained by the body to any great extent.

Other estimates of the average daily cobalt intake range from 0.16 to 0.69 mg.[146] This range brackets the 0.3 mg estimated by Beliles[131] and the 0.16 to 0.58 mg determined by Schroeder et al.,[143] but it is orders of magnitude greater than the 5.6 to 7.8 μg/day recorded by Harp and Scoular.[147] The discrepancy could easily be due to the differences in methodology and perhaps an increased occurrance of cobalt in foods over the 17-year period between determinations.

Excess cobalt appears to be eliminated from the body fairly rapidly. Six days after intravenous injection and oral administration of $^{60}CoCl_2$ to rabbits, Comar and Davis[142] found that less than 10% and 5% of the administered dose was retained in the organs measured, respectively. In pigs, only 11.6% of the injected dose was retained 24 hours later and less than 2% remained 4 days after oral administration of $^{57,60}CoCl_2$. The work of Copp and Greenberg[148] with rats showed less than 3% of the dose retained in the organs analyzed four days after oral administration

of $^{60}CoCl_2$. Evidence that cobalt does not accumulate in the body with age has been presented by Tipton et al.[144] from their analysis of human tissue. Their results showed no trends suggestive of either accumulation or depletion of bodily cobalt levels with age.

Excretion

The cobalt absorbed after oral or parenteral administration and inhalation exposure appears to be excreted mainly in the urine. An average of 63.5 ± 8.5% of a 0.1-mg dose of a radioactive cobalt salt injected intraperitoneally was found in the urine of rats 72 hours after exposure.[149] Feces and bile contained 8.4% of the dose. When cobalt was administered orally, the amount excreted in urine by 72 hours was 18.5 ± 12.0%. After intravenous injection of radioactive $^{60}CoCl_2$ to young calves, 46.7% of the injected dose was found in the bladder urine at 17 hours.[142] In rabbits receiving an intravenous injection of radioactively labeled $^{60}CoCl_2$, 44% of the injected dose appeared in the bladder and urine after 5 hours.[142] Copp and Greenberg's work[148] with rats also demonstrated the rapid excretion of parenterally administered $^{60}CoCl_2$; 70% was excreted within the first 10 hours, and over 90% within 2 days. Valberg et al.[135] administered 10 μmoles of labeled $^{60}CoCl_2$ to six human control subjects by intramuscular injection. Within 5 days 56.4% of the injected dose was excreted, 52% accounted for in the urine. The fecal excretion of soluble cobalt after oral administration is quite high due to the incomplete absorption of cobalt from the gastrointestinal tract.[135,144,149] After the early fecal excretion of material deposited in the upper respiratory tract, urinary excretion was the predominant route of elimination when dogs were exposed to an aerosol of ^{60}CoO.[134]

In summary, the absorption of CoO from the lung after inhalation is much higher than would be expected from its solubility in water or from previous experience with other metal oxides (e.g., NiO). Only 3.25% of the initial lung burden remained 24 hours after inhalation exposure of hamsters to CoO. Similar results were obtained after inhalation exposure of dogs: the high degree of absorption of the retained lung deposition was reflected by elevated levels of blood and urinary cobalt. Absorption of cobalt from the gastrointestinal tract from CoO given by gavage is low, less than 0.5%. Much higher absorption is seen when soluble salts of cobalt are given orally. Almost 50% of an oral dose of $^{60}CoCl_2$ was absorbed in normal human subjects. Despite the high absorption after inhalation and oral administration, cobalt does not appear to accumulate in the organs of exposed animals to any great extent. The main pathway for excretion of cobalt administered as a soluble salt, such as $CoCl_2$, or as the insoluble CoO, appears to be the urine. Biliary excretion of $CoCl_2$ has also been observed.

7.4.2 Toxic Responses and Human Health Implications

Hematopoietic System

The administration of cobalt has been shown to produce polycythemia in many animal species, including humans.[150-161] Mixtures of cobalt and iron have been used for many years to treat certain types of anemia. Much of the information on cobalt dose-response is derived from the case report literature of cobalt chloride therapy. Table 7.8 is a condensation of much of that literature. Oral administration of 100 mg $CoCl_2$ daily for six days followed by 200 mg per day for 11 weeks produced polycythemia in hematologically normal psychotic humans.[150] An average increase of one million erythrocytes/mm^3 and a 17% increase in hemoglobin were seen. The daily oral dose of cobalt necessary to produce a hemopoietic response is estimated to be

Table 7.8. Cobalt Dose-Response in Humans

Age	*Cobalt Dose*	*Treated for*	*Outcome*	*Time to Outcome*	*Remarks*	*Reference*
4 children	3 mg/kg/day	Sickle cell anemia	Goiter in 3	6-8 wks	Enteric coated; 2 had thyroid hyperplasia; dangers outweigh benefits; 1 died	a,b
Infant (8 mo)	20 mg/day	Microcytic anemia	Goiter	8 wks	Co/Fe liquid	c
Infant (8 mo)	20 mg/day	Microcytic anemia	Thyroid enlargement	8 wks	Co/Fe liquid	c
Infant (17 mo)	40 mg/day	Undefined anemia	Goiter	12 wks	Co/Fe liquid; at critical point, evidence of carciac failure	d
Infant (newborn)	13 mg/day (first 3 days) 24 mg/day	Anemia	Death	8 wks	Thyroid hyperplasia	e
Infant (9 mo)	12 mg/day	Anemia	Goiter	12 wks	Recovered	e
Child (21 mo)	12 mg/day few days, thereafter 6-8 mg/day	Anemia	Goiter	4 wks		e
Child (4 yrs)	10 mg/day + 44 mg Fe/day	Anemia	Goiter, hypothyroidism	12 wks	Also had lipoid nephrosis treated with thiosemicarbazone	f
Adult (84 yrs)	200 mg/day	Subacute bone marrow hypoplasia	Hemoglobin increased, recovered	~ 18 days	No bad side effects	g
Adult (38 yrs)	200 mg/day	Chronic anemia bone marrow hypoplasia	No response, hemoglobin remained low	17 days		g
Child (23 mo)	120 mg/day	Bone marrow hypoplasia	Hemoglobin increased, bone marrow returned to normal	Months?		g
23 infants (1-24 mo)	40 mg/day + Fe sulfate (only given to some)	Iron deficiency anemia	Uneventful	Up to 40 days	Toxic reactions in 3 who were given $CoCl_2$ before meals	g

Table 7.8. Continued

Age	*Cobalt Dose*	*Treated for*	*Outcome*	*Time to Outcome*	*Remarks*	*Reference*
58 adults (pregnant)	60-90 mg/day	Anemia of pregnancy	Uneventful	12-24 wks	No toxic reactions in mother or offspring, 1 developed skin rash, which disappeared with discontinuance of treatment	h
20 adults (pregnant)	75-100 mg/day	Anemia of pregnancy	Uneventful	12-24 wks		h
17 children (5-9 yrs)	1,2,4,6 (mg/kg)/day	Possible thyroid problems (function tests performed)	Uneventful	10 wks	Studied thyroid function (I-131 uptake + other tests) no change in any child	i
Child (13 yrs)	2.8-3.9 (mg/kg)/day	Hyperthyroidism	No response	8+ wks		j
7 adults (22-50 yrs)	2.8-3.9 (mg/kg)/day	Hyperthyroidism	2-no response, 3 thyroidectomy, evidence of hyperplasia	8+ wks	1 responded, then reverted to previous condition, 4 had toxic symptoms at beginning, but subsided	j
12 adults	150 mg/day	Healthy	Reduced thyroid function in 11/12	4 wks	Clinically euthyroid	k
11 adults	25 mg/day 4 wks 50 mg/day 8 wks	Anemia, complicated by severe renal failure	23% decrease in hematocrit, ~ 40% had hearing loss	8 wks	3 more didn't finish regime; enteric-coated tablets	l
18 adults	50 mg/day	Anemia, complicated by severe renal failure	Significant increase in hemoglobin, but returned to normal after treatment stopped	12 wks	4 more didn't finish regime; some had nausea, vomiting, constipation; serum Co stabilized at 40-100 µg/dl with continuous therapy of 8 wks or more, declined rapidly with end of therapy; enteric-coated tablets	l
23 adults	50 mg/day	Renal failure (8 anephric)	1/2 had hemoglobin increase	12 wks	4 had nausea and vomiting 1 died of congestive heart failure (Co in heart 25-80 x normal)	l

Table 7.8. Continued

[a] R.T. Gross, J.P. Kriss and T.H. Spaet, "Hematopoietic and Goitrogenic Effects of Cobaltous Chloride in Patients with Sickle-cell Anemia," Am. J. Dis. Children 88: 503-504, 1954.

[b] J.P. Kriss and W.F. Carnes, "Hypothyroidism and Thyroid Hyperplasia in Patients Treated with Cobalt," J. Am. Med. Assoc. 157: 117-121, 1955.

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[d] J.F. Robey, P.M. Veazey and J.D Crawford, "Cobalt-induced Myxedema. Report of a Case," N. Eng. J. Med. 255(20): 55-57, 1956.

[e] J.L. Chamberlain, III, "Thyroid Enlargement Probably Induced by Cobalt," J. Pediatrics 59: 81-86, 1961.

[f] T. Sederhold, K. Kouvalainen and B.A. Lamberg, "Cobalt-induced Hypothyroidism and Polycythemia in Lipid Nephrosis," Acta. Med. Scand. 184: 301-306, 1968.

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[h] R.G. Holly, "Studies on Iron and Cobalt Metabolism," J. Am. Med. Assoc. 158: 1349-1352, 1955.

[i] C.H. Jaimet and H.C. Thode, "Thyroid Function Studies on Children Receiving Cobalt Therapy," J. Am. Med. Assoc. 158: 1353-1355, 1955.

[j] E. Pimentel-Malaussena, M. Roche and M. Layrisse, "Treatment of Eight Cases of Hyperthyroidism with Cobaltous Chloride," J. Am. Med. Assoc. 167: 1719-1723, 1958.

[k] M. Roche and M. Layrisse, "Effects of Cobalt on Thyroidal Uptake of I-131," J. Clin. Endocrin. 16: 831-833, 1956.

[l] "Cobalt in Severe Renal Failure," Lancet II (7975):26-27, 1976.

between 1 and 5 mg/kg.[153] The mechanism by which cobalt induces polycythemia is not clearly defined. Several studies have shown that *in vivo* or *in vitro* administration of cobalt reduces cellular respiration of liver, kidney, diaphragm, and skeletal and cardiac muscle.[157,158] Isolated dog kidneys perfused with blood containing cobalt showed significant increases in erythropoietin levels.[159] Erythropoietin release from the kidney is believed to stimulate erythropoiesis in other species also.[160] Similar effects (a stimulation of erythropoietin secretion by the kidney) were observed when the oxygen levels in the perfusing blood were reduced. In simple terms, the polycythemic response may be the body's way of compensating for the tissue hypoxia caused by cobalt administration.

The polycythemia induced by cobalt is accompanied or preceded by an increase in circulating reticulocytes in rats, dogs and humans.[154,161-164]

Cardiovascular System

Evidence that cobalt has been a factor in the development of heart disease in chronic beer drinkers (consuming beer which contained cobalt) gained much attention from the mid-1960s through the 1970s. Since the initial outbreak of heart disease following the addition of cobalt to beer, there have been numerous investigations on the role of cobalt in the etiology of this disease.[165-173] The clinical description of the disease consists of massive pericardial effusion, low cardiac output, and raised venous pressure accompanied by polycythemia.[169] Since these early investigations, cobalt has been shown to be cardiotoxic to the rat,[166,167,170] rabbit[168] and pig[165,172] at fairly high doses. Lin and Duffy[170] have investigated the cardiotoxic response of rats to intraperitoneal administration of $Co(NO_3)_2$ at 5 mg Co/kg body weight. The two major changes in the organelles of myofibers of rat heart were segmental contraction bands in the myofibrils and swelling of the mitochondria with fragmented and shortened cristae. The study done by Grice et al.[167] correlated changes in electrocardiography with the pathology produced in the rat heart after cobalt administration. Multiple intraperitoneal injections of $CoSO_4$ at 6 mg/kg produced myofiber degeneration, interstitial edema and cell infiltration. These pathological changes were accompanied by increased T wave amplitude and a dimunition in the R wave potential. If no changes in the EKG were observed the histopathological changes were minimal to negative. In a previous study, Grice et al.[166] administered $CoSO_4$ orally to rats at varying dose levels ranging from 3 mg/kg to 100 mg/kg per day. They investigated the effect of diet on cobalt toxicity using three different diets: (1) casein used as the protein source, (2) dried cooked peas (a protein source deficient in sulfur-containing amino acids), and (3) wheat gluten (a protein deficient in lysine). The rats given the diet containing wheat gluten demonstrated a toxic response to cobalt at the lowest dose level (3 mg/kg). At dose levels above 6 mg/kg, an increasing fraction of rats demonstrated a cardiotoxic response to cobalt administration. The histological lesions in the heart consisted of necrosis of the myofibers with prominent vacuolation. Necrotic areas were sometimes replaced with fibrous tissue. The lesions produced were centered around the apex of the left ventricle and the lower part of the intraventricular septum. When higher doses were administered the entire myocardium was affected. Old age, inadequate diet and increasing doses augmented the cardiotoxic response to cobalt. Hall and Smith[168] found a similar cardiotoxic response in rabbits exposed to 15 to 25 mg/kg daily by subcutaneous injection for 9 to 13 days. Cobalt sulfate administered orally to pigs (100 mg/kg daily for 3 days) produced mitochondrial and myofibrillar lesions in the heart.[172] One interesting observation in this investigation was the fact that a group of stress-susceptible pigs exposed to cobalt showed more severe myocardial damage

at similar dose levels. An attempt was made to quantitate the myocardial damage inflicted by excess cobalt by grading the severity of histopathologic myocardial injury. The results of this grading system showed the stress-susceptible pigs to be 4 to 5 times more sensitive to cobalt than the ordinary controls. Only one reference to fatal cardiomyopathy due to industrial cobalt exposure was noted in the literature.[174] The mode and level of cobalt exposure were not specified, but the clinical features and necropsy findings resembled beer drinkers' cardiomyopathy. Tissue levels of cobalt were measured in the case of the cardiomyopathy and in two control cases. The heart from the cobalt-exposed individual contained 37 μg Co/100 g wet tissue compared with 5 and 2 μg Co/100 g for the control cases. Cobalt levels in the hearts of patients dying from beer drinker's cardiomyopathy had a mean ± S.D. of 69 ± 32 μg/100 g[171] and 48 μg/100 g.[169] Pigs exposed to 20 mg/kg Co (as $CoCl_2$) in their diet daily for seven weeks had 450 μg Co/100 g in their heart.[171]

In summary, the cardiotoxicity of cobalt is well documented when very high doses are repeatedly given. The myocardial lesion produced involves degeneration of the myofibers, with subsequent replacement by fibrotic tissue. Inadequate nutrition and a predisposition to heart problems seem to potentiate the cardiotoxic effect of cobalt. The electrocardiogram appears to be a sensitive indicator of cobalt-induced heart injury.

Thyroid

The ability of cobalt to affect thyroid function was first reported by Gross et al.[175] After treatment of anemic children with oral cobalt salts he observed the development of goiter and hypothyroidism. Initially his conclusions were refuted by investigators who were unable to reproduce his results in other species.[176-178] However, since then, other investigators have confirmed Gross' initial observations[137,179-183] after administering cobalt salts orally and parenterally.

The thyroids of one-day-old chicks showed decreased thyroidal ^{131}I uptake and increased phosphorus uptake after intraperitoneal injections of cobaltous ion at 4 mg/kg to 104 mg/kg.[150] Intraperitoneal administration of 8 mg/kg $CoCl_2$ to guinea pigs induced thyroid hyperplasia with a decrease in the amount of colloid within one month.[184] The effect of oral cobalt salts on human thyroid function has been variable. Administration of $CoCl_2$ to 78 pregnant women in dosages ranging from 60 to 100 mg daily for 90 days did not result in goiter formation.[188] The same author was also unable to induce goiters in mice or rats given dosages up to 40 mg/kg per day. Radioiodine tests were not done in the studies by Holly.[176] Conversely, the thyroidal uptake of radioiodine was decreased in 12 adults given $CoCl_2$ in a dosage of 50 mg three times daily for two weeks. By the second week of cobalt administration the thyroidal ^{131}I uptake values were near zero. Paley et al.[137] administered $CoCl_2$ to patients orally at 37.5 mg daily. They found a marked depression of thyroidal ^{131}I uptake following oral administration of $CoCl_2$ in 50% of the patients tested. Sederholm et al.,[156] saw the development of hypothyroidism and goiter in a 12-year-old patient administered cobalt for the treatment of anemia. The inconsistent results obtained in humans after oral administration of soluble cobalt salts can possibly be explained by the work of Paley and Sussman,[185] who found that the absorption of cobalt is generally diminished when given after a meal or if the cobalt is pretagged to protein. These results suggest that cobalt absorption takes place in the gastrointestinal tract above the region of protein digestion. The possibility exists that a wide variation in cobalt absorption results when conditions during oral administration are not standardized.

Hyperlipemic Effect

The ability of cobalt to induce a sustained hyperlipemia after repeated parenteral administration of a soluble cobalt salt has been demonstrated in the rabbit,[186-192] rat[193] and fowl.[194] The levels of cobalt necessary to induce hyperlipemia were toxic, as evidenced by a body weight loss as compared with controls.[186,187,191,193] In the investigation of Caren and Carbo[188] with rabbits, the intravenous administration of $CoCl_2$ (50 mg/kg) produced an elevation of plasma cholesterol 2 to 5 times control values. The increase in plasma cholesterol started 24 hours after cobalt treatment, reached a peak value by the second or third day, and gradually returned to pre-treatment levels by about the seventh day. Caplan and Block[187] treated rabbits with $CoCl_2$ (40-50 mg/animal) and observed a rise in all lipid components of the serum (i.e., cholesterol, phospholipids, triglycerides and fatty acids). The serum lipid fraction that increased most consistently and to the greatest extent was the triglyceride fraction. Brody et al.[186] treated rabbits with ten daily intramuscular injections of $CoCl_2$ (20 mg/kg), and found total plasma lipids elevated almost fourfold over control values. Mukherjee et al.[191] used an identical cobalt exposure and also obtained serum lipid values significantly higher than control values. Zarafonetis et al.[192] induced hyperlipemia in rabbits after daily intramuscular injections of 25 mg/kg $CoCl_2$. After the first week of injections, total serum lipids increased over four-fold in the cobalt-treated rabbits. Fiedler and Hoffman[190] administered cobalt to rabbits in the form of amino acid complexes at a dosage of 5 mg/kg body weight. A marked increase in total lipids (phosphatides, free fatty acids and cholesterol) was observed in the serum or plasma of rabbits treated with cobalt amino acid complexes. Eaton[193] injected rats subcutaneously with 2 μmoles/100 g body weight of $CoCl_2$ in two 5-day courses separated by a nine-day period of no injections. Serum lipid concentrations were determined five days after the last cobalt injection. All lipid components of the blood of cobalt-treated rats showed a significant increase. The plasma triglyceride concentration increased to the greatest extent, attaining levels over twice that of the controls. The serum free fatty acid and cholesterol levels were also significantly higher in the cobalt-treated rats. The plasma of cobalt-treated rats usually became lactescent after the first five injections and was consistently lactescent at the end of the 24-day treatment period. In a more recent investigation, Shabaan et al.[195] described the results of daily subcutaneous injections of $CoCl_2$ at a dose of 4 mg $CoCl_2$/100 g body weight in rats. In addition to the production of fibrosarcomas in a significant fraction of the rats (this will be discussed in a later section), the experimenters observed hyperlipemia lasting for 12 months in those rats receiving subcutaneously injected cobalt.

The case for induction of hyperlipemia in humans given cobalt (for the treatment of anemia) is not so well documented. Robey et al.[182] observed a 17-month-old girl who had received a cobalt-iron preparation for the treatment of anemia. The child was given the cobalt in a dosage of 4 mg/kg/day for a total of 12 weeks. At the end of this period the child's serum was grossly lipemic, the cholesterol level being 694 mg/100 ml. Kriss et al.[196] reported on the effect of cobalt therapy used to treat sickle-cell anemia in a six-year-old boy. During the third month of therapy (100 mg/day) the total serum lipids were almost four times the level measured four weeks after discontinuing the cobalt therapy.

The ability of cobalt to induce hyperlipemia in rabbits, rats and fowl has been substantiated with parenteral administration of toxic levels of a soluble cobalt salt. The question of whether cobalt produces hyperlipemia in humans when administered orally is not completely answered. There is an indication that this may be the case, but further clinical investigations examining the role of cobalt in producing hyperlipemia will be needed to answer this question.

Tumorigenic and Carcinogenic Effects

At present there is no epidemiological evidence that occupational exposures to cobalt are associated with increased risk of cancer. There is, however, experimental evidence showing that elemental cobalt powder, CoO and cobalt sulfide (CoS) are capable of inducing local sarcomas following parenteral administration to rabbits and rats.[197]

Intramuscular injection of pure cobalt metal powder (28 mg/rat) into the thigh muscle of 60- to 90-day-old rats produced malignant tumors in 17 of 30 rats so treated.[198] All tumors occurred locally at the injection site; a high proportion were rhabdomyofibrosarcomas and the remainder sarcomas of various types. A later paper[199] by the same investigator describes the histogenesis of the malignant tumors induced by the same method. Gilman et al.[200,201] reported on the production of local sarcomas induced in 50% of Wistar rats after intramuscular injection of 30 mg CoO. The majority of tumors produced in this manner were rhabdomyosarcomas. However, in the same investigation, cobalt failed to induce tumors in 92 Swiss mice injected intramuscularly with 10 mg of the same preparation. Gilman[201] also administered a single 20-mg dose of CoS into both left and right thigh muscles of Wistar rats, resulting in a 96% incidence of local tumors. On autopsy it was observed that the group of rats injected with CoS had a larger percentage of metastases than the rats receiving CoO. Shabaan[195] treated 20 rats with $CoCl_2$ by injecting a 4 mg/100 g body weight dose subcutaneously in two courses of five days, separated by a nine-day interval. Of the 11 rats that survived the first year, eight developed fibrosarcomas; in four of these rats the tumor was far removed from the injection sites. In a second experiment of similar design, six of the 16 rats surviving eight months after cobalt treatment had developed tumors of the same kind as those found in the first experiment.

Experimentally, the ability of different cobalt compounds to induce tumors locally at the injection sites in rats is documented. The lack of epidemiological evidence correlating cobalt exposure with an increased risk of cancer and the fact that only high parenteral doses of cobalt were able to induce local tumors in rats (but not mice) would suggest that cobalt is a very weak carcinogenic agent.

Effects on Drug Metabolizing Enzymes

The ability of metals to modify the activity of heme-containing microsomal drug-metabolizing enzymes has been investigated. *In vitro*, nickel, cobalt and iron have been found to impair cellular heme-dependent metabolism.[202] Cobalt specifically inhibits the heme biosynthetic pathway and induces the degradative pathway.[202-206] Cobalt also reduces cellular glutathione content, and may alter the activity of glutathione-dependent enzymes. The respiratory activity of tissues is also depressed by toxic levels of cobalt[157,158,202] and indirectly reduces the drug-detoxifying ability of cells.

Respiratory System

Investigations into adverse reactions of the respiratory system to cobalt were stimulated by increased incidences of pulmonary distress observed among workers in the cemented tungsten carbide industry.[207-210] In the production of hard metal alloys from tungsten carbide and cobalt, there are numerous opportunities for worker exposure to dusts, the potentially most harmful of which are silicon carbide and cobalt.[211] The symptoms (dyspnea, tight chest, productive cough)

occur quite suddenly after a variable latency of 2 to 20 or more years.[132,211] The incidence rate is very low (about 2% of British hard metal shop workers).[132] Inhalation of cobalt (as metal dust and fume) has been implicated as the etiological factor responsible for the chronic interstitial pneumonitis seen among these workers[14] although the carbides of tungsten, tantalum and titanium were also commonly present in the exposure atmosphere.[209] A U.S. Public Health Service study reported by Fairhall et al.[207,208] on the working conditions and medical status of 1802 workers in the cemented tungsten carbide industry revealed average cobalt concentrations of 1 to 2 mg/m^3 in the powder-pressing operation and a significant increase in conjunctivitis, gingivitis and irritation of the upper respiratory tract among the workers studied. The average work-area level in Great Britain is now 0.02 mg/m^3, which is quite low compared to the TLV of 0.1 mg Co/m^3. Another technical and medical investigation of approximately 200 workers exposed to cobalt in the latter industry failed to define the agent responsible for the chronic lung fibrosis seen in a small percentage of the exposed workers.[210] These same authors found[212] that intraperitoneal injection of cobalt dust or a powder of mixed metal compounds used in the manufacture of cemented tungsten carbide tools did not show any tendency to produce fibrosis in the peritoneum of guinea pigs.

An acute response of the respiratory tract of experimental animals to cobalt metal dust has clearly been shown by Schepers.[213] In his studies, guinea pigs were exposed by intratracheal injection to 50, 25, and 10 mg of cobalt metal dust. The acute response of the guinea pig lung to the 50- and 25-mg dose was marked pulmonary edema with moderate alveolar wall hyperemia. At eight months after exposure the lungs showed diffuse perivascular cellular infiltration, with eosinophil cells appearing in large numbers within the alveoli. At this same time, regional obliterative bronchiolitis with peribronchiolar fibrosis and arteriolar spasm were observed. The acute response to CoO was not nearly as severe.[214] By 12 months after exposure of guinea pigs to 150 mg of cobaltic oxide by intratracheal injection, the focal, peribronchial, subacute inflammatory reaction observed earlier was no longer present. In another study Stokinger and Wagner[215] investigated the possibility of finding a test which would show pretoxic signs of exposure to cobalt. Rabbits and dogs inhaled cobalt fume (1.5 mg/m^3 for 6 hr/day, and intermittently, each third week for a total of 24 weeks) or cobalt metal (5 mg Co/m^3 for 6 hr/day alternating three weeks for a total of 30 weeks). An elevation in the α-globulin fractions of the serum proteins and the serum neuraminic acid (an acidic polysaccharide chiefly associated with the α-globulin fractions) was observed at a level of cobalt exposure below that necessary to produce an erythropioetic effect, for hemoglobinogenesis, for pancreas cell injury or for generalized toxicity. However, the serum protein changes did not occur uniformly in all animals and the changes were not of great magnitude. The serum response to cobalt exposure was delayed (5-26 weeks); and serum levels sometimes returned to normal with continued exposure.

In summary, while cobalt has been implicated as the etiological factor responsible for the pneumoconiotic lesions seen in workers in the cemented tungsten carbide industry, the presence of other aerial contaminants has obscured the importance of cobalt in producing the pulmonary reaction. Animal experiments have documented the ability of cobalt metal and CoO dust to produce an acute pneumonitis. The pulmonary response is thought to be benign in the case of exposure to cobaltic oxide; 12 months after exposure, the histopathology of the lungs of exposed animals had returned to normal.

7.4.3 Protection, Remediation and Diagnostic Techniques

Protection from exposure to cobalt-containing dusts has in the past been afforded by the use of dust collectors on equipment such as grinding wheels used in tool manufacture[209] and by careful ventilation of workroom areas.[208] Even with these precautions, measurable levels of cobalt were found in all areas of three plants studied[208] in which cemented tungsten carbide tools were made. Cobalt levels measured ranged from 3 or 4 μg Co/m^3 to 79 mg Co/m^3.[208] In nickel-electrode manufacturing for the Ni/Fe and Ni/Zn batteries, prevention of exposure to vapors from the cobalt-containing baths is presently afforded by ventilation of the tanks to a fume scrubber and by use of full face masks, aprons and long rubber gloves.

Removal from exposure has been the main mode of therapy for persons exposed to cobalt dusts.[209] Measurement of urinary cobalt levels should indicate the extent of current exposure to cobalt, as it has clearly been shown that cobalt in the form of the oxide or the soluble chloride is eliminated rapidly from the body and that urinary excretion is the predominant route of elimination.[134,135,142,144,149]

7.5 LEAD

Environmental releases of lead expected to result from the expansion of lead/acid battery manufacture for EV use, and the related emissions from lead mining, milling, and primary and secondary smelting have been discussed in Sections 2 and 5. The production of 3.0×10^6 lead/acid battery-powered EVs by the year 2000 will require industries that are 55%, 44%, and 94% of present U.S. capacities (1973) for primary smelting, secondary smelting, and lead/acid battery manufacturing, respectively (see Tables 2.11, 2.28, and 2.20). Significant impacts on occupational exposure and environmental release of lead can thus be anticipated.

The environmental releases expected per day per MWh of electric vehicle battery installed are summarized in Table 7.9 for each of the processes involved in battery production and use. As can be seen, the greatest amount of lead released will be in solid form, as slag or sludge to

Table 7.9. Process Emissions of Lead for Lead/Acid EV Battery Production and Use[a]

	Lead Emissions,[a] (kg/day)/MWh		
Process	*In Air*	*In Water*	*As Solid Waste*
Primary Lead Production			
Mining and milling	0.044	$< 2.3 \times 10^{-4}$	0.22
Smelting and refining	0.017	5.8×10^{-6}	0.20
Secondary Smelting	0.012	none	0.077
Battery Manufacture	0.0014	2.9×10^{-4}	0.0053 to 0.010
Battery Breaking	--	1.4×10^{-6}	3×10^{-4}
TOTAL	0.074	5.3×10^{-4}	0.51

[a] *Data from Tables 2.3, 2.5 through 2.10, 2.18, 2.19, 2.22, 2.24, 2.25, and 2.26.*

be put into secured land fills or old mines. The final net effect of EV use on human exposure to lead will depend on a combination of factors, including increased use of lead for battery manufacturing, replacement of gasoline-powered vehicles by electric vehicles, and use of alternate power sources, such as coal- or nuclear-based electrical energy, for EV charging.

In the present report, the metabolic and biological effects of lead on the human system in the physical and chemical forms pertinent to the battery industries are presented where such data are available. The two major exposure pathways covered are inhalation and ingestion.

7.5.1 Metabolic Uptake, Retention and Excretion

Absorption through Inhalation

Relevance to Battery Industries. The major species of lead accounting for occupational and environmental exposure of humans to lead by inhalation due to the processes associated with lead/acid battery manufacture and use are lead, lead sulfide (PbS), lead oxide, and lead fume. Workers in the lead mining and milling industries are exposed by inhalation to lead sulfide dust (PbS) present in galena, the ore which accounts for most of the lead mined in the U.S. Air emissions of lead to the environment from mining and milling consist mainly of fugitive dusts (see Table 2.3). At the primary smelter, PbS is oxidized to lead oxide and then reduced to metallic lead. At the primary smelter, particulates from the blast furnace and also fugitive dust emissions account for most of the environmental air releases of lead (Table 2.8). Secondary smelting involves retrieval of lead from scrap lead, recycled batteries, etc. High temperatures are achieved during both primary and secondary smelting, resulting in exposure of workers to lead fume. According to published reports, lead smelting results in higher levels of lead in the blood of smelter workers than mining and milling employees.[216-218] In addition, lead smelters have been reported to have a measurable impact on the lead exposure of persons living in the vicinity of the smelter.[219-221] During battery manufacture, occupational exposure to lead oxide and at some stages to lead fume occurs, resulting in elevated levels of lead in the blood of employees, in some cases similar to those seen in smelter workers.[222] Air emissions of lead oxide, and some lead fume and lead sulfate ($PbSO_4$) have been reported during battery manufacturing (Table 2.18).

Initial Lung Deposition following Inhalation. The fraction of inhaled lead initially deposited in the lungs of persons exposed to known levels of lead has been reported by several experimenters. This fraction depends on both particle size and the rate and depth of respiration. In Kehoe's studies,[223] men were exposed to lead oxide (Pb_2O_3) at 105 $\mu g/m^3$, a level equal to the threshold limit values (1977) for inorganic lead fumes and dusts in workroom air adopted by The American Conference of Governmental Industrial Hygienists.[224] The Pb_2O_3 was generated by burning tetraethyl lead. The group exposed to particles with a mass median equivalent diameter (MMED) of 0.26 μm deposited 36% of the inhaled lead in their lungs, while the group exposed to particles of 2.9 μm MMED deposited 46%. Nozaki[225] studied particle deposition from lead fumes at very high concentrations of lead (10 mg/m^3) and particles carefully controlled in size, varying from 0.05 to 1.0 μm MMED. With the latter increase in particle size, the amounts deposited in the airways increased from 42.5% to 63.2%, when respiration was slow and deep (10 respirations/min). When respiration was fast and shallow (30 respirations/min), amounts deposited increased from 21.0% to 35.5% over the same particle size range. Studies of initial lung deposition of lead in humans in the chemical forms encountered in battery industry and

related activities are few. The studies that do exist indicate that from 30% to 60% deposition may be encountered for the smaller particle size ranges (0-3 μm MMED), with fractional depositions increasing with increasing particle size and with decreasing rate of respiration.

Translocation from the Lung. The fate of lead in the body following its deposition in the human lungs is not very well understood. From Kehoe's studies, it appears that inhaled Pb_2O_3 of 0.25 μm MMED was nearly completely retained, as he saw only a small increase in fecal lead excretion following exposure.[223] In contrast, when particles were larger (approximately 2.9 μm MMED), inhalation for many weeks at 150 μm Pb/m^3 resulted in a substantial increase in fecal excretion. This increase was attributed to clearance of lead from the lungs with subsequent swallowing, and accounted for approximately 40% of the lead deposited in the airways. Thus, particle size not only influences the initial fractional deposition of lead, but also affects the mechanism of clearance from the lung.

Because alveolar macrophages participate in the clearance of particulates from the lung, and because these cells are damaged by inorganic lead *in vitro*[226] and *in vivo* in rats and guinea pigs at 10 and 150 μg Pb/m^3,[227] the toxicity to lung cells of inhaled lead compounds must also influence their clearance rate from the lungs. The high retention of small lead particulates indicated in Kehoe's studies may thus have been influenced by the toxicity of lead on lung-clearance mechanisms at 150 μg Pb/m^3.

Human absorption of lead through inhalation of lead as it appears in ambient air has been reported by Rabinowitz in a series of studies using stable isotopes of lead.[228] In these studies, five men living in a metabolic unit and breathing air with 0.91 μg Pb/m^3 were calculated to have absorbed an average of 14 μg Pb per day through respiration. Daily exposure through inhalation can be calculated to have been about 35 μg Pb/day for subject B. This subject was estimated to have absorbed 17 μg Pb/day through respiration, or 48% of the lead inhaled. Since initial deposition of lead is estimated to be close to 40% of that inhaled, total retention in the body of lead deposited in the lungs is also indicated by Rabinowitz's data.

In summary, then, the translocation of lead from the human lung to the body appears to be substantial in the case of small particles of Pb_2O_3 (Kehoe's studies) and of lead particles inhaled from the ambient air (Rabinowitz's studies).

Absorption through Ingestion

Relevance to Battery Industry. Among workers industrially exposed to lead, ingestion is a potentially important mode of exposure to the same forms of lead as inhalation. Even when levels of lead in air are kept below a given standard, personal hygiene must also be controlled so as to restrict intake through ingestion (personal habits will vary with the individual and with the type of job supervision). Ingestion of lead present in the environment as a result of lead/acid battery production and use is also a potential problem as 89% of the lead released from industries related to battery production is in solid form (Table 7.9). Leaching of disposal sites by acid rain could thus result in elevated levels of lead in surrounding water supplies.

Gastrointestinal Absorption in Humans. Several studies on gastrointestinal absorption of lead in humans have been conducted. Long-term balance studies by Kehoe[223] showed that, with a

normal diet as the source of lead, the daily excretion of lead into the urine was close to 10% of the intake from food and beverages. Because a portion of this urinary excretion must have originated from the inhalation of airborn lead, somewhat less than 10% of the ingested lead must have been absorbed, on the assumption that (1) lead uptake and excretion were equal in the people studied and (2) gastrointestinal excretion of lead was small compared to 10%. Rabinowitz[228] reported a mean of 9.8% gastrointestinal absorption of a Pb-204 nitrate tracer in five human subjects. This value represents the difference between tracer intake and fecal output, with a correction made for gastrointestinal excretion of the tracer lead. Lead absorption levels following ingestion of the insoluble lead oxides and sulfides encountered in the industries of concern to this report can only be said to be less than or equal to the 10% reported above for other forms of lead. In addition, a greater fraction of the lead encountered through inhalation enters the body than that encountered through ingestion.

It should be noted here that the contribution of dietary lead to daily intake in infants and children appears to be much higher than for adults. Enhanced absorption In the young rat has been observed[229,230], with 55% to 83% of a tracer dose absorbed during the period before weaning. Human studies by Alexander[231] and Ziegler et al.[232] indicate high levels of lead absorption in children (53%) and infants (42%).

Tissue Distribution and Retention

Human autopsy data have revealed that, of the total body lead, approximately 95% is in the skeleton,[233-236] indicating the well-known high affinity of lead for bone. The highest concentration of lead in the body is also found in bone, with an intermediate level in the liver, kidney, and aorta, and a low level in muscle and brain. The reports listed above also indicate that the concentration of lead in bone increases with age, and reflects the accumulation of lead throughout life. In contrast, the soft tissues, including blood, do not appear to increase in lead concentration with age past early adulthood, but reflect more closely the current exposure conditions.

From his studies using stable isotopes of lead nitrate, Rabinowitz[228] described three body pools of lead, with the largest by far consisting of lead in bone (see Fig. 7.6). A second, much smaller pool consisted of blood and tissues in rapid equilibrium with blood, giving rise to lead in urine. A third pool consisted of the soft tissue and possibly the more active parts of the skeleton, giving rise to lead in bile, hair, sweat, nails, etc. Other models identify only two general pools of lead, distinguishing between bone, which accumulates lead, and other organs and systems, whose lead levels tend to stabilize to various degrees in early adult life.

The size of the smaller, exchangeable body burden of lead is considered important in producing the toxic responses to lead, while the skeletal pool functions to sequester lead, decreasing its bioavailability. Although mobilization of the large bone stores of lead into the body's exchangeable pool may occur, the conditions under which this might be of concern have not yet been explored.

Excretion

Elimination of lead from the human body is thought to be mainly through the urinary and the gastrointestinal tracts. Relative contributions of various excretion routes in people not

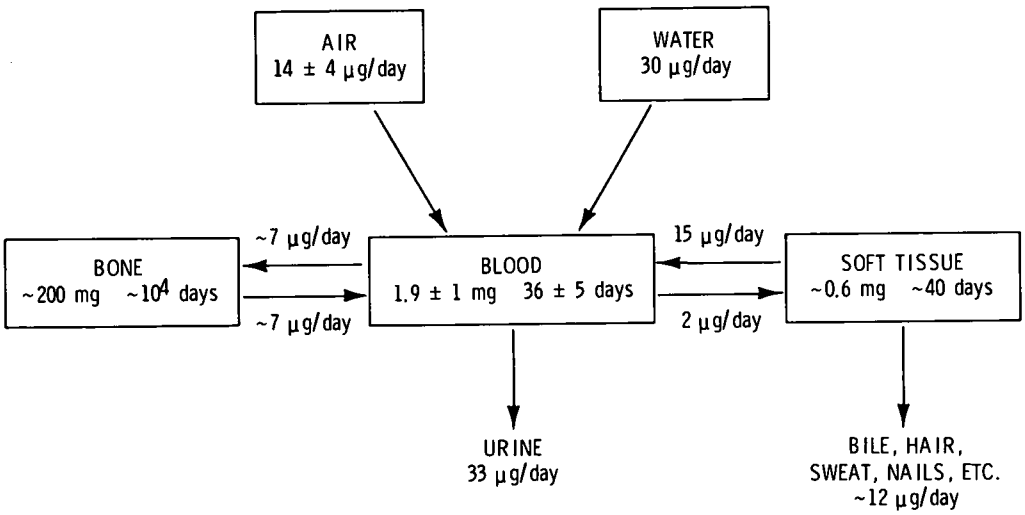


Fig. 7.6. A Model of Lead Metabolism in Normal Adults Representing the Quantities, Mean Lifetimes, and Exchange Constants of a Representative Lead Body Burden (from G.B. Morgan and E.W. Bretthauer, *Metals in Bioenvironmental Systems*, Anal. Chem. 49(14): 1210A-1214A, 1977.)

industrially exposed to lead have been estimated by Rabinowitz et al.[228] using a Pb-204 nitrate tracer. A mean of 36 µg Pb/day excreted into urine was measured for five humans, and a mean of 9 µg/day excreted through endogenous fecal secretions was estimated for three of these subjects. This excretion level for lead in urine of humans agrees with that observed by others.[237] The 4:1 ratio of urinary to fecal excretion for humans differs from the 2:1 ratio observed in the baboon.[238] Data in rats[239] and sheep[240] indicate a considerable species variation in the routes of lead excretion; biliary and endogenous fecal excretion of lead is greater in these species than urinary excretion.

The nature of the lead excreted into urine has been found to differ between persons with normal exposure to lead and lead-industry workers.[241] All of the lead in the urine of normally exposed individuals can be precipitated by such agents as oxalate, phosphate or carbonate; in contrast, only one-third to two-thirds of the lead in the urine of lead workers is available for precipitation. A stable complex of lead appears to be present in the urine of exposed workers.

7.5.2 Toxic Responses and Human Health Implications

The primary literature on lead toxicity is vast, and for purposes of this assessment three major reviews[242-244] of the literature as it relates to human exposure are used as the basis to present the major known toxic responses to lead. These responses are described independently of exposure mode. Instead, where data are available, those blood lead levels observed to accompany the toxic effects described have been specified. Blood lead levels are indicators of current exposure, but not necessarily of the history and magnitude of past exposure. Blood lead levels have been seen to rise quickly in response to changes in lead exposure,[228,245,246] reaching a new plateau by 60[246] to 110[228] days after a change in exposure. In addition, it has been seen experimentally in rats[247,248] that exposure to lead in early life can produce toxicity symptoms that persist into adulthood even after blood lead levels have returned to normal. Blood lead levels are thus given here as partial indicators only of anticipated toxic responses to lead.

Hematopoietic System

The response to lead most widely monitored in man has been its effect on the hematopoietic system. Mild hypochromic, and sometimes microcytic, anemia accompanied by shortened red blood cell life span, reticulocytosis, and basophilic stippling of peripheral blood cells are characteristic responses to lead exposure.[244] Interference by lead in the biosynthesis of both heme and globin has been observed.[244] An accumulation of nonheme iron and protoporphyrin 9, or free erythrocyte protoporphyrin (FEP), in red blood cells, an increase in δ-aminolevulinic acid (ALA) in serum, and an increase in ALA and coproporphyrin III (CP) in urine are signs of lead-induced alterations in heme biosynthesis. The normal sequence of reactions in heme biosynthesis and the consequences associated with lead exposure are indicated in Figure 7.7. The most sensitive indicator of blood lead levels is the inhibition of δ-aminolevulinic acid dehydratase (ALAD) in red blood cells. This inhibition occurs, however, at blood lead levels below those which normally accompany other responses to lead exposure. The average no-effect blood lead level for inhibition of ALAD was found to be about 15 μg/100 ml in children[249] and lead workers.[250] Others[251] have found a correlation between blood lead and ALAD inhibition even below 10 μg/100 ml.[251] Another early indicator of lead exposure is the increase in erythrocyte protoporphyrin (FEP) concentration. This increase has been observed at lower blood lead levels than the increase in ALA in urine[242] and in women and children, appears to occur at lower blood lead levels than in men. In the range 31 to 40 μg Pb/100 ml blood, increases in FEP over normal levels were observed in 19% of adult male subjects, while 90% of the women in this range showed this increase. Decreases in hemoglobin concentration as well as increases in FEP have been measured at been blood lead levels below 80 μg/100 ml.[218]

Nervous System

Central Nervous System. Symptoms of acute lead encephalopathy include dullness, restlessness, irritability, headaches, muscular tremor, hallucinations, loss of memory and ability to concentrate. These symptoms may lead to delirium, mania, convulsions, paralysis and coma. Its incidence is seen more in children than in adults, and is seldom seen in children at blood lead levels below 120 μg/100 ml.[243] In about 25% of the children with attacks of acute encephalopathy, permanent long-term effects occur, involving impaired motor coordination, lack of sensory perception, and inability to concentrate, in the milder cases, and, in severe cases, cortical atrophy, hydrocephalus, convulsions, seizures and idiocy. Apparently the incidence of such sequelae can be decreased by chelation therapy prior to the onset of encephalopathy.[243]

One major current social concern is that young children with blood lead levels from 40 to 80 μg/100 ml may be experiencing subtle neurological damage without signs of lead encephalopathy, and many investigations into this possibility have been reported. Two comprehensive reviews of this topic are of interest.[243,252] The first report[243] states that

> among the major studies that are widely cited in regard to this issue, those of de la Burde and Choate[253,254] Perino and Ernhart,[255] Albert et al.[256] and Landrigan et al.[257] suggest significant effects of asymptomatic low-level lead exposure. In contrast, the studies of Kotok,[258] Lansdown et al.,[259] and McNeil et al.[260] report generally negative results. Two other studies, by Landrigan et al.[261] and by Kotok et al.,[262] although not reporting clearly statistically significant differences between moderately lead-exposed subjects and control subjects, nevertheless report certain findings that are highly suggestive of a relationship between moderate lead exposure and cognitive impairment.

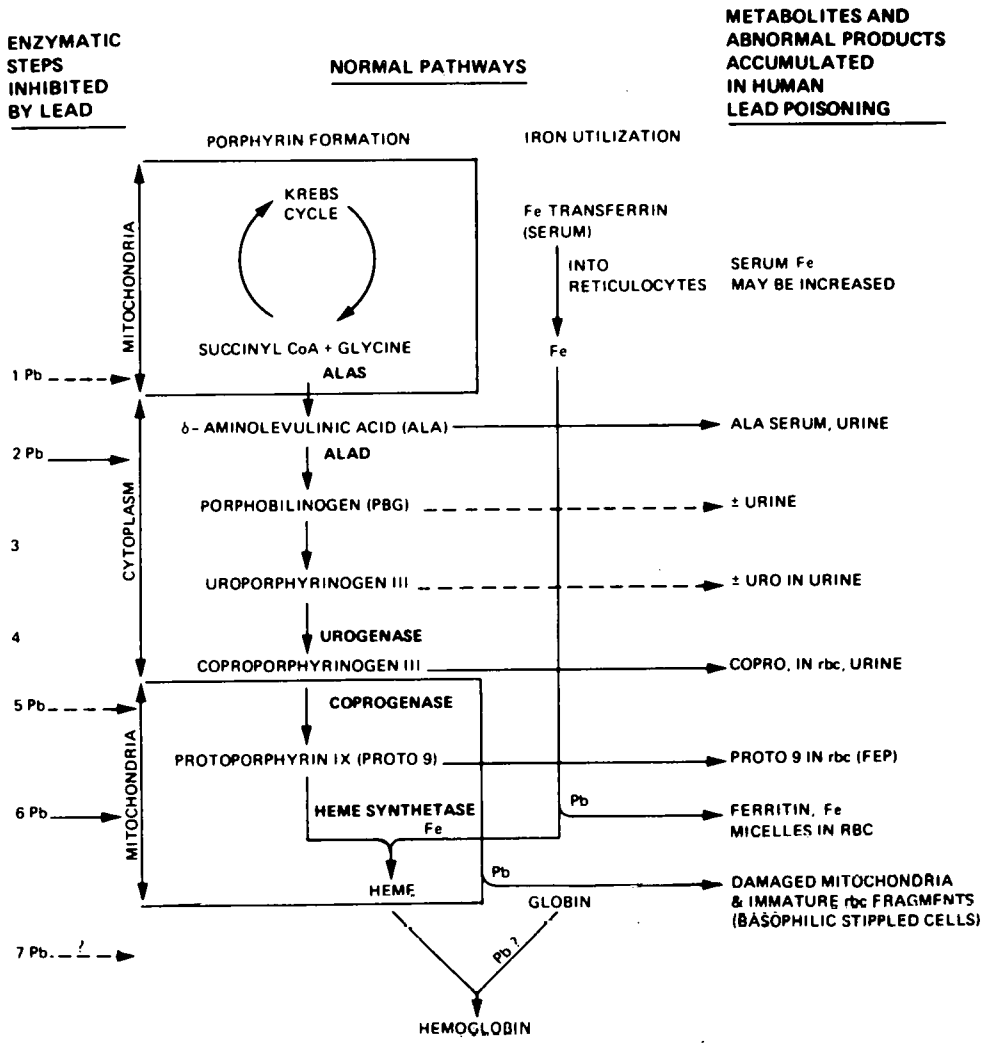


Fig. 7.7. Lead Interference with the Biosynthesis of Heme. A schematic representation of the pathway for heme biosynthesis demonstrating known and postulated sites of interference by lead, normal metabolites, and abnormal products accumulated as a result of poisoning. (From *Lead. Airborne Lead in Perspective*, Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Academy of Sciences, National Research Council, Washington, D.C., 1972.)

After careful analysis of many of the same articles referred to in the first review, the second report[252] concludes that "the data presently available are still inadequate to conclude that subtle psychological, emotional, and neurological sequelae occur in children as a result of lead exposure at levels below those causing clinical symptoms." Controversy on this subject appears not as yet satisfactorily resolved.

Peripheral Nervous System. Inorganic lead has toxic effects on the peripheral nervous system. The incidence of lead palsy in occupationally exposed persons is reported in the earlier literature on lead, with the main symptom being weakness of the most heavily used extensor muscles. In more recent years, measurement of a reduced peripheral nerve conduction velocity has been reported[263,264] by several workers, indicating that nerve conduction impairment is induced in some workers at blood lead levels exceeding 50 μg/100 ml.

Renal System

Two types of effects of lead exposure on renal function have been observed[242,243] in the human population. The first involves proximal tubular damage leading to decreased tubular reabsorption of glucose and α-amino acids in children and adults with subtle signs of lead poisoning. It is characterized by aminoaciduria, hypophosphatemia with hyperphosphaturia, and glycosuria. In some cases, aminoaciduria appears by itself. The condition has been studied in some detail in children.[265] The second type of effect, due to prolonged exposure to lead, is a progressive chronic nephropathy which sometimes leads to renal failure even long after exposure to lead has ceased. Kidneys become contracted, with arteriosclerotic changes, interstitial fibrosis, glomerular atrophy and hyaline degeneration of the vessels. Currently, this second set of effects is only rarely encountered in occupational exposure.

Gastrointestinal Tract

Lead colic involves diffuse, crampy abdominal pain and constipation, often preceded by headache and general muscle aches. Vomiting and anorexia with weight loss may occur, with the sequence of events developing over a period of one to two weeks.[244] The occurrence of colic is an early warning of more serious effects likely to occur from prolonged exposure. Its occurrence usually is accompanied by other signs of poisoning, such as elevated coproporphyrin in urine, basophilic stippling, reticulocytosis or anemia.

Cardiovascular System

Dingwall-Fordyce and Lane[266] reported an increase in cerebrovascular disease as a cause of death among workers heavily exposed to lead. Under less severe exposure conditions, such increases were not observed.[242,243] Conflicting results exist on the incidence of hypertension;[243] there are some indications that lead has a toxic action on the heart, as evidenced by alterations in the electrocardiograms of lead-exposed workers.[242,243]

Respiratory Tract

In animal experiments, alveolar macrophages have been seen to be damaged[226,267] and reduced in number[227] in response to lead exposure. In addition, the ability of alveolar macrophages to degrade benzo(a)pyrene,[268] and of the lungs to eliminate bacteria,[242] are reduced by exposure to lead. These respiratory defense mechanisms have been observed to be impaired in animals at lead exposure levels ranging from 10 to 200 $\mu g/m^3$.

Reproduction

High-level lead exposure among women industrial workers around the turn of the century resulted in decreased fertility and an increased rate of spontaneous abortions.[242] More recently, ovulatory disfunction was reported to occur at a higher rate among women working in the lead industries than in a control group.[242] (Shortcomings in this work have been indicated.[243]) Decreased fertility in men occupationally exposed to lead has also been reported.[269]

Carcinogenicity

A Working Group of the International Agency for Research on Cancer has concluded[270] there is no evidence to suggest that exposure to lead salts causes cancer in man. In rats and mice, feeding of high levels of lead acetate or injection of lead phosphate led to the development of renal tumors.[242,243] In Syrian hamsters, a synergistic effect of lead oxide and benzo(a)pyrene administered intratracheally was observed with respect to the induction of lung tumors.[271] The significance to man of the findings of lead-induced tumors is not known at present; however, the comparable level of lead exposure that has been associated with malignant tumors in experiments on rodents in considerably higher than the toxic dose in humans.

Mutagenicity

Reports of chromosomal abnormalities in men occupationally exposed to lead have been contradictory.[241,242] The question therefore remains open as to the effects of lead on chromosomal integrity.

Summary

A main triad of toxic responses to lead exists involving the hematopoietic, the renal and the nervous systems. The influence of lead on the nervous system during early developmental stages has been indicated as being of particular concern due to the demonstrated placental transfer of lead, the increased absorption of lead by young animals, and an apparent susceptibility of the developing nervous systems to adverse effects of lead exposure.

7.5.3 Protection, Remediation and Diagnostic Techniques

The following procedures have been used for the protection of workers involved in the lead industries: ventilation of work areas using baghouses or scrubbers, wearing of respirators in critical work areas, washdown of areas where high levels of lead dust occur, changing of clothes and showering by workers, establishment of regular programs for monitoring blood lead levels in workers. Chelation therapy has been used to counteract problems of lead toxicity.[272,273] In a recent study of 63 workers, intravenous Ca EDTA and oral penicillamine caused the greatest increases in urinary lead excretion.[272] In addition, administration of chelating agents (Ca EDTA; 2,3 dimercaptopropanol) in treatment of acute and chronic lead intoxication in children has been indicated as being of some benefit.[71,274] Diagnostic tests for lead toxicity mainly center around the known interference of lead with the hematopoietic system. Blood lead levels below which specific signs and symptoms of lead intoxication have not been shown to occur are given in Table 7.10. The table demonstrates the usefulness of hematological indices for lead exposure, as these indices are observable at blood levels that are much below those associated

Table 7.10. No-Detected-Effect Levels in Terms of Pb-B[a]

Level of No Detected Effects[b]	*Effect*	*Population*
<10	Erythrocyte ALA-D inhibition	Adults, children
20-25	FEP	Children
20-30	FEP	Adult, female
25-35	FEP	Adult, male
30-40	Erythrocyte ATPase inhibition	General
40	ALA excretion in urine	Adults, children
40	CP excretion in urine	Adults
40	Anemia	Children
40-50	Peripheral neuropathy	Adults
50	Anemia	Adults
50-60	Minimal brain dysfunction	Children
60-70	Minimal brain dysfunction	Adults
60-70	Encephalopathy	Children
>80	Encephalopathy	Adults

[a] *Source: Environmental Health Criteria 3, Lead, World Health Organization, Geneva, 1977.*

[b] *µg Pb/100 ml blood.*

with irreversible damage. Changes in blood indices can serve as monitors for prevention of excessive absorption of lead. Thus, in addition to measurements of lead in blood and urine, measurements of ALA and CP in urine, and of FEP in blood are used as diagnostic tests for evidence of unacceptable degrees of occupational exposure. According to Zielhuis,[275] if the blood lead level is greater than 80 µg/100 ml and one of the following is greater than the stated value, evidence for undue exposure to lead is provided: lead in urine > 800 µg/L. More recently, blood lead levels in industrial workers of 60 µg/100 are recommended[243] as indicative of a need for concern.

Early diagnosis of lead exposure requires establishment of quantitative dose-effect relationships. Clinical symptoms of lead poisoning appear only at toxic levels; early detection in the subclinical phase is desirable, before these signs of serious damage are apparent. This section catalogues the results of numerous studies that have attempted to correlate exposure to lead with physiological responses.

Blood lead (Pb-B) concentration is generally seen as a reasonable measure of an exposure to environmental lead; however, it is less widely accepted as an absolute value, because the extent to which Pb-B reflects the body burden or concentration in critical organs is not well established. Pb-B is assumed in this discussion to be an indicator of the effective exposure (dose), and use of the expression dose-effect relationship refers to the apparent association of dose (as estimated by Pb-B) and the intensity of a specified effect observed in individual subjects.

Satisfactorily accurate dose-response relationships have been established only for the hematologic effects of lead. The earliest demonstrable effect is the inhibition of ALA-dehydratase (ALA-D), which becomes measurable at about 10 to 20 µg Pb/100 ml of blood and suggests a lower-limit threshold for an inhibitory effect on the blood-component formation. The dose-dependence of this relationship has been demonstrated by a number of investigators (see Table 7.11, Part A).

Urinary ALA excretion begins to rise when the Pb-B exceeds 35 to 40 µg/100 ml and demonstrates a distinct threshold for the effects. Above this level, ALA excretion tends to increase rapidly. However, the correlation of ALA with Pb-B is not as strong as that of ALA-D. The results of investigations on the dose-dependence of ALA are shown in Table 7.11, Part B.

Increased concentration of "free erythrocyte porphyrins" (FEP) represents the next class of detectable hemotological disturbances induced by lead. This class of response has special significance because, unlike changes in ALA-D activity, it is believed that significant modifications of free circulating porphryins are toxic and responsible for clinical manifestations of diverse types of porphyria.[235] It has also been suggested that mental disturbance in children may occur at lead levels capable of raising FEP concentration.[236]

FEP is a mixture of protoprophyrin (90% Proto 9) and coproporphyrin. Analyses of FEP, Proto 9 or ZPP (zinc-bound protoporphyrin) are measures of approximately the same response parameter. Detectable protoporphyrin concentrations occur in women and children at Pb-B levels of about 25 to 30 µg/100 ml, and in adult men at 35 to 40 µg/100 ml.[237] Roels identifies an FEP response in children with a Pb-B below 25 µg/100 ml and suggests that children should be defined as a highly sensitive population for this parameter.[238] The dose-effect relationship of FEP and Pb-B for several studies is reported in Part C of Table 7.11.

Reticulocytosis becomes observable at Pb-B levels between 60 to 80 µg/100 ml; shortening of erythrocyte life span also occurs in this range. The degree of anemia correlates poorly with Pb-B; however, a slight drop in hemoglobin level has been observed with Pb-B levels of about 50 to 80 µg/100 ml in workers studied for two to four months. Lilis has shown a weak dose-dependent relation for hemoglobin levels in adult workers ($r = 0.14$) (see Table 7.11, Part D).

Neurological effects of lead exposure can arise in the peripheral or central nervous systems. Lead encephalopathy occurs in both acute and chronic forms, usually among children. In the former it is often impossible to separate the effects of protracted exposure from acute exposure in the past. As a result, dose-response relationships for central nervous system disorders are not well defined. Peripheral neuropathy exists in both clinical and subclinical forms. Nerve damage has been demonstrated in workers with Pb-B levels of 80 to 120 µg/100 ml, e.g., slowed conduction velocity in nerves of the upper limbs.[240] Landrigan[261] has reported an apparent relationship between peroneal nerve conduction velocity and blood lead in children living in the vicinity of a lead smelter (see Table 7.11, Part E).

Impairment of kidney function has also been observed in severe lead poisoning. Renal response of a degenerative and irreversible nature is associated with heavy, prolonged lead exposure, e.g., in occupational situations with poor industrial hygiene. Lead nephropathy is

Table 7.11. Hematological Dose-Effect Relationships Established for Blood Lead Concentrations

Compound and Statistical Relationships
A. α-aminolevulinc acid dehydratase (ALA-D) inhibition
1. Ln (ALA-D) = 7.374 - (0.035) (Pb-B) ± 0.229[a] adult workers, n = 1147, r = -0.84
2. Ln (ALA-D) = 2.281 - (0.019) (Pb-B)[b] adult males, n = 150, r = -0.64
3. Ln (ALA-D) = 1.864 - (0.015) (Pb-B)[c] children (10-13 years), n = 143, r = -0.87 log (ALA-D) = 1.7 - 0.01 (Pb-B) n = 100, r = -0.668[d] newborn (Belguim) log (ALA-D) = 6.7685 - (0.0436) (PB-B)[e] children (1-12 years), n = 51, r = -0.80
4. Ln (ALA-d) = 5.0827 - (0.031) (Pb-B)[f] adult workers, n = 170, r = -0.97
B. Urinary excretion
1. Ln (ALA = (0.016) (Pb-B) - 0.215[g] adults, n = 14, r = 0.801
2. Ln (ALA) = (0.030) (Pb-B) - 1.981[h] male workers, n = 110
3. Ln (ALA) = (0.298) (Pb-B) - 0.8388[b] male workers, n = 150, r = 0.33
C. Free Erythrocyte Porphyrin FEP, µg/100 ml RBC
1. Ln (FEP) = 0.321 + 0.025 (Pb-B)[c] children (10-13 years), n = 51, r = 0.73
2. (FEP) = (0.043 [Pb-B])^2 + (0.45) (Pb-B) - 2.14[i] children (1-9 years), n = 1056, r = 0.79
3. Ln (FEP) = 1.65 + 0.08 (Pb-B)[j] adult males, n = 48, r = 0.9
4. Log (FEP) = 1.469 + 0.022 (Pb-B)[k]
Protoporphyrin (PP)
(PP) = 3.3718 (Pb-B) - 41.303[h] workers, n = 38, r = 0.50
Zinc Protoporphyrin (ZPP)
[ZPP] = 0.0053 (Pb-B)$^{5/2}$[e] male workers, n = 144
D. Hemoglobin Level, (Hb)
(Hb) = 15.8 - (0.0227) (Pb-B)[m] adult workers, n = 103, r = 0.14
E. Peroneal Nerve Conduction Velocity, (V)
V = 54.8 - (0.045) (Pb-B)[i] children (1-9 years), n = 202, r = -0.38

Table 7.11. Continued

[a] S. Tola, "The Effect of Blood Lead Concentration, Age, Sex, and Time of Exposure upon Erythrocyte δ-aminolevulinic Acid Dehydrotase Activity," Work-Environ. Health 10: 26-35, 1973.

[b] A. Azar, "An Epidemiologic Approach to Community Air Lead Exposure using Personal Air Samplers," in "Environmental Quality and Safety," Suppl., Vol. II: "Lead," T. B. Griffin and J. H. Koels, eds., pp. 254-290, New York, 1975.

[c] H.A. Roels et al., "Impact of Air Pollution by Lead on the Heme Biosynthetic Pathway," Arch. Environ. Health 31: 310-315, 1976.

[d] R. Lauwerys et al., "Placental Transfer of Lead, Mercury, Cadmium and Carbon Monoxide in Women. I. Comparison of the Frequency Distributions of the Biological Indices in Maternal and Umbilical Cord Blood," Environ. Res. 15: 278-289, 1978.

[e] J. A. Millar, "Lead and δ-aminolevulinic Acid Dehydratase Levels in Mentally Retarded Children," The Lancet, October 3, pp. 695-698, 1970.

[f] H. Sakurai, "Biological Response and Subjective Symptoms in Low Level Lead Exposure," Arch. Environ. Health 29: 157-163, 1974.

[g] A. Cavalleri, "Determination of Plasma Lead Levels in Normal Subjects and in Lead-exposed Workers," Brit. J. Indust. Med. 35: 21-26, 1978.

[h] B. Haeger-Aronsen, "An Assessment of the Laboratory Tests Used to Monitor the Exposure of Lead Workers," Brit. J. Indust. Med. 28: 52-58, 1971.

[i] P.J. Landrigan et al., "Increased Lead Absorption with Anemia and Slowed Nerve Conduction in Children Near a Lead Smelter." J. Pediatr. 89: 904-910, 1976.

[j] S. Piomelli, "A Micromethod for Free Erythrocyte Porphyrins: The FEP Test," J. Lab. Clin. Med. 81(1): 932-941, 1973.

[k] H.A. Roels et al., "Lead and Cadmium Asorption among Children Near a Nonferrous Metal Plant," Environ. Res. 15: 290-308, 1978.

[l] W.E. Blumberg, "Zinc Protoporphyrin in Blood as a Biological Indicator of Chronic Lead Intoxication," J. Environ. Path. Toxicol. 1: 897-910, 1977.

[m] R. Lilis et al., "Prevalence of Lead Disease among Secondary Lead Smelter Workers and Biological Indicators of Lead Exposure," Environ. Res. 44: 255-285, 1977.

associated with exposures of ten years and more or with repeated and severe acute poisonings. No dose-effect relationships are available for manifestations of lead nephropathy.

The classic signs and symptoms of lead poisoning (digestive disturbance, epigastric discomfort, constipation or diarrhea, etc.) tend to occur with doses over 80 μg/100 ml. Blood lead concentrations above 100 μg/100 ml increase the likelihood of more severe symptoms, such as classic lead colic (noted at levels >150 μg/100 ml). Other effects involve isolated biochemical processes such as serum enzyme activity.

The relationship between blood lead levels and the onset of various symptoms is shown diagrammatically in Figure 7.8, based on data from Herberg. Because of the wide variability in individual sensitivities, no definite cut-off points are defined. The broken portions of the lines in the figure represents effects that occur in part of an exposed population as blood levels increase. These effects become more pronounced and more prevalent with increased blood levels, and the continuous segments of the lines represent the presence of severe effects in most exposed individuals.

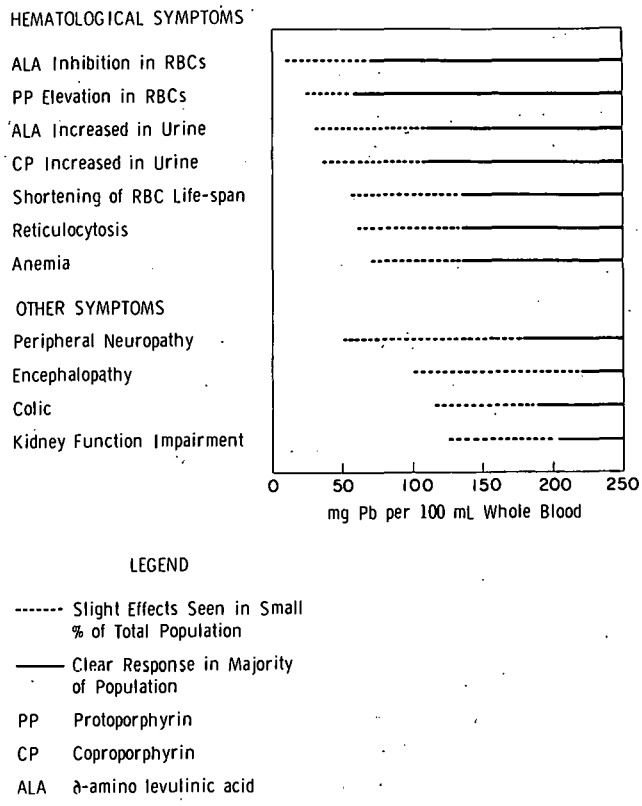


Fig. 7.8. Relationship between Blood Lead Levels and Symptoms of Lead Toxicity. (From S. Iternberg, *Biochemical, Subclinical and Clinical Responses to Lead and Their Relation to Different Exposure Levels, as Illustrated by the Concentration of Lead in the Blood*, Chapter B-15, Section 4, Figure 3 in *Effects and Dose-Response Relationships of Toxic Metals*, G.F. Nordberg, ed., Amsterdam, Elsiever Scientific Publ. Co., 1976.)

7.6 NICKEL

The use of nickel in the United States will increase significantly if Ni/Zn or Ni/Fe battery powered electric vehicles are commercialized. By the year 2000, nickel production will have to have increased by 48% and 30% of 1975 world production levels in order to meet projected demands of nickel for use in the Ni/Zn and Ni/Fe battery systems, respectively (see Tables 3.15 and 4.14). Although this nickel may be imported from Canada and thus not increase U.S. mining or milling, exposure to nickel associated with its mining, milling and refining are still evaluated in this assessment. In addition, in order to make nickel-containing batteries economically viable, a method for domestic recycling of nickel will have to be established to make use of the nickel from spent electrodes. The level of domestic handling will thus be increased, and health related concerns associated with the potential increase should be evaluated.

During the various processes used for refining of nickel, human exposure to nickel subsulfide (Ni_3S_2), nickel sulfate ($NiSO_4 \cdot 6H_2O$), nickel oxide (NiO) and nickel carbonyl ($Ni(CO)_4$) may occur.[276] Manufacture of the nickel electrode for the Ni/Zn battery, as described in Section 3, involves use of nickel powder, a metallic nickel grid, and a high-temperature impregnation bath containing nickel nitrate [$Ni(NO_3)_2$] and a small percent of either cobalt or cadmium nitrate. In the case of the Ni/Fe battery (Sec. 4), nickel electrode manufacture as presently anticipated involves electrolytic plating of nickel onto a steel grid structure. The potential for exposure to both soluble and insoluble forms of nickel during battery manufacture thus exists. In addition, compounds released into water as solutions or suspensions resulting from recycling of electrode components, and electrolyte disposal with electrolyte neutralization, include $Ni(NO_3)_2$, NiO, $Ni(OH)_2$ and $NiSO_4$. The nickel compounds mentioned here are those considered in this assessment. Occupational and environmental exposure by inhalation have received special attention, as nickel toxicity by ingestion occurs only after intake of large amounts of nickel.[277]

7.6.1 Metabolic Uptake, Retention and Excretion

Few data are available on nickel metabolism following inhalation of nickel compounds such as might be encountered by persons occupationally exposed during nickel refining or battery manufacture. Those data that are available on nickel uptake, tissue distribution, and excretion come from two main sources: experimental results obtained using animals exposed to $NiCl_2$[277-281] and $Ni(CO)_4$,[282,283] and analysis of human tissues for nickel content by various analytical methods (colorimetric,[284,285] spectrographic[286,144] and atomic absorption spectrophotometry[287]). The source of nickel exposure in the latter case is mainly food; nickel in air and water contribute little to man's daily nickel intake.[284]

Absorption through Inhalation

In one experiment[133] Syrian golden hamsters were exposed daily for three or more weeks to NiO by inhalation at mean concentrations of 39 and 61.6 mg NiO/m^3. By day three or four after cessation of exposure, these animals retained in their lungs approximately 20% of the total nickel inhaled.[133] The NiO aerosols to which they were exposed had mass median aerodynamic diameters (MMAD) between 1.0 and 2.5 μm. Pulmonary clearance studies showed that NiO cleared quite slowly from the lungs. By six days after exposure the lungs contained 70% of the NiO originally deposited. By 100 days, 20% to 30% of the original lung burden remained. No nickel was found in the carcass, liver or kidney. Either very little nickel was translocated from the lungs to the circulation, or, more likely, any nickel that was translocated was promptly excreted.

Tissue Distribution and Excretion

Animal Studies with $NiCl_2$. Soluble $NiCl_2$ is not a form of nickel encountered in the Ni/Fe or Ni/Zn battery industries, but a portion of the nickel translocated from lung following solubilization might be handled by the body in a way similar to $NiCl_2$. As a result, its metabolism is reviewed here.

Smith and Hackley[278] injected rats with 2.5 or 5 μCi $^{63}NiCl_2$ intravenously and showed that urinary excretion of nickel was close to complete by four hours after injection, with 61% of the injected dose in the urine by 72 hours. Fecal excretion accounted for 5.9% of the injected dose

by 72 hours. The nickel concentration in tissues was highest in the kidney at all times measured. The nickel in all tissues cleared quickly, and by 72 hours only the kidney had retained a significant amount. At two hours after injection, the kidney contained 2.6% dose/g tissue, with the adrenal, ovary, and lung next in concentrations.

Wase et al.[279] injected mice intraperitoneally with 1 μCi of $^{63}NiCl_2$ and analyzed tissues from 2 to 72 hours after injection. The concentration of nickel was greatest in the kidney >> lung >> plasma > liver at two hours. Tissue concentrations peaked at four to eight hours and declined thereafter, except in the liver and lungs, which retained the nickel to some extent. Excretion was greater in the feces than in urine, possibly due to intraperitoneal injection of the nickel.

Sunderman et al.[282] confirmed the data of Smith and Hackley,[278] showing that by 12 hours after intravenous injection of $^{63}NiCl_2$ into rats, 81.4% of the injected dose was excreted in the urine; by four days 89.9% was in the urine and 3.0% in the feces.

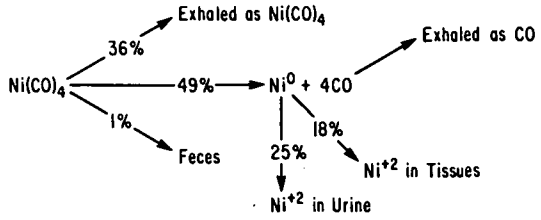
When $^{63}NiCl_2$ was instilled intratracheally into rats, again the nickel was rapidly cleared from the body; 90% was excreted by 72 hours, mainly (75%) into urine.[280] Tissue concentrations fell rapidly, with concentrations in the kidney > lung >> adrenal > liver and other tissues at six hours after instillation.

Clary[280] and Parker and Sunderman[288] noted a striking localization of ^{63}Ni in the pituitary of guinea pigs and rabbits, respectively.

In summary, $NiCl_2$ administered to animals clears rapidly from the body, with the main route of excretion being into urine. The highest concentrations of nickel are found in the kidney, with the pituitary and lungs also relatively rich in nickel.

Animal Studies with $Ni(CO)_4$. The metabolism of $Ni(CO)_4$--one of the forms of nickel involved in nickel refining--has been carefully investigated. Studies in the rat have been performed using both $^{63}Ni(CO)_4$[282] and $Ni(^{14}CO)_4$.[283] These reports showed that, following intravenous injection of $^{63}Ni(CO)_4$ (2.2 mg Ni/100 g body weight), 38.4% of the injected dose was exhaled as intact $Ni(CO)_4$ by six hours after injection, and none was detected in the exhaled air after that. By four days, 31% of the injected nickel was recovered in urine and 2.4% in feces. Nickel excreted into urine was not in the form of $Ni(CO)_4$. At 24 hours, 17.7% of the injected nickel was found in the tissues, with the following distribution (expressed as percent of the 24 hour body burden of nickel): muscle and fat, 41.2%; bone and connective tissue, 31.1%; viscera and blood, 26.6%; brain and spinal cord, 1.1%; lung, 4.3%; and liver, 1.9%. When $^{63}Ni(CO)_4$ was administered by inhalation exposure rather than intravenous injection, a greater percentage of the 24-hour body burden of nickel appeared in the brain and spinal cord (4.4% vs. 1.1%) and the lungs (18.3% vs. 4.3%). Combining the results from the studies with $^{63}Ni(CO)_4$ and $Ni(^{14}CO)_4$, the following pathway diagram for nickel retention and excretion at

24 hours following intravenous injection into rats can be constructed:



(% = percent of injected dose of Ni)

It may be noted that most, but not all, of the injected nickel is accounted for in this diagram.

In summary, following exposure to $Ni(CO)_4$, the two main pathways for excretion of nickel are exhalation of intact $Ni(CO)_4$ and urinary excretion of ionic nickel. Unlike $NiCl_2$, which appears to clear from the body rapidly, with 81.4% the injected dose appearing in the urine by 12 hours,[282] nickel administered as $Ni(CO)_4$ is retained by the body somewhat longer.

Distribution and Excretion in Humans. For an adult American, oral intake of nickel is estimated at between 300 to 600 µg/day.[284] Ingested nickel is poorly absorbed and most of it appears in the feces. Nickel excretion in the feces of healthy American adults averages 258 µg/day. The rate of excretion has been observed to vary over a range of 80 to 540 µg/day.[289] To a lesser degree, (between 1% and 10%) nickel also appears in urine. Other minor routes of excretion include sweat, hair and, in certain situations, respiration.

The concentration of nickel in tissues from humans not occupationally exposed have been analyzed by several investigators.[133,284-287] Koch et al.[285] measured nickel by spectrochemical analysis and found the highest concentrations in bladder (35 µg/g ash) and small intestine (55 µg/g Ni ash), followed by the stomach (17 µg/g ash), lung (12 µg/g ash), and cardiac muscle (11 µg/g ash). Other tissues ranged from 3 to 8 µgNi/g ash, with striated muscle, kidney, and prostate having the lowest concentrations. Schroeder et al.[284] measured nickel concentrations colorimetrically using dimethylglyoxime. They analyzed kidney, liver, intestine and lung, looking for variations with geographical location and age. Kidneys from persons in the U.S. had a mean nickel concentration of 7 µg/g ash, with nickel occurring in 27% of the kidney samples analyzed. Livers from persons in the U.S. contained 6 µg/g ash, with 22% occurrence. Nickel was found in both the intestine and lung of the newborn, with mean concentrations of 38 µg/g ash (90% occurrence) and 11 µg/g ash (55% occurrence), respectively. There appeared to be an accumulation of nickel with age in the lung, reaching a mean concentration of 35 µg/g ash by 65 years of age. Tipton and Cook[144] did a very thorough study of the trace-element composition of 29 different human tissues using emission spectrography. For the ten tissue groups in which nickel occurred in over 50% of the samples analyzed, the percent occurrence, median concentration (µg/g ash), and average mean concentration (µg/g ash), respectively, were as follow: skin, 95%, 47, 110; cecum, 94%, 21, 34; rectum, 90%, 16, 32; omentum, 84%, 22, 34; adrenal, 77 10, 30; sigmoid colon, 75%, 12, 27; ileum, 62%, 9, 21; trachea, 52%, 5, 9; duodenum, 52%, 5, 8 and lung, 50%, < 5, 20. For the other tissues analyzed, the median concentrations were all below the limit of detection (5 µg/g ash). Sunderman[287] determined nickel concentrations by atomic absorption spectrophotometry in the lung, liver and heart of four previously healthy subjects who died suddenly from murder or suicide. Expressing his results in terms of µg/g

ash, he cited the following mean concentrations: lung, 1.7 μg/g ash; liver, 9 μg/g ash; heart, 5 μg/g ash. These values are close to the average mean concentrations reported by Tipton and Cook[144] for lung (20 μg/g ash), liver (4 μg/g ash), and heart (9 μg/g ash). None of the above authors reported nickel concentrations in the pituitary of humans, so the observation of a high nickel concentration in the pituitary of animals could not be confirmed in humans by these studies.

Analyses of sweat collected from healthy humans during sauna bathing indicate that sweating can represent a significant excretion route for nickel from the body.[290] During 15 minutes of sauna bathing, approximately 1 μg of nickel was shown to be excreted. This compares with the daily urinary excretion of 2.5 ± 1.4 μg/day nickel measured for residents of Hartford, Connecticut.[291]

Combining the tissue distribution and excretion results of animals and man, the following observations can be made: (1) Ni normally appears in very low concentrations in human tissues; (2) Ni^{+2} appears to concentrate most in the skin, intestine, and pituitary; (3) a relatively high concentration is also found in lung, adrenal, trachea, and omentum; (4) endogenous Ni^{+2} is most likely excreted mainly by way of the urine, as shown to be the case for rats,[279-282] dogs,[292] and guinea pigs;[281] (5) sweating may result in a significant excretion of nickel; (6) the detailed metabolism of insoluble nickel compounds following inhalation exposure has not yet been well investigated.

7.6.2 Toxic Responses and Human Health Implications

The aspects of nickel toxicity of concern to human health in the development of an expanded Ni/Fe or Ni/Zn battery manufacturing industry pertain to the occupationally exposed. Epidemiological studies have clearly shown that exposure to nickel in various types of nickel refineries has led to an increased incidence of tumors of the lung and paranasal sinuses.[276,293,294] The Mond process for producing pure nickel has the potential for industrial exposure to nickel carbonyl, which is very toxic in acute exposure situations and has been shown to be carcinogenic under low-level exposure conditions.[294] Ni/Zn and Ni/Fe battery manufacture, as now anticipated, involve the potential for exposure by inhalation to nickel powder, vapors containing a soluble form of nickel and cobalt or cadmium from a high-temperature impregnation bath, and nickel-containing vapors from the electrolytic plating of nickel. Nickel has been shown to be an essential component of human metabolism, and toxic responses following oral intake of nickel requires very high[277] exposure levels; as a result, the focus of this assessment is on toxic effects of airborne nickel on the occupationally exposed.

Respiratory System

Epidemiological Studies of Nickel Carcinogenesis. Thorough and carefully controlled studies have been made of the incidence of respiratory cancer in nickel refinery workers in Wales,[295,296] Canada,[297] Norway[298] and Russia,[299] documenting increases in cancers of the lung and nasal cavities among these workers (see Table 7.12). In a breakdown of work categories at the Norwegian refinery, electrolysis workers were among those shown[298] to have an increased incidence of respiratory cancers. This study points to a potential risk associated with the manufacture of the nickel electrode for the Ni/Fe battery, which involves electrolytic plating of nickel. In addition to respiratory cancers, increased incidences

Table 7.12. Respiratory Cancers among Workers in Two Nickel Refineries

Location of Refinery	Year of First Employment	Respiratory Cancers Observed/Expected	
		Lung	Nasal Cavities
Clydach, Wales (Mond process)[b]	Before 1910	9.5	308
	1910 - 1914	10.5	870
	1915 - 1919	5.7	400
	1920 - 1924	6.3	116
	1925 - 1944	1.3	-
Kristiansand, Norway (Electrolytic process)[c]	1910 - 1929	10.4	100
	1930 - 1940	4.5	64
	1945 - 1954	4.5	4.3
	1955 - 1960	2.5	-

[a] *As tabulated in F. W. Sunderman, Jr., "Metal Carcinogenesis," in "Advances in Modern Toxicology," Vol. 2, "Toxicology of Trace Elements," R. A. Goyer and M.A. Mehlman, eds., Hemesphere Publishing Corp., Washington, D.C., 1977.*

[b] *R. Doll, L. D. Morgan and F. E. Speizer, "Cancers of the Lung and Nasal Sinuses in Nickel Workers," Brit. J. Cancer 24:623-632, 1970.*

[c] *E. Pedersen, A. C. Hogetveit and A. Andersen, "Cancer of Respiratory Organs among Workers at a Nickel Refinery in Norway," Int. J. Cancer 12:32-41, 1973.*

of laryngeal cancer in Norwegian nickel refinery workers and increased risks of gastric carcinomas and soft tissue sarcomas in Russian nickel refinery workers have been reported.[293] Three cases of renal cancer among 225 Canadians involved in electrolytic refining of nickel may be related to nickel exposure.[293] Furnace operations in nickel refineries appear to be associated with the greatest hazard of respiratory cancers.[295-299] Respirable particles of metallic nickel, nickel subsulfide (Ni_3S_2), and nickel oxide (NiO) are considered as the principle respiratory carcinogens in nickel refineries[294] although the identity of the compounds responsible remains uncertain and other compounds being considered include the vapor of nickel carbonyl and soluble aerosols of nickel sulfate, nitrate, or chloride.[293]

In addition to increased incidences of cancers, nickel exposure has been associated with other toxic responses of the respiratory tract. In several studies, chronic rhinitis and nasal sinusitis in workers at electrolytic nickel refineries have been described.[293] In another investigation,[300] precancerous nasal lesions (atypical epithelial metaplasia) were found in 16% of 92 workers exposed to nickel in an electrolytic nickel refinery.

Animal Studies: Nickel Carcinogenesis Related to Battery Industry Operations. Studies on the induction of respiratory tumors in animals in response to inhalation exposure to powdered nickel compounds are few. Ottolenghi et al.[301] exposed 226 rats to nickel subsulfide (Ni_3S_2) at a concentration of 1 mg/m^3 for 78 weeks, 5 days/week, 6 hr/day. This concentration is equivalent to the current OSHA standard for occupational exposure to nickel compounds. This exposure resulted in a 14% incidence of lung tumors in the exposed animals, as compared to 1% observed incidence in approximately 240 control rats.[301] Table 7.13 shows the hyperplastic and neoplastic lung changes observed in the study. Most tumors appeared nearly two years after initiation of exposure, the first appearing at 76 weeks. Exposure of rats and guinea pigs to prolonged inhalation of powdered metallic nickel at a level of 16 mg/m^3 for 6 hr/day, 4 to 5

Table 7.13 Hyperplastic and Neoplastic Changes in Lungs of Rats Exposed to Nickel Sulfide[a]

	Controls[b,c]		*Nickel Sulfide*[b,c]	
Pathologic Changes	*Males*	*Females*	*Males*	*Females*
Typical Hyperplasia	26 (24)	20 (19)	68 (62)	65 (66)
Atypical Hyperplasia	17 (16)	11 (10)	58 (53)	48 (49)
Squamous Metaplasia	6 (6)	4 (4)	20 (18)	18 (18)
Tumors:				
Adenoma	0 (0)	1 (1)	8 (7)	7 (7)
Adenocarcinoma	1 (1)	0 (0)	6 (5)	4 (4)
Squamous cell carcinoma	0 (0)	0 (0)	2 (2)	1 (1)
Fibrosarcoma	0 (0)	0 (0)	1 (1)	0 (0)

[a] *A. D. Ottolenghi et al., "Inhalation Studies of Nickel Sulfide in Pulmonary Carcinogenesis of Rats," J. Nat Cancer Inst. 54:1165-1172, 1974.*

[b] *Values represent the number of affected animals in each group. Percent of affected animals is given in parentheses.*

[c] *Number of animals: Controls - 108 males, 107 females; Ni_2S_3 - 110 males, 98 females.*

days/week until death (maximum of 21 weeks) resulted in a production of multicentric adenomatoid formations of the alveoli and hyperplastic proliferations of the terminal bronchiolar epithelium in almost all guinea pigs and many of the rats.[302] Quantitation of these lesions was not reported. Toxicology of inhaled NiO was studied in Syrian golden hamsters exposed to 53 mg/m^3 NiO for 7 hr/day, 5 days/week for life.[276] Heavy burdens of NiO were found in the lungs, causing a development of pneumoconiosis with consolidation of the lungs. NiO exposure had no effect on lifespan. Inhaled NiO was thus reported to be neither carcinogenic nor particularly toxic in hamsters. Numerous investigations using other modes of administration of nickel dust, NiO, or Ni_3S_2 indicate that the carcinogenic potency of Ni_3S_2 is greater than that of any other metallic compound that has been investigated.[303] As an example, intramuscular injection of only 5 μ moles of Ni_3S_2 in Fischer rats produced an incidence of sarcomas at two years of 23/30, or 77%, compared with 0/180 in control rats.[303] From the results of these animal experiments, and the fact that Ni_3S_2 is a major component of nickel refinery flue dust,[303] it appears that Ni_3S_2 is one of the likely etiological agents in the induction of respiratory tumors in nickel refinery workers. Reduction of the OSHA standard for inorganic nickel to 15 μg/m^3 has been recommended by the National Institute for Occupational Safety and Health.[303a]

Nickel Oxide: Lung Pathology at Levels below the OSHA Standard. Responses to inhalation of nickel compounds other than tumorigenic responses are relevant to our considerations, especially if they have been observed at concentrations below current exposure limits set for nickel. Bingham et al.[304] subjected rats to inhalation of aerosols of $NiCl_2$ and NiO at concentrations of 110 to 120 μg/m^3 (the OSHA standard for occupational exposure to nickel is about 10 times higher). After two weeks of exposure to NiO for 17 hr/day, 6 days/week, at 120 μg/m^3, the number of alveolar macrophages washed from rat lungs increased 2.8-fold over controls.

After four to six weeks, the increase was more than threefold. In addition, significant accumulations of macrophages in the alveolar spaces, hypersecretion of mucus in bronchial epithelium, and lymphocytic infiltration in the alveolar walls and perivascular spaces were observed. With exposure more prolonged than two weeks, a thickening of the alveolar walls was observed. Alveolar macrophage cell numbers were not altered significantly by $NiCl_2$ exposure, but the bronchial epithelium became hyperplastic, with evidence of marked mucus secretion. Peribronchial lymphocytic infiltration was also observed. In addition, inhalation of $NiCl_2$ for one week resulted in an especially cloudy washing that was still evident after the ninth lavage. The histological alterations in the lungs in response to nickel did not occur following exposure of rats to similar aerosols of Pb_2O_3 or $PbCl_2$, indicating that the lung response was more than a nonspecific response to particulates. Alterations in lung structure in response to several weeks exposure to NiO at concentrations 10 times below the OSHA standard for nickel bear further investigation.

Toxicity of Nickel Carbonyl. Nickel carbonyl is a colorless, volatile liquid with a boiling point of 43°C. The pure vapor decomposes spontaneously above room temperature; decomposition is rapid in the presence of oxidizing agents; the dilute vapor in air is 50% decomposed in approximately 30 minutes.[305] It is readily formed from carbon monoxide and metallic nickel or a nickel compound that can be reduced under preparation conditions.[306]

Acute Toxicity. Both clinical experience and experimental work with animals indicates that nickel carbonyl is highly toxic when inhaled. An acutely lethal atmospheric concentration for man is considered to be 0.24 g/m^3, or 30 ppm by volume.[306] The symptoms in man immediately following exposure include frontal headache, giddiness, chest tightness, nausea, weakness, perspiration, cough, vomiting, and shortness of breath.[306] These immediate symptoms often subside within a few hours of removal from exposure, and usually between 12 and 36 hours after exposure, severe delayed symptoms develop if exposure has been great enough. Delayed symptoms frequently observed include shortness of breath with painful inspiration, nonproductive cough, muscular weakness, substernal pain, chilling sensations, muscular pain, and sweating.[294] Poisoning from inhalation commonly goes unrecognized, because the "sooty" odor of nickel carbonyl vapor is difficult to detect and the immediate symptoms described above are usually mild, nonspecific and transitory. In humans, if death occurs, it will occur in 3 to 13 days after exposure and is attributed primarily to respiratory failure with diffuse interstitial pneumonitis, and cerebral hemorrhage or edema. Patients who recover from nickel carbonyl poisoning often have a long period of convalescence due to pulmonary insufficiency, unless therapy with dithiocarb has been initiated soon after exposure.[307]

Short-term inhalation exposures (20-120 min) of animals to concentrations of nickel carbonyl from 0.1 g/m^3 to 3 g/m^3 have produced acute toxicity responses.[294] The pulmonary parenchyma is the target tissue for all species tested, regardless of route of administration. The symptoms and pathologic lesions observed in animals are similar to those described above for humans. Symptoms of acute exposure include dyspnea, tachypnea, cyanosis, fever, apathy, anorexia, vomiting, diarrhea, and, occasionally, hind-limb paralysis. Generalized convulsions frequently occur at the time of death. In rats which survive acute exposure, cytological alterations in the lung regress with time, and from 14 to 21 days after exposure the pulmonary parenchyma is essentially normal except for interstitial fibrosis.[277] A small percent of the survivors of acute exposure have been observed to develop lung cancer after a two-year latency period.[308,309]

Toxicity from Chronic Inhalation Exposure. One reported case of human lung disease has been attributed[310] to chronic inhalation of low concentrations of nickel carbonyl. Asthma and Löffler's syndrome (condition characterized by severe cough, breathlessness, anorexia, weight loss and eosinophilia) developed during a period of the man's employment at an industrial plant manufacturing nickel carbonyl. Concentrations of nickel carbonyl to which he was exposed were not reported. The patient recovered completely after removal from all contact with nickel, but died five years later from carcinoma of the lung.[294] The possibility exists that there might be a connection between tumor induction and nickel carbonyl exposure in this case. Animal experiments conducted to evaluate the carcinogenesis of nickel carbonyl following chronic inhalation exposure have been reported. Sixty-four rats exposed to 4 ppm (30 mg/m^3) of nickel carbonyl for 30 minutes, three times per week till death grew less rapidly and died sooner than a group of 32 controls.[309] Most rats died of causes other than tumors: inflammatory conditions (pneumonitis, pneumonia, bronchitis, bronchiectasis, bronchial abcesses and reactive fibrosis) occurred commonly in the animals, and frequently were the cause of death. After two years, only seven to eight rats (12%) remained in the exposure group (vs. 30% in controls), and one of these exposed animals was found to have an adenocarcinoma of the right lung. Taking into account results of this and a similar study[308] by the same laboratory, a 21% incidence of lung cancer was observed in animals surviving two years vs. 0% in controls;[294] the percentage of animals surviving more than two years was small, however. Chronic exposure to nickel carbonyl thus produced pulmonary carcinomas in animals that closely resemble the lung cancers that have developed in nickel workers, but lung cancer occurred in only a small number of animals and the lung cancer incidence observed, though greater than in control groups, was low.

Other Systems

Skin. Dermatitis is a well-known response to nickel exposure that deserves mention in an assessment of nickel toxicity. Present occupational exposure to nickel includes nickel mining, extraction and refining, plating, casting, grinding and polishing; nickel powder metallurgy; nickel alloys and nickel/cadmium batteries; the chemical industry; electronics and computers; food processing, and nickel waste disposal and recycling.[277] The clinical symptoms of nickel dermatitis, which were first observed in nickel miners, smelters, and refiners, include itching or burning papular erythema that starts in the web of the fingers and spreads to the fingers, wrists and forearms. Epidemiological studies have suggested an incidence of nickel reactivity in the range of 4% to 13% of selected patient populations.[277] In the general population, the incidence of allergic contact sensitivity to nickel has remained constant over the past 40 years at about 12%.[311] It has been estimated that 5% of all eczema is caused by nickel or its compounds. The incidence of nickel dermatitis resulting from occupational exposure has decreased due to improvements in industrial technology and advances in industrial medicine.[294]

Sunderman suggests that dermatitis is a sensitization reaction, and in some cases, contact may produce proxymal asthmatic attacks and pulmonary eosinophilia.[312] In fact, a case of asthma associated with inhalation of nickel sulfate has been documented for a nickel-plating worker with nickel dermatitis.[313] The public health implications of asthmatic disease from nickel sensitization are far reaching. Given that 12% of the general public is identified as a nickel-sensitive subgroup, an industrial process that significantly increases population exposure to finely divided nickel aerosols has the potential for adversely influencing the general state of health by increasing the incidence of asthmatic attacks.

Renal System. Proteinuria, aminoaciduria, and reduction of urea clearance associated with morphological lesions in the renal glomeruli and tubules have been observed following intraperitoneal administration of $NiCl_2$ to rats at 2 to 5 mg/kg body weight.[293] Proteinuria has also been observed in a few workers accidentally exposed to $Ni(CO)_4$ by inhalation.[293] A study of the renal function of workers chronically exposed to soluble nickel compounds has not yet been carried out.

Hyperglycemic Response. A transient hyperglycemia has been measured[293] in response to both intraperitoneal and intratrachael injection of $NiCl_2$ and inhalation exposure to $Ni(CO)_4$. The importance of this hyperglycemic response to the toxicity of nickel among the occupationally exposed has not yet been clarified.

Fetotoxic Response. Relevant clinical data are not available on the fetotoxicity of nickel. Evidence for such an effect is derived exclusively from animal studies. Schroeder has shown reduced neonatal survival, litter size and individual size of offspring of rats exposed to a nickel concentration of 5 ppm in drinking water.[314] Other studies have reported similar findings for fetotoxicity.[315-317] Such findings may have significance to female nickel industry workers during early pregnancy.

7.6.3 Protection, Remediation and Diagnostic Techniques

Indices of Exposure to Nickel

In the case of human exposure to $Ni(CO)_4$, the concentration of nickel in urine has been used to indicate the extent of acute exposure. Exposure is considered *mild* if the initial eight-hour collection of urine has a nickel concentration less than 10 µg/100 ml, *moderately severe* if the initial eight-hour urine specimen contains 10 to 50 µg/100 ml, and *severe* if the nickel concentration is greater than 50 µg/100 ml urine.[307] In addition to urinalyses, $Ni(CO)_4$ can be detected in expired breath by use of gas chromatography[318] or a chemiluminescent detector.[305] Measurements of nickel in serum and urine have also been shown[291] to give a valid indication of environmental exposure to nickel. This is indicated by serum and urine nickel concentrations of 4.6 ± 1.4 µg/L (n = 25) and 7.2 ± 3.9 µg/L (n = 19), respectively, measured in residents of Sudbury, Ontario, the site of the largest nickel mines in North America; and serum and urine concentrations of 2.6 ± 1.0 µg/L (n = 26) and 2.0 ± 0.9 µg/L (n = 20), respectively, in residents of Hartford, Connecticut, a city with relatively low environmental concentrations of nickel.[291]

Therapeutic Measures for Treating Cases of Nickel Poisoning

Sunderman[307] has recommended that patients in the "moderately severe" and "severe" categories listed above for $Ni(CO)_4$ exposure be treated immediately by administration of the chelating agent, sodium diethyldithiocarbamate ("Dithiocarb"). After initiation of oral Dithiocarb therapy, urinary excretion of nickel is promptly increased and the clinical signs of nickel carbonyl poisoning are relieved within a few hours. With continued Dithiocarb therapy, patients show uneventful recoveries. Fifty men treated with Dithiocarb for acute nickel carbonyl poisoning returned to work three weeks after treatment.[307] Of 31 acute nickel carbonyl poisoned patients treated with dimercaprol,[319] two died, and the period of convalescence for most of the others lasted several months. In the case of Ni(II) poisoning in rats, Horak et al.[320] found that triethylenetetramine and penicillamine were very effective antidotes, and that sodium

diethyldithiocarbonate was much less effective. However, the use of chelating agents for Ni(II) poisoning in humans is not a common practice.

Protection from Exposure

Protection from exposure to nickel in the occupational setting includes the use of ventilation hoods for operations such as nickel powder pressing during manufacture of the nickel electrode for the Ni/Zn battery, ventilation of workroom areas, and use of personal respirators for dusty operations. Substitution of nickel-containing products with cloth or plastic, and coating of nickel objects with plastic, fingernail polish or lacquer, or some other physical barrier can be used to prevent nickel dermatitis. In the case of potential exposure to $Ni(CO)_4$, a device should be used for continuous monitoring of $Ni(CO)_4$ levels in working areas.[305,306]

7.7 COMPARATIVE HEALTH RISK ASSESSMENT

When we define a primary use scenario for storage-battery technology in which the battery represents the power supply for an electric vehicle (EV), then environmental effects resulting from vehicle use can be directly attributed to battery technology. The analysis given here considers the effect of central power station air emissions resulting from the increased electric power demand for the daily EV charging requirement. These increased emissions are related to anticipated changes in air quality from which population health impacts can be estimated.

The initial step of the analysis was to estimate ambient air concentrations that would occur if a coal-fired power plant were used to meet the energy demand for vehicle charging. Nelson et al.[321] report the factor of 0.22 kWh/ton·mile for the energy required at the wheels of an electric vehicle, which conforms to the profile of a small commuter car (four passenger, 10,000 miles/yr use). This factor was used to derive a total daily energy demand for 10,000 such vehicles. The electrical power output of a generating station was then equated to the overall demand (incorporating efficiency factors for each step throughout the system) from which energy input into the plant was derived. Assuming such a plant would meet new source performance standards (NSPS) for pollutant emissions, emission rates for total suspended particulates (TSP) and sulfur dioxide (SO_2) were calculated. These emission rates were then incorporated into a simplified plume dispersion model from which maximum ground-level pollutant concentrations (χ_{max}) were estimated. Continuous population exposures were assumed to be approximately 1% of the calculated χ_{max}. These ambient exposure levels were then used to project population health effects (see App. D).

The estimates of mortality risk were derived using the predicted effective air pollution increments shown in Table 7.14 and our computer-based projection model, DEMPAK. The exposure-response assumptions of DEMPAK have been described elsewhere.[322] The risk estimate results from the division of the total person-years lived between 1980 and 2010 by the total number of excess mortalities due to the increased air pollution during that same period.

Three factors enter into the range estimate to give the extremes: air stability, charging cycle time, and the specific health-risk model employed. The air may be either highly stable or unstable, the charging cycle six or eight hours long, and the health risk based on TSP alone or both TSP and SO_2. The result is the health-risk estimates given in Table 7.15.

Table 7.14. Maximum 24-hour Ground-level Concentrations of TSP and SO_2 ($\mu g/m^3$) from Charging 10,000 Batteries--by Type, Air Conditions and Charge Cycle

Battery Type	Stable Air Conditions				Unstable Air Conditions			
	8-hr Charge		6-hr Charge		8-hr Charge		6-hr Charge	
	TSP	SO_2	TSP	SO_2	TSP	SO_2	TSP	SO_2
Lead/Acid								
Present	1.40	16.9	1.88	22.6	0.40	4.80	0.53	6.40
Improved	1.37	16.4	1.82	21.9	0.39	4.65	0.52	6.21
Ni/Fe								
Present	1.24	14.9	1.66	19.9	0.35	4.23	0.47	5.64
Improved	1.22	15.0	1.62	19.4	0.34	4.24	0.46	5.51
Ni/Zn								
Present	1.46	17.5	1.94	23.3	0.41	4.96	0.55	6.61
Improved	1.52	18.2	2.03	24.3	0.43	5.15	0.57	6.88

Table 7.15. Comparison of Health Risk from Charging 10,000 Electric Vehicles Over the Period 1985-2010 (by Battery Type)[a]

Battery Type	Risk of Death/10^0 Person-years
Lead/Acid	
Present	3.99 - 47.76
Improved	3.89 - 46.28
Ni/Fe	
Present	3.50 - 42.06
Improved	3.40 - 41.01
Ni/Zn	
Present	4.01 - 49.24
Improved	4.29 - 51.37

[a] *Factors affecting the range of estimate are explained in the text.*

An inter-battery comparison of health impact from charging 10,000 electric vehicles (Table 7.14) shows that the Ni/Zn battery would have the greatest health effect. Lead/acid batteries are second in rank order of impact. The least impact is expected from charging the Ni/Fe batteries. The risk is lessened with both the "improved" lead/acid and Ni/Fe batteries as compared to their respective "present" specification models, but the reverse is true of the powerful, long-range "improved" Ni/Zn battery. The differential between the battery systems with the greatest and least risk is about 0.8 to 10.0 mortalities per 10^8 person years over the 40-year period.

A second estimate of risk was calculated for EV use, based on the following scenario:

Population (urban area)

1980 - approx. 1×10^6

2010 - approx. 1.3×10^6

Age Structure of the United States, 1970

Number of batteries (charging energy from coal-fired electric power plant)

	1985	1995
Lead/acid	4,000	10,000
Ni/Fe	11,000	27,000
Ni/Zn	11,000	27,000

"Present" battery systems were used for the 1980-1995 period and "improved" systems for the 1995-2010 period. In Figure 7.9 the effects of the air stability, charging cycle and health risk model can be seen. The scenario with the lowest risk is that with highly unstable air and an eight-hour charging cycle. The overall estimate of health risk ranges from 12.9 to 153.8 mortalities per 10^8 person-years.

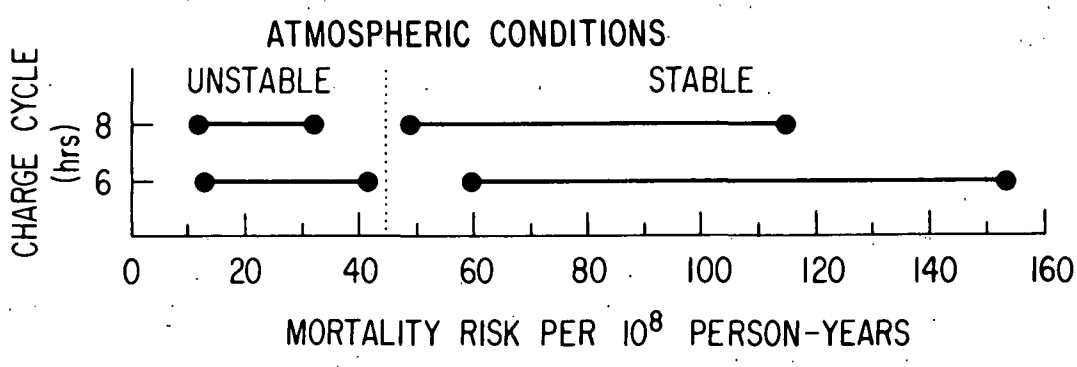


Fig. 7.9. Projected Health Risk Resulting from the Use of a Combination of EVs Having the Following Present and Improved Battery Types: Lead/Acid, Ni/Fe, Ni/Zn.

7.8 SUMMARY

Emissions from the three near-term battery cycles contain several potentially hazardous chemicals. The biomedical effects of these chemicals have been assessed in context of the scenarios described in Sections 2 through 5. The anticipated effects of these chemicals are summarized below, and a list of research recommendations is given in Appendix G.

1. Antimony and the Gas Stibine (Sec. 7.1)
   - Based on measurements made with a load-leveling lead/acid battery and assuming total release without decomposition of the stibine produced inside the battery, maximum possible stibine levels in home garages during battery charging are calculated to be 1 to 7 times

the TLV (Threshold Limit Value) of 0.5 mg/m^3. The decomposition of stibine could result in maintenance of antimony trioxide levels in the garage close to the TLV of 0.5 mg/m^3. Antimony trioxide will be deposited in the garages, and higher levels can be expected in air during garage cleaning operations.

- Stibine is a potent hemolytic agent. Information on toxic responses to low levels of stibine is not available. Analogy to arsine must be relied upon for estimating toxic responses to stibine. Inhalation exposure to high levels of antimony trioxide (45 to 125 mg/m^3) has produced degenerative changes in the lungs, liver and spleen after 30 to 45 days; effects of low-level exposures have not been reported.
- Past experience among antimony smelter workers has indicated a pneumoconiosis incidence rate of 10 to 15 percent. Exposure of smelter workers to antimony sulfide dust could result in degenerative changes of the heart muscle. Such effects have been observed in both animals and man following exposure for short periods (6 weeks for animals, 2 years or less for man) to antimony sulfide at 5 mg/m^3 (10 times the OSHA standard).

2. Arsenic and the Gas Arsine (Sec. 7.2)
   - The potential for occupational and public exposure to arsenic will increase with the increased use of lead and zinc ores, which contain arsenic as a contaminant.
   - Arsine is generated along with stibine during charging of the lead/acid battery; maximum garage levels of arsine are estimated at less than one-tenth the TLV.
   - Arsine is a potent hemolytic agent and low-level chronic exposures in humans have been known to cause slight decreases in hemoglobin and red blood cell levels.

3. Cadmium (Sec. 7.3)
   - As a contaminant of lead and zinc ores, cadmium is of primary health concern for the smelter work force, the population living in the vicinity of the lead and zinc smelters, and the workers involved with nickel electrode impregnation during Ni/Zn battery manufacture.
   - Cadmium oxide fumes at levels at or below the OSHA standard (0.1 mg/m^3) have been associated with emphysema. Occupational exposure data indicate that proteinuria develops with a few years' exposure to CdO fumes at 0.07 to 0.2 mg/m^3 or dust at 0.02 to 0.7 mg/m^3. Dusts are generally considered less toxic than fumes. Studies linking cadmium exposure to specific kinds of cancer are inconclusive.

4. Cobalt (Sec. 7.4)
   - Cobalt is a component of the nickel electrode in the Ni/Zn and Ni/Fe batteries. Cobalt is toxic only at high levels of exposure.
   - There is no epidemiological evidence associating occupational exposure to cobalt with increased risk of cancer.

5. Lead (Sec. 7.5)
   - Significant increase in the occupational exposure and environmental release of lead can be expected with expansion of lead/acid battery industry.
   - Toxic responses to lead of concern to exposed populations include effects on the hematopoietic, the renal and the nervous systems. Young children are particularly susceptible to the toxic effects of lead exposure.

6. Nickel (Sec. 7.6)

- Epidemiological studies have shown that exposure to nickel in various types of nickel refineries has led to an increased incidence of tumors of the lung and paranasal sinuses. Chronic rhinitis, nasal sinusitis, and precancerous nasal lesions have also been observed in the occupational setting.
- Nickel subsulfide, a major component of nickel refinery flue dust, has been demonstrated to produce a 14% incidence of lung tumors in rats at the OSHA standard for nickel compounds (0.1 mg/m^3). Nickel carbonyl, a compound involved in the Mond process for nickel refining, is lethal to man following acute exposure to 30 ppm by volume.
- Nickel oxide and nickel chloride at 0.1 mg/m^3 produce detrimental lung changes in rats following several weeks of exposure. Inhalation exposure of workers to soluble salts of nickel during the impregnation and electroprecipitation steps of nickel electrode manufacture of Ni/Zn and Ni/Fe batteries could result in such adverse impacts.

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8. REGULATORY ASPECTS

ABSTRACT

Existing regulatory actions and standards related to the near-term battery systems are reviewed, together with proposed standards and recommendations for anticipated constituents in the air emissions, water effluents and solid wastes from battery-related operations. The likelihood of compliance with existing and proposed limitations assessed, and likely impedements to commercialization of the near-term batteries are discussed. In-plant occupational health and safety standards for metallic compounds contained in battery-cycle emissions are summarized.

8.1 ENVIRONMENTAL REGULATION

8.1.1 Air

Federal standards for U.S. air quality have been set by the Clean Air Act (1963, 1967) and Amendments (1970, 1977).[1] The national ambient air quality standards (NAAQS) promulgated to date by the U.S. Environmental Protection Agency (EPA) apply to sulfur oxides (SO_x), total suspended particulates (TSP), carbon monoxide (CO), photochemical oxidants, hydrocarbons (HC),[1] nitrogen dioxide (NO_2) and lead (Pb) (Table 8.1). There are two levels of standards--primary standards to protect human health, and stricter secondary standards to protect public welfare. The 1977 amendments require that by 31 December 1982, all areas of the country meet federal standards; to do this, each state is required to develop a state implementation plan (SIP) containing standards for emissions from new stationary sources, a preconstruction review process for new sources, and timetables for compliance by new and existing sources. Thus, each state may set its own emissions limitations and compliance schedules, with the federal standards serving as the maximum allowable levels. SIPs are based on regional air quality management district standards. Most responsibility for enforcement is at state or local levels.

The standard (NAAQS) for airborne lead is 1.5 μg lead/m^3 air, based on a monthly average. Airborne lead levels in 1977 averaged 2 to 4 μg/m^3 nationwide. Some large cities have concentrations as high as 6 μg/m^3. The standards for lead have particular importance to primary and secondary lead smelting, primary copper smelting, and lead/acid storage-battery-manufacturing industries. These industries are now required to control fugitive lead emissions from process steps and smokestacks. The technology is available to control these emissions; however, the cost for the control equipment may be as high as seven percent of the revenues for some of these industries.[2] States currently are developing plans (through SIP) to control emissions from specific sources. The states are required to show how they will attain NAAQS lead standards by 1982.

The Lead Industries Association has forecast severe economic impacts on the lead industry as well as the battery industry as a result of these standards.[3] The industry forecasts that, because of the high background ambient air-lead concentrations, an estimated six battery plants

Table 8.1. Ambient Air Quality Standards[a]

Pollutant / *Time Period*	*Maximum Permissible Concentration*[b] *Primary*	*Secondary*
Total Suspended Particulates (TSP)		
Annual	75 $\mu g/m^3$	60 $\mu g/m^3$
24-hour	260 $\mu g/m^3$	150 $\mu g/m^3$
Sulfur Oxides (measured as SO_2)		
Annual	80 $\mu g/m^3$	-
24-hour	365 $\mu g/m^3$	-
3-hour	-	1300 $\mu g/m^3$
Carbon Monoxide (CO)		
1-hour	40 mg/m^3	-
8-hour	10 mg/m^3	-
Oxidants/Ozone (O_x/O_3)		
1-hour	160 $\mu g/m^3$	-
Nitrogen Dioxide (NO_2)		
Annual	100 $\mu g/m^3$	100 $\mu g/m^3$
Hydrocarbons (HC)[c]		
3-hour	160 $\mu g/m^3$	160 $\mu g/m^3$
Lead (Pb)		
30-day	1.5 $\mu g/m^3$	-

[a] *Information based on 40 CFR 50.*

[b] *Primary--to protect public health; secondary--to protect public welfare.*

[c] *Hydrocarbon standard does not have to be met if oxidant standard is met.*

in the Los Angeles vicinity will be forced to close because of technological inability to meet the EPA standards. The Association has filed suit in the Federal District Court of Appeals for the District of Columbia to overturn EPA's ambient air standard for lead, claiming that the 1.5 $\mu g/m^3$ standard is more stringent than necessary to provide proper health protection to the public.

Section III of the Clean Air Act directs the EPA to establish maximum allowable pollutant emissions from new or substantially modified stationary sources that are major emitters of pollutants, such as ore smelters or coal-fired power plants. It is further required in the law that these stationary sources use the "best available control technology" to reduce emissions substantially. The administration of emission control requirements will be conducted by the states with SIP agreements, on a case-by-case basis.

In 1976, the EPA established an "offset policy" for non-attainment areas (i.e., those areas in violation of NAAQS). For a new facility to be constructed in such an area, steps must be taken to more than offset the new emissions by further reducing emissions of the same pollutants from existing sources. A state may request a waiver from such an offset policy if it can demonstrate that its own SIP will achieve the same goal. The attainment/nonattainment status of an

Air Quality Control Region is made on a pollutant-specific basis. Consequently, a firm desiring to construct a facility in a particular area of the country may be subject to non-attainment rules for one pollutant and to PSD rules (see paragraph below) for another.

In December 1974, EPA promulgated "Prevention of Signification Deterioration" (PSD) provisions. Generally, an area's designation of attainment status indicates that its air quality is better than national standards. In compliance with the Clean Air Act Amendment of 1977, the states, through their SIPs, are required to show how they will prevent air-quality deterioration. The PSD system is based on three classes of clean-air areas for which maximum allowable increases in pollution levels for SO_2 and TSP are established. PSD review occurs in all areas of the country where SO_2 and TSP levels do not exceed NAAQS. Maximum increases in SO_2 and TSP concentrations in these regions are set by specified increment limits over the NAAQS concentrations for these pollutants. PSD permitted increments are shown in Table 8.2. The EPA also is developing PSD rules for carbon monoxide, hydrocarbons, nitrogen oxides, lead, and photochemical oxidants. These regulations have a statutory deadline of August 1979.

Table 8.2. PSD Permitted Increments, $\mu g/m^3$[a,b]

	Class I	*Class II*	*Class III*	*NAAQS*[c]
SO_2				
Annual	2	20	40	80
24-hour	8	91	182	365
3-hour	25	512	700	1300(s)[d]
TSP				
Annual	5	19	37	7560(s)
24-hour	10	37	75	260 150(s)

[a] *Implications of the 1977 Clean Air Act Amendments for Stationary Sources, Environ. Sci. Technol. 12(2):144-149, February 1978.*

[b] *All 24-hour and 3-hour values may be exceeded once per year.*

[c] *National Ambient Air Quality Standards.*

[d] *(s) indicates a secondary standard.*

There are 28 specifically named categories of industry designated as major emitting facilities that must comply with the PSD numerical increment limitations. These industries have emissions of more than 100 tons/yr of any pollutant. In addition, any other industry emitting more than 250 tons/yr of any pollutant also is designated as a major emitting facility. Included in the list of industries categorized as major stationary sources are primary zinc smelters, sulfuric acid plants, primary lead smelters sintering plants, and secondary metal production facilities.

The Clean Air Act also imposes controls on emissions from a new source for those categories of industry for which the EPA has issued New Source Performance Standards (NSPS). The EPA is currently in the process of establishing regulations covering the operation of all nonferrous smelters. The industries for which EPA has issued NSPS of particular relevance to the battery program are as follows:

- Nitric acid plants (40 CFR Part 60, Subpart G)
- Sulfuric acid plants (40 CFR Part 60, Subpart H)
- Secondary lead smelters (40 CFR Part 60, Subpart L)
- Primary zinc smelters (40 CFR Part 60, Subpart Q)
- Primary lead smelters (40 CFR Part 60, Subpart R)

A summary of the NSPS for smelters is presented in Table 8.3. In effect, the standards are the same for all new smelting operations currently subject to emission limitations. Secondary lead smelters are not subject to federal NSPS for SO_2. Under Section 119 of the Clean Air Act of 1977, owners/operators of primary nonferrous smelters can obtain two 5-year delayed-compliance permits in meeting SO_2 requirements; these can then be incorporated into the state's SIP.

Table 8.3. Existing New Source Standards for Smelters[a]

Substance or Quality	*Primary Lead Smelters*[b]	*Secondary Lead Smelters*[c]	*Primary Zinc Smelters*[d]
Particulates, mg/dscm[e]	50	50	50
SO_2, % by volume	0.065	0.065	-
Opacity, %	20	20	20

[a] *Based on information in 40 CFR Part 60.*

[b] *Applies to sintering machine, sintering machine discharge and blast furnace, dross reverberatory furnace, electric smelting furnace, and converter.*

[c] *Applies to pot furnaces of more than 250 kg (550 lb) charging capacity, blast (cupola) furnaces, and reverberatory furnaces.*

[d] *Applies to primary zinc smelters, roaster and sintering machine: (a) from any roaster gases, (b) for sintering machines that eliminate more than 10% of the sulfur initially contained in the zinc sulfide ore concentrates.*

[e] *dscm = dry standard cubic meter.*

On 31 January 1979, the EPA published (40 CFR 57) proposed requirements for the first primary nonferrous smelter orders and the procedures for issuing them. Nonferrous smelter orders will allow certain copper, lead, and zinc smelters a delay in compliance with the requirements for constant or continuous control of SO_2 emissions, and allow the use of tall stacks and supplementary control systems for meeting ambient standards. The first order is effective until 1 January 1983, and the second until 1 January 1988. The first order is to be issued without public hearings. However, prior to granting the second order there must be notice and public hearing. Constant control for smelters ultimately will be required whenever

it can be shown in a public hearing that additional controls are demonstrated and economically feasible. The smelters may be allowed to operate without installing a continuous control when it can be shown that a one-year shutdown of the plant would result from such a requirement.

The specific NSPS standards used in a given region will differ depending on the attainment status of the area in which a new source is to be located. The published limitations set minimum standards; states and local regulatory agencies may use stricter standards. For a new plant to be located in an area which exceeds national ambient standards for the pollutant involved strict control equipment (equipment that achieves "lowest achievable emissions rates") must be utilized. A new source to be located in an area where air quality is significantly better than national standards is subject to "best available control technology" (BACT),* which cannot be less stringent than the NSPS. In nonattainment areas, a new source must also comply with the "offset policy." This entails finding emission offsets from existing sources in the area.

The emission limitations must be met by a new source prior to the granting of an operating license. Consequently, if a company installs BACT in a plant and the emissions from the plant do not meet the new source limitations, the operating license will not be granted.

Sulfuric acid, lead-smelting and zinc-smelting plants are significant sources of sulfur dioxide. Consequently, sulfur dioxide standards for these industries were established in order to maintain the air quality required for a region. Approximately 10% of the total sulfur oxides emitted by stationary sources are generated by the nation's 27 primary copper, lead-acid, and zinc smelters. One such smelter is the major cause for an area's non-attainment designation for sulfur dioxide.[2] Currently 11 primary smelters are in full compliance with SIP sulfur dioxide standards or compliance schedules. An additional 13 are operating in violation, and 20 smelters are located in areas where SIPs for sulfur dioxide have not been developed or where the SIP is subject to litigation.

The specific requirements for new facilities will depend upon the existing air quality in a region. For non-attainment areas, a new facility will require offsets to accommodate the new emissions from the proposed plant. The availability of these offsets may limit the construction of a plant, such as a battery manufacturing facility, near a preferred market. Non-attainment areas for the criterion pollutants are presented in Figure 8.1. The construction of new sulfuric acid, lead-smelting and zinc-smelting plants in these areas would appear to be difficult, as offsets may be difficult to obtain, and the pollution control equipment which would be required would be very costly.

The EPA also is developing standards to limit atmospheric emissions of lead from new, modified, and reconstructed lead/acid battery plants which have a production capacity equal to or greater than 500 batteries per day.** These standards will be proposed and published in the *Federal Register* in the fall of 1979. The proposed regulations[4] would establish the following

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**BACT is also defined as the best continuous emission control available.*

***Of the approximately 190 lead-acid manufacturing plants in the United States, about 100 are estimated to have capacities greater than or equal to 500 batteries per day.*

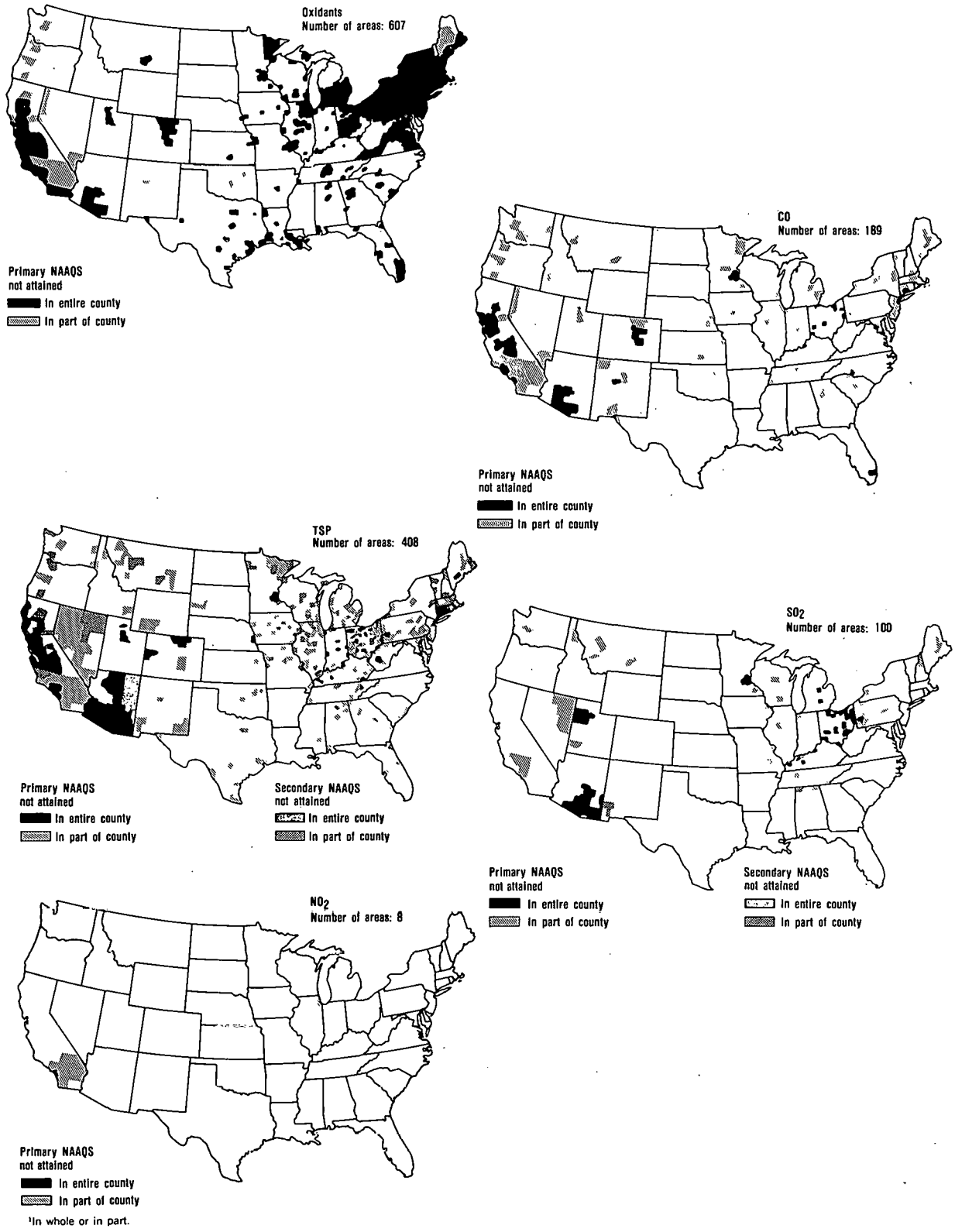

Fig. 8.1. Nonattainment of Criteria Pollutants, August 1977
Source: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards.

emission limits for the facilities listed below:

Facility*	Lead Emission Limit**
Lead-oxide production	5.0 mg/kg (0.010 lb/ton)
Grid casting	0.05 mg/m^3 (0.00002 gr/dscf)
Paste mixing	1.00 mg/m^3 (0.00044 gr/dscf)
Three-process	1.00 mg/m^3 (0.00044 gr/dscf)
Lead reclamation	2.00 mg/m^3 (0.00088 gr/dscf)
Other lead-emitting operations	1.00 mg/m^3 (0.00044 gr/dscf)

The EPA considers the economic impact of these proposed emission limits to be reasonable, and does not expect that they will prevent or hinder the expansion of the lead-acid battery manufacturing industry. The projected growth of 3 to 5 percent (over the period 1979 to 1984) in the industry's manufacturing capacity is expected to take place by the expansion of existing large plants. The best demonstrated systems for reduction of lead emissions entails the use of fabric filters. Impingement scrubbers can also be employed, but require greater operating costs. The average incremented cost from these proposed standards is expected to be about 30 cents per battery, or about 1.6% of the wholesale price.[4]

The draft proposed standards do not include limitations for sulfuric acid mist. However, the federal new source standards for acid mist from sulfuric acid plants is 0.075 kg per metric ton of acid produced (0.15 lb/ton). Several states have allowable air concentrations of sulfuric acid mist. New Jersey limits concentrations to 357 mg/m^3 (0.156 gr/dscf). Other allowable air concentrations include: Montana, 30 µg/m^3 for one hour; Missouri, 10 µg/m^3 for 24 hours, and New York, 100 µg/m^3 maximum.[5] The EPA recognizes the potential for health and environment impact from such mists, and has indicated that the NSPS for lead/acid battery manufacturing may be revised in the future to include standards limiting sulfuric acid mist emissions.[4]

There is considerable likelihood that acid mist regulation will occur in the future. Mist emissions can generally be controlled by containment, venting systems, and fiber mist eliminators. Acid mist control of 95% to 99% can be achieved. Therefore, the minimization of the impacts of emissions can be achieved technically. In so doing the industry will incur a financial burden. For new sources, however, it would appear prudent to plan for such control systems rather than retrofitting at some future time.

Mist emission control may ultimately be related to forthcoming lead emissions control by EPA as well as OSHA. The mist contains lead compounds. After drying there remains a fine, dry powder of high lead content that is a hazard to workers, as well as those outside the plant.

The small droplets of acid or caustic mist can cause damage to nearby property and houses. There have been specific cases where metal finishers, for example those employing chromic acid, have had to make restitution for this type of damage. Some regulations contain "nuisance" clauses that in effect are included so that a company cannot interfere with the right of any

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**The rationale for the facility breakdown is that most operations above are independent of one another.*

***The emission limit for lead oxide is expressed in terms of mg of lead emitted per kilogram of lead processed, while those for other facilities are expressed in terms of lead concentrations in exhaust air (grains per dry standard cubic foot).*

individual to the pursuit of happiness and well-being or cause a nuisance that interferes with that basic right. One company involved in discharging its chromic acid contaminants, for example, was obligated to install pollution-control equipment to mitigate the impact on nearby property and houses.

There are several processes in the near-term battery cycles that will be subject to improved air pollution abatement. For example, the "front end operations", such as battery breaking and crushing processes, do not have air pollution-abatement facilities. Under Section 112 of the Clean Air Act, EPA is promulgating emissions standards for substances designated as hazardous air pollutants. National Emissions Standards for Hazardous Air Pollutants (NESHAP) are presented in 40 CFR, Part 61. Of the NESHAPs currently listed, only that given for mercury potentially applies to near-term batteries. There is an emission limitation of 2300 grams/24 hours from mercury ore processing plants. Both arsenic and lead compounds are currently under study to be listed as NESHAPs. Proposed NESHAPs for arsenic are scheduled for release in December 1979. EPA is currently conducting a health-risk assessment of arsenic emissions. Emission standards will be proposed if it is determined that the emissions (primarily from copper smelters) are hazardous.

The SIPs have impacted the smelting industries. For example, the U.S. copper industry is expected to have little if any growth in smelting capacity through 1985.[2] The lead time required to convert to environmentally sound processes, the large capital outlays, and unsolved problems with fugitive dust emissions may impact the industry beyond 1985.

Similarly, the SIPs for sulfur dioxide--as well as those being developed for lead--are likely to have significant impacts on primary and secondary lead smelting and on the lead/acid battery manufacturing industry. The economic impact may slow industrial growth to the extent that the lead/acid battery may not be economically feasible as a near-term battery. The SIP's for sulfur dioxide and those forthcoming for lead may impede the industry's development to the extent that it cannot meet the requirement for an expanded EV fleet.

8.1.2 Water

EPA is mandated to maintain and restore the integrity of rivers, lakes, and streams as a result of the Federal Water Pollution Control Act of 1972 (P.L. 92-500) and the 1977 Amendments to the Act. The principal purpose of the Act was to establish uniform and enforceable regulations of discharges, so that by 1983 the nation's water quality would be adequate to protect fish, shellfish and wildlife and to provide for water recreation. The Act also calls for no discharges into streams or waterways by 1985.

The Act and Amendments are rather complex, and for purposes of simplicity, only those sections applicable to the battery program (i.e., Secs. 301-309 and Sec. 316) are discussed below.

Sections 301 and 304 of the Act are directed toward establishing effluent limits and guidelines for all pollutants, including toxic substances, from point source discharges.

Municipal point sources should have attained technology-based effluent limitations for secondary treatment by July 1977. Industrial sources were scheduled to have been using "best practicable technology" by the same date. The amendments provide time extensions for those industries that made "good faith" efforts to comply. The deadline for best practicable technology use was April 1979; for municipal sources, it is July 1983.

If compliance with water quality standards cannot be achieved by the use of best practicable technology or secondary treatments, then more stringent, site-specific limitations may be imposed (Sec. 302). The water-quality standards and criteria are to be specified by the states. If the states do not formulate their own standards and criteria, these will be established by the EPA.

New point-source discharges will be regulated for pollutant discharges under Section 306 of the Act. This section calls for national standards for performance or new source performance standards based on control technology processes, operating methods, or other alternatives.

The EPA and state and local authorities are developing pretreatment requirements under Sections 307 (b) and (c), with the intent of controlling these discharges (including toxic materials) into publicly owned treatment works that either interfere or are not consistent with the operation of such facilities.

The 1977 amendments establish cleanup requirements for three categories of industrial discharges: toxic pollutants, conventional pollutants and nonconventional pollutants.

- Toxic Pollutants. In 1975, in a Settlement Agreement resulting from a lawsuit against EPA, EPA initiated a process to establish effluent limitations, pretreatment standards and toxic pollutant standards. To that end EPA is to develop best available technology effluent limitations and guidelines, new source performance standards, and pretreatment standards to govern 65 classes of toxic pollutants. (EPA has subsequently expanded the list to 129 specific pollutants, primarily in industrial discharges.) In this list of pollutants, those associated with the "metals" category are most relevant to the battery program and are listed in Table 8.4. The sources of the toxic materials, such as industrial discharges from nonferrous metals manufacturing and mining, will be regulated for these metals.

Currently, water quality criteria are being developed for these toxic pollutants to update the 1976 EPA Water Quality Criteria (Table 8.5). The toxic pollutant limitations and standards must be achieved by applicable discharges as soon as possible but no later than 30 June 1983. Best available technology will be required of these facilities to meet water quality criteria pursuant to Section 304 (a). More stringent limitations and standards may be imposed if those developed are inadequate to maintain 1983 water quality goals. NSPS are being developed for the category "Nonferrous Metals Point Source." Standards proposed for the secondary metals industry are reprinted in Table 8.6. The table shows a summary of recommended effluent limitations by category and subcategory for secondary lead/antimony and secondary zinc smelting. For battery processing, no secondary zinc smelting is anticipated, although some hydrometallurgical processing is. In this instance the recommended "best available technology (BAT)" limitations, the "best practical technology (BPT)" limitations, and the "new source performance standards (NSPS)" limitations are identical. New sources must comply with the schedules promulgated under Section 306 for NSPS and under Section 307 for pretreatment standards.

Table 8.4 The Priority Toxic Pollutants--Metals

Pollutants	*Characteristics*	*Sources*
	METALS	
Antimony	Not biodegradable	Industrial discharges
Arsenic	Persistent in sediments	Mining activity
Beryllium	Toxic in solution	Urban runoff
Cadmium	Subject to biomagnification	Erosion of metal-rich soil
Copper		Certain agricultural uses (e.g., mercury as a fungicide)
Lead		
Mercury		
Nickel		
Selenium		
Silver		
Thallium		
Zinc		
	OTHER INORGANICS	
Asbestos	May cause cancer when inhaled Aquatic toxicity not well understood	Manufacture and use as a retardant Roofing material Brake lining, etc. Runoff from mining
Cyanide	Variably persistent Inhibits oxygen metabolism	Wide variety of industrial uses

Source: "Environmental Quality," The Ninth Annual Report of the Council on Environmental Quality, Washington, D.C., 1978.

Table 8.5. 1976 Water Quality Criteria[a]

Criteria	*Lead*	*Arsenic*	*Nickel*	*Zinc*	*Cadmium*	*Mercury*
Domestic Water Supply (health)	50 µg/L	50 µg/L		5 mg/L	10 µg/L	2.0 µg/L
Sensitive Freshwater Resident Species					0.4-4 µg/L soft 1.2-12 µg/L hard	0.05 µg/L
Irrigation of Crops		100 µg/L				
96-hour LC^{50} for Freshwater and Marine Aquasystems			0.01 µg/L	0.01 µg/L		
Marine Criteria					5 µg/L	0.10 µg/L

[a] *U.S. Environmental Protection Agency, U.S. Water Quality Criteria, Washington, D.C., 1976.*

Table 8.6. Effluent Limitations Proposed for the Secondary Lead/Antimony and Zinc Industries[a]

Category	*Subcategory*	*Zero Discharge*	*Effluent Limitations Recommended*
Secondary Lead/ Antimony	Battery Cracking		As below
	No Battery Cracking	Yes	
Secondary Zinc		Yes	

Effluent Limitations for Secondary Lead Smelting, with Battery Cracking

Parameter	*Concentration, mg/L*		*Effluent Limitations, kg/MT or lb/1000 lb*	
	30-day Average	*24-hr Maximum*	*30-day Av.*	*24-hr Max.*
pH	6.0 to 9.0		Within the range 6.0 to 9.0	
TSS	25	50	0.025	0.05
Lead	0.5	1.0	0.0005	0.001
Arsenic	0.05	0.1	0.00005	0.0001
Cadmium	0.02	0.04	0.00002	0.00004

[a] *Source: Development Document for Effluent Limitations Guidelines and New Source Performance Standards for the Miscellaneous Nonferrous Metals Manufacturing Point Source Category (Draft), Effluent Guidelines Division, Office of Water and Hazardous Materials, U.S. Environmental Protection Agency, Washington, D.C., March 1977.*

Best available technology requirements for toxic substances, as well as pretreatment standards for discharges of pollutants to publicly owned treatment works, will impact the water-pollution control requirements for the manufacturing and recycling phases of all three near-term battery systems and will require appropriate capital expenditures. It is not anticipated that these technology requirements will place an undue hardship on the industry.

The best practicable technology for use in the lead-acid manufacturing/recycling industry is lime treatment followed by settling.[5] The use of flocculants as well as an eight-hour retention time for settling is also suggested. This wastewater treatment sequence applies also to sites that conduct battery-breaking operations. The EPA assumes there will be no significant discharge from sites not conducting breaking operations. We, however, believe there will be liquid emissions associated with these sites, for example, battery-storage and landfill-associated residual electrolyte, which will require treatment. This need may or may not be accommodated by local, publicly owned treatment-works permits. The liquids may be collected and shipped elsewhere for disposal. The best available technology for use in sites having both battery breaking and smelting is lime precipitation and settling, with optimization of pollutant reduction. Again, zero discharge is assumed for sites not conducting battery-breaking operations.

Due to the toxic nature of the constituents (e.g., nickel, zinc and cadmium) in liquid effluents from Ni/Fe and Ni/Zn manufacturing and recycling, NPDES permits or pretreatment will be required. The effluent limitations will vary depending on the location of the plant. The pollution control requirements for point-source compliance has not been formulated for these industries, particularly because hydrometallurgical processes are anticipated. However, wastewater treatment systems, such as ion-exchange treatment systems, are anticipated. These systems also provide for metal recovery and process solution regeneration steps in addition to pollution control. In the case of nickel recovery, for example, there will be substantial economic credit for utilizing these advanced wastewater treatment systems.

- Conventional Pollutants. Conventional pollutants include suspended solids, fecal coliforms, biological oxygen demand and alkalinity-acidity. Best control technology will be required for these pollutants by July 1984, though it is unclear what the nature of the required technology will be. The best control technology will be at least as stringent as best practicable technology but less than or as stringent as best available technology while not violating water-quality standards. It is anticipated that best control technology will be economically "reasonable" for the industry and should pose no exceptional hardship to the near-term battery systems considered in this report.

- Nonconventional Pollutants. Nonconventional pollutants are those considered neither toxic nor conventional. For these pollutants best available technology is required by July 1987. If (1) the use of best practicable technology results in satisfying the water quality criteria standards, (2) there are no other burden of other discharges in the area, and (3) no public health or other environmental risks are anticipated, then the EPA and/or authorized states may waive best available technology requirements.

Nonconventional pollutant control requirements have not been promulgated for the various phases of cycles for the lead/acid, Ni/Zn and Ni/Fe batteries. It is difficult to ascertain what these limitations and regulations will be, but it is anticipated that the wastewater treatment systems that will be required for compliance with best available technology requirements for toxic materials will also accommodate nonconventional pollutants. Both types of pollutants are generally found in the wastewater stream.

Best Management Practices

The EPA is also publishing regulations to control plant site runoff, leaks, waste disposal, spillage, and drainage from raw material storage associated with an industrial manufacturing or treatment process. These are called best management practices and will ultimately be required in the NPDES permit process associated with Section 402 of the Act.

Best management practices will be required to minimize runoff from waste disposal and other storage around mines and mills. Similar practices will be required for site runoff and leachate collection during battery manufacturing and recycling. For example, spent batteries, which will contain residual electrolyte, will require storage in concrete facilities in order to collect hazardous liquids and thereby minimize groundwater intrusion. With the need to reduce fugitive dust around such sites, enclosed storage facilities will probably also be constructed.

NPDES Permits

Section 402 of the Clean Water Act also sets permit requirements for the discharge of pollutants from a point-source into navigable waters. This systems is entitled the National Pollutant Discharge Elimination System (NPDES). NPDES permits will be issued by the EPA, or by a state having an EPA-approved permit program, and will be reviewed every five years. Under the NPDES the EPA or the state establishes the maximum permissible levels established for each pollutant, as well as a compliance schedule. The monitoring and reporting requirements are also established by each state or the EPA. Prior to receiving NPDES authority by the EPA, each state must comply with continuing planning process requirements (see next heading). A list of states administrating NPDES permits as of January 1978 is presented in Table 8.7.

The major implication of the NPDES process on wastewater emissions from the various operations in the near-term battery programs, is that compliance will be site-specific. The limitations and compliance schedules will, in part, depend on the water quality of a given region.

Continuing Planning Process

Under Section 303 of the Act, states are required to undertake a continuing planning process in order to analyze the water quality standards, effluent limitations and compliance schedules for the state. This is required before a state is given NPDES authority.

The effluent-limitation guidelines, pretreatment standards and new source standards adopted, proposed and planned as of August 1978 for various phases of near-term battery systems are listed in Table 8.8.

8.1.3 Solid Wastes

The Resource Conservation and Recovery Act of 1976[6] is the principal avenue for Federal involvement in solid waste management. The Act is administered through the EPA. The law provides for a hazardous-waste regulatory program; a program to eliminate open dumping; financial and technical assistance for planning enhanced solid waste management programs; grants to rural communities to improve solid waste management systems; and authority for research, demonstrations and studies. In addition to solid and hazardous waste management, the law also directs EPA to administer efforts to conserve natural resources directly and through the management, reuse or recovery of solid and hazardous wastes.

Although the Act is in relatively early stages of promulgation, there are several proposed regulations which will require compliance by those who generate, store, dispose of or transport hazardous wastes in various operations of the near-term battery cycles. The regulations for hazardous waste management arise from Subtitle C (3000 series Sections) of the Act. (Notices concerning promulgation for Subtitle C are listed in 40 CFR 250.) The specific sections of Subtitle C affecting battery-cycle operations are discussed in the following subsections. In addition, the Act encourages states to manage their own solid and hazardous waste programs. Regional solid waste management and planning agencies may also be established following existing Section 208 (Federal Water Pollution Control Act) area-wide water planning agencies. Subtitle D

Table 8.7. Status of EPA Approval for States to Administer Their Own NPDES Permits, January 1978[a]

Approved	*Not Approved*
California	Alabama
Colorado	Alaska
Connecticut	Arizona
Delaware	Arkansas
Georgia	Florida
Hawaii	Idaho
Indiana	Iowa
Illinois	Kentucky
Kansas	Louisiana
Maryland	Maine[b]
Michigan	Massachusetts[b]
Minnesota	New Hampshire
Mississippi	New Jersey
Missouri	New Mexico
Montana	Oklahoma
Nebraska	Pennsylvania
Nevada	Rhode Island
New York	South Dakota
North Carolina	Texas
North Dakota	Utah
Ohio	West Virginia
Oregon	
South Carolina	
Tennessee	
Vermont	
Virginia	
Washington	
Wisconsin	
Wyoming	

[a] *Source: U.S. Environmental Protection Agency, Office of Water Enforcement, "Enforcement Progress Report for FY 1977" (Draft), Washington, DC, March 1978.*

[b] *Cooperative programs with U.S. Environmental Protection Agency.*

Table 8.8. Interim and Final Industry Effluent Standards Affecting EV Battery Production

Industrial Category	*Number of Subcategories*	*Date Proposed*	*Date Promulgated*
Nonferrous Metals Manufacturing (Phase I)	3	12/4/73	4/8/74
Nonferrous Metals Manufacturing (Phase II)	5	2/27/75	12/15/76
Mineral Mining and Processing	17	10/16/75	6/10/76
Ore Mining and Dressing	7	11/6/75	7/11/78
Battery Manufacturing	13		

of the Resource Recovery Act (4000 series Sections) and Section 3006 are directed toward providing guidelines to aid states in developing their plans. Section 1008 of the Act directs EPA to develop guidelines for the landfill disposal of solid waste. Proposed guidelines were published on 26 March 1979 (40 CFR Part 241). These contain recommended considerations and practices for the location, design, construction, operation and maintenance of solid waste landfill disposal facilities that meet the provisions contained in "Criteria for Classification of Solid Waste Disposal Facilities" (40 CFR 257) in accordance with Section 4004 (a) of the Act. Section 4004 Criteria identify the level of performance necessary to assure that no reasonable probability of adverse affects on health or the environment will result from disposal of solid waste at such facilities. At a minimum, disposal of solid waste in landfills should achieve compliance with the criteria. Hazardous waste disposal facilities, however, are subject to the standards developed under Section 3004 in the Act (see below).

Subtitle C

The following guidelines and regulations on identification and listing of hazardous wastes were proposed on 18 December 1978. The date scheduled for final action is December 1979.

Section 3001. Hazardous Waste Criteria - Identification and Listing. These regulations define wastes that will be controlled under the nationwide hazardous waste management program. Criteria are provided to identify characteristics of hazardous wastes based on ignitability, corrosiveness, reactivity and toxicity. Testing procedures are included to determine whether a waste meets the described characteristics. The regulation also lists certain hazardous wastes, and processes presumed to generate hazardous wastes. The following are classified as hazardous waste generators or materials:

SIC

- 3332 Primary lead blast furnace dust
- 3332 Primary lead lagoon dredging from smelter
- 33[illegible] Zinc anode sludge
- 3339 Primary antimony-electrolytic sludge

- 3339 Primary lead sinter dust scrubbing sludge
- 3341 Secondary lead scrubber sludge from sulfur dioxide emission control, soft lead production
- 3341 Secondary lead - white metal production furnace dust
- 3691 Lead/acid storage battery production wastewater treatment sludges
- 3691 Lead/acid storage battery production clean-up wastes from cathode and anode paste production.

The implication of this proposed listing is that a generator of a hazardous waste so classified must follow special handling and disposal practices established by the EPA and/or the state. The industry in possession of these wastes has special responsibilities, and must apply for a permit to dispose of the wastes. A manifest system that follows the waste from its generation to final disposal is required.

Section 3002. Standard for Generators of Hazardous Wastes. This regulation establishes national standards for generators of hazardous wastes, covering such items as recordkeeping, containerization, labelling, waste identification and reporting. This regulation also contains provisions for a hazardous waste manifest system. These provisions will potentially vary from state to state, with Federal regulations as guidelines for minimum compliance.

Section 3003. Standards for Transporters of Hazardous Wastes. These national standards require transporters of hazardous wastes to ship only properly labeled containers and to ship these only to permitted facilities. These standards were published on 28 April 1978 (40 CFR 250).

Section 3004. Standards for Hazardous Waste Treatment, Storage and Disposal Facilities. Hazardous waste management facilities will be required to meet certain technical performance standards regarding operating practices, location and design. These standards contain provisions to protect surfacewater, groundwater, and air quality.

The implication to industries in the near-term battery cycles of Subtitle C regulations when they are finalized is that additional costs will be incurred by such industries, because those wastes classified as hazardous will require special treatment. For example, disposal in "secured" landfills with lining and leachate control, or by some other method acceptable to the EPA and/or the state, is expensive. However, some of the wastes contain valuable materials. Consequently, recycling programs in the industry may reduce the costs associated with final disposal. The industry subject to these regulations may dispose/recycle its wastes on-site, or offsite (in which case they are subject to transportation regulations), or may contract with a hazardous waste management company to dispose of the waste. In the latter case, the industry continues to have responsibilities under the manifest system.

Wastes not classified as hazardous must be disposed in facilities in compliance with Sections 4004 (a) and 1008 of the Act (previously discussed). The criteria and guidelines for these solid waste facilities will most likely result in increased "tipping fees" or fees for the disposal of the waste. Increased costs to the battery industry will occur unless maximum recovery/recycling is practiced.

In general, the regulations do not pose technical barriers for compliance on the part of various segments of the near-term battery industries. Rather, they will result in an

economic impact, the magnitude of which has been studied for one portion of the industry: battery manufacturing. These were addressed by Arthur D. Little, Inc. (1979)[7] and Kearney Management Consultants (1978)[8] for the EPA. The results of these analyses indicate that the industry will not experience disproportionately high hazardous waste management costs. The studies also conclude that differentially high costs may be a problem for lead/acid storage battery plants with wastewater treatment facilities that depend on lime for acid neutralization and solids precipitation (lime treatment produces considerable sludge). The EPA, however, in another study[5] concludes that the economic impact on the industry of utilizing such treatment will be reasonable. In either case, it appears that the regulatory limitations outlined in this section, as well as Sections 8.1.1 and 8.1.2 of this report, will result in a reduction in the number of smaller plants and firms in the battery industry in favor of larger-scale operations.

8.2 AIR CONTAMINANT STANDARDS FOR WORKPLACE EXPOSURES APPLICABLE TO THE ELECTRIC STORAGE BATTERY INDUSTRY

The provisions of the Occupational Safety and Health Act of 1920 (Pu. L. 91-596) require the Secretary of Labor to promulgate exposure standards dealing with toxic materials in the workplace. Such standards must adequately assure, on the basis of best available data, that no employee will suffer material impairment of health or functional capacity even if the employee undergoes regular exposure to the toxic agent for the period of his working life.

The responsibility for setting standards and for their enforcement is shared by the National Institute of Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA). Under Public Law 91-596, NIOSH is authorized to develop and establish recommended safety and health standards. OSHA is empowered to set mandatory job safety and health standards and to enforce them through an effective program of workplace inspections.

Exposure to workplace air contaminants is covered under Section 1910-1000 of the law, wherein OSHA exposure standards are published based on the NIOSH recommendations. Two classes of standards are recognized, the threshold limit value and time weighted average, and the acceptable ceiling value. The time weighted average requires that an employee's exposure to a specific material in any eight-hour shift of a 40-hour workweek should not exceed the eight-hour time weighted average limit.

For specific agents, the acceptable ceiling value requires that an employee's exposure should not exceed at any time during the eight-hour shift the acceptable ceiling concentration, except for a time period, and up to a concentration not exceeding the maximum duration and concentration allowed under "acceptable maximum peak above the acceptable ceiling concentration for an eight-hour shift.

Table 8.9 presents the OSHA exposure standards for compounds identified in the manufacture of electric storage batteries. In this table the OSHA standard (Time Weighted Average) is associated with the recommended threshold limit value (TLV) and, where applicable, with the recommended short-term exposure limit (STEL). Other information included in this table covers known doses and toxic responses of test animals exposed under experimental conditions. This information is representative of the data providing a basis for OSHA exposure standards.

Table 8.9. In-Plant Occupational Health and Safety Standards for Metal Compounds Associated with Various Aspects of Battery Technology[a]

Metal and Components	OHSA Stds[3a] mg/m³	TLV[3b] mg/m³	STEL[3b] mg/m³	Human Toxicity Data[c]	Effects Observed in Humans	Human Carcinogen Determination	Animal Toxicity Data[c,e]			
Antimony					Dermalitis, keratitis, conjunctivitis, and nasal septum ulceration by contact with fumes or dust	EPA: selected for priority attention as point-source water-effluent pollutant				
Sb	0.5	0.5					LDLO: 100 µg/kg	rat		
SbH_3	0.5	0.1	1.5				LCLO: 1100 mg/m³	mus	10 M[d]	
Sb_2O_3	0.5	0.5					LD50: 3250 mg/kg	rat		
Arsenic					Dermalitis, bronchitis, skin cancer, gastro intestinal disturbances; inhalation of AsH_3, hemolysis, interference with total metabolism					
As	0.5	0.5		LDLO: 5 mg/kg		Indefinite human carcinogen	LDLO: 120 mg/kg	mus		teratogenic
AsH_3	0.2	0.2		TCLO: 230 gm/m³			LCLO: 300 mg/m³	rat	15 M[d]	
As_2O_3	0.5	0.05		LDLO: 16 mg/kg		Suspected human carcinogen	LD50: 20 mg/kg	rat		carcinogenic
As_3O_5	0.5			LDLO: 5 mg/kg		Indefinite human carcinogen	LD50: 8 mg/kg	rat		carcinogenic
Cadmium					Systemic toxin, pulmonary emphysema, hypertension, kidney damage, gastrointentinal inflmation	EPA: selected for priority attention as point source water-effluent pollutant				
Cd dust	0.2	0.04	0.15	TCLO: 88 mg/m³			LDLO: 15 mg/kg	rat		carcinogenic
CdO fume	0.11	0.04	0.2	LCLO: 9 mg/m³			LCLO: 500 mg/m³	rat	10 M[d]	carcinogenic
Cobalt					Dermalitis (via ingestion), polyeythemia, asthma					
Co	0.1	0.1					LDLO: 1500 mg/kg	rat		neoplosia
$Co(OM)_2$										
$CoSO_4$							LD50: 54 mg/kg	mus		
CoO							LD50: 1700 mg/kg	rat		carcinogenic
Nickel					Dermalitis, probable nasal cavity and sinus carcinogen	Nickel refinery dust (NiO, NiS) human carcinogen				
Ni	1.0	0.15	0.3				TCLO: 15 mg/m³	rat		carcinogenic
NiO	1.0	0.15					TDLO: 100 mg/kg	rat		carcinogenic
NiS										carcinogenic
$Ni(Co)_4$	0.7	0.35				Indefinite human carcinogen				carcinogenic
$NiCl_2$	1.0	0.15	0.3							
Ni_3S_2		0.15					TDLO: 970 µg/m³	rat	6 H, 76 WI[d]	carcinogenic
Lead					Cumulative poison: produces behavioral disorders, brain damage, convulsions	EPA: Selected for priority attention as point source water-effluent pollutant				
Pb	0.2	0.15	0.45	TDLO: mg/kg 6Y[d]			LDLO: 1000 mg/kg	rat		
Pbo	0.2	0.15					LDLO: 430 mg/kg	rat		
Pb_3O_4	0.2	0.15					LDLO: 1000 mg/kg	gpg		
PbS	0.2	0.15					LDLO: 10 mg/kg	gpg		
$PbSO_4$	0.2	0.15					LDLO: 30 mg/kg	gpg		

[a] *Occupational Safety and Health Act Safety and Health Standards (29 CFR 1910) U.S. Dept. of Labor, Occupational Health and Safety Administration, OSHA 2206 (Revised January 1976), Subpart 2, Sect. 1910.1000, Toxic Substances, Air Contaminants, Washington, D.C., 1976.*

Threshold Limit Values for Chemical Substances in Workroom Air Adopted by ACGIH for 1977, American Conference of Governmental Industrial Hygenists, Cincinnati, OH, 1977.

National Institute for Occupational Safety and Health, Registry of Toxic Effects of Chemical Substances, Vol. II, 1977 ed., NIOSH 78-104B, Washington, D.C., 1977.

Suspected Carcinogens, 2nd ed., A Subfile of the NIOSH Toxic Substances List, 1976, PHS-CDC, National Institute for Occupational Safety and Health, NIOSH 77-149, Washington, D.C., 1977.

[b] *TLV: time-weighted average concentration for a normal 8-hour workday.*
STEL: short-term exposure limit - the maximum concentration to which workers can be exposed for a period up to 15 minutes; a maximum allowable concentration; the absolute ceiling.

[c] *LDLO: lowest reported dose causing mortality.*
TCLO: lowest reported air concentration causing toxic response.
LCLO: lowest reported air concentration causing mortality.
LD50: experimental dose resulting in 50% mortality of the animal test population.

[d] *Length of exposure for air concentrations: M-minutes, H-hours, W-weeks, Y-years, I-intermittent.*

[e] *Experimental test animals are rat, mouse (mus) and guinea pig (gpg).*

8.3 SUMMARY

1. New sources which may be needed to meet the demands of an expanded EV industry will require compliance with minimum Federal standards as promulgated under the Clean Air Act and its Amendments and local limitations. This generally entails permits prior to construction and operation based upon emissions from the new source.

2. Congress has mandated that the U.S. ENvironmental Protection Agency proceed with an enforcement program for the smelters. This provides incentive for hydrometallurgical metal separation rather than smelting operations for copper, cobalt, nickel and zinc.

3. The EPA has proposed standards for atmospheric emissions of lead from new, modified and reconstructed lead/acid battery plants that have a production capacity equal to or greater than 500 batteries per day. Severe economic impacts are predicted for lead/acid battery manufacturing due to proposed ambient air quality standards for lead.

4. Sulfuric acid mist is generally not regulated. The control of hazardous mists may be required for all near-term batteries.

5. The EPA is currently considering National Emission Standards for Hazardous Air Pollutants for arsenic and lead.

6. The State Implementation Plans for the limitations on atmospheric emissions of sulfur dioxide and those proposed for lead may pose an impediment to the lead/acid battery industry's development to meet the requirements of large-scale commercialization of lead/acid battery electric vehicles.

7. Best available technology requirements for toxic substances, as well as pretreatment standards for discharges of pollutions to publicly owned treatment works, will impact the water-pollution control requirements for the manufacturing and recycling phases of all three near-term battery systems.

8. There will be liquid emissions associated with battery-breaking facilities requiring treatment prior to disposal.

9. The following are classified as hazardous waste generators for the three near-term batteries under Section 2001 of the Resource Conservation and Recovery Act:

   - primary lead blast furnace dust
   - primary lead lagoon dredging from smelter
   - zinc anode sludge
   - primary antimony-electrolytic sludge
   - primary lead sinter dust scrubbing sludge
   - secondary lead scrubber sludge from sulfur dioxide emission control, soft lead production
   - secondary lead - white metal production furnace dust

- lead/acid storage battery production wastewater treatment sludges
- lead/acid storage battery production clean-up wastes from cathode and anode paste production.

Any generator of a hazardous waste so classified must follow special handling and disposal practices as established by the EPA and/or the state.

10. The implication to industries in the near-term battery cycles of Resource Conservation and Recovery Act regulations, when they are finalized, is that additional costs will be incurred by such industries because those wastes classified as hazardous will require special treatment. In general, the regulations do not pose technical barriers for compliance on the part of various segments of the near-term battery industries. Rather, they will result in an economic impact which is currently a matter of debate.

11. It appears that the regulatory limitations will result in a reduction in the number of smaller plants and firms in the battery industry in favor of larger-scale operations.

References

1. The Clean Air Act (42 U.S.C. 1857 et seq.) includes the Clean Air Act of 1963 (P.L. 88-206) and amendments made by the Motor Vehicle Air Pollution Control Act (P.L. 89-272), the Clean Air Act Amendments of 1966 (P.L. 89-675), The Air Quality Act of 1967 (P.L. 90-148), The Clean Air Amendments of 1970 (P.L. 91-604), The Comprehensive Health Manpower Training Act of 1971 (P.L. 92-157), The Energy Supply and Environmental Coordination Act of 1974 (P.L. 93-319), Clean Air Act Amendments of 1977 (P.L. 95-95), and the Safe Drinking Water Act of 1977 (P.L. 95-190).

2. Council on Environmental Quality, *The Ninth Annual Report of the Council on Environmental Quality,* Washington, D.C., 1978.

3. *Economic Impact of the Proposed EPA National Ambient Air Quality Standard for Lead,* prepared by Prather, Suger, Doolittle and Farmer (attornies to the Lead Industries Association, Washington, D.C.), Prepared by Charles River Associates, Inc., Cambridge, MA, CRA Report #398, March 1978.

4. *Standards of Performance for New Stationary Sources: Lead-Acid Battery Manufacture,* 40 CFR (draft), U.S. Environmental Protection Agency, Research Triangle Park, NC, 1978.

5. *Lead-Acid Battery Manufacture Background Information: Proposed Standards,* U.S. Environmental Protection Agency, Washington, D.C., 1979.

6. Amendment to the Solid Waste Disposal Act, P.L. 94-580, 90 Stat. 2795.42 U.S.C., 6901 et seq., 1976.

7. *Draft Economic Impact Analysis of Subtitle C, Resource Conservation and Recovery Act of 1976 (Requestory Analysis Supplement),* prepared by Arthur D. Little, Inc., for The Office of Solid Waste, Hazardous Waste Management Division, U.S. Environmental Protection Agency, January 1979.

8. *Economic Impact Analysis of Hazardous Waste Management Regulations on the Batteries, Electronics and Special Machinery Industries,* to The Office of Solid Waste, Hazardous Waste Management Division, U.S. Environmental Protection Agency under Contract No. 68-01-4714 by Kearney: Management Consultants, February 1978.

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APPENDIX A. GROWTH-RATE SCENARIO FOR LEAD/ACID BATTERY DEVELOPMENT

This appendix presents equations that represent material flow rates in a cycle, growth of the electric vehicle fleet as a function of the time, and battery lifetime. The formulae assume an exponential growth rate, which yields an important simplification; however, the formulae can be easily generalized to any reasonable functional representation of the growth rate.

Typically, the growth rate of any new industry is represented by an S-shaped curve. This can be represented in the following form:

$$N(t) = \frac{N_\infty}{1 + e^{\lambda(t_0 - t)}} \tag{1}$$

where, in this case, $N(t)$ is the number of EVs at time t, N_∞ represents the final number of EVs, λ is a growth rate parameter, and t_0 is the time at which $N(t)$ is halfway to the final number, N_∞. The actual value of N_∞ is of no importance here.

For time t less than t_0 one can write

$$N(t) = \left(\frac{N_\infty}{e^{\lambda(t_0 - t)}}\right)\left(\frac{1}{1 + e^{-\lambda(t_0 - t)}}\right) \simeq N_0 e^{\lambda t} \tag{2}$$

where $N_0 = N_\infty / e^{\lambda t_0}$ and $1/[1 + \exp(-\lambda(t_0 - t))] \simeq 1$ has been used.

This approximation is valid for times t such that $\lambda(t_0 - t) \gtrsim 1$ or $t \lesssim t_0 - (1/\lambda)$. For a growth rate of 25%/year, which is used in this report, $\lambda = 1/4$, which gives the result that the approximation is valid for $t \lesssim t_0 - 4$.

A recent detailed growth model for the EV industry based on Eq. 1 gives values of t_0 ranging from calendar years 2003 to 2008. Because the projections in this report are made only up to the year 2000, the assumption of an exponential growth rate is justified.

The formulae that represent the flow of materials in a cycle under the assumption of an exponential growth rate are obtained as follows:

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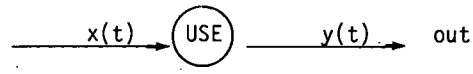

where $x(t)$ gives the total flow rate into use of the new product and $y(t)$ the total flow rate out after use of the product, both at time t. Let $N(t)$ be the total amount of the product in use at time t, λ the annual growth rate of the product, and τ the lifetime (in years) of the

product before it needs replacing. Then $x(t)$ and $y(t)$ as a function of t, λ, and τ are obtained as follows:

In a time interval dt, $N(t)$ increases by the net flow in and decreases by the net flow out. That is:

$$N(t) + x(t)dt - y(t)dt = N(t + dt) .$$

Rearranging gives

$$\frac{dN(t)}{dt} = x(t) - y(t). \tag{3}$$

Because λ gives the annual growth rate, one has, assuming an exponential growth, $dN(t)/dt = \lambda N(t)$, or

$$x(t) - y(t) = \lambda N(t) \tag{4}$$

as the first condition that $x(t)$ and $y(t)$ must satisfy. The second condition to be satisfied is that, for any short-time interval Δ, the amount of used product removed must equal the amount of new product added $t - \tau$ years earlier. That is the condition in which

$$y(t)\Delta = x(t - \tau)\Delta ,$$

or

$$y(t) = x(t - \tau) . \tag{5}$$

One solves Equations 4 and 5 by successive iterations of substituting for $y(t)$ in Eq. 4 by use of Eq. 5, then using Eq. 4 in the result to express $x(t - \tau)$ in terms of $y(t - \tau)$, and then using Eq. 5 again, etc. The first few steps of this process are

$$x(t) = \lambda N(t) + x(t - \tau)$$

$$= \lambda N(t) + \lambda N(t - \tau) + y(t - \tau)$$

$$= \lambda N(t) + \lambda N(t - \tau) + x(t - 2\tau)$$

and so on. After ℓ steps back in time, one has

$$x(t) = \sum_{j=0}^{\ell-1} \lambda N(t - j\tau) + x(t - \ell\tau) \tag{6}$$

The solution of the equation $dN(t)/dt = \lambda N(t)$ gives $N(t) = N_0 e^{\lambda t}$. Substitution of this into Eq. 6 gives

$$x(t) = \sum_{j=0}^{\ell-1} \lambda N_0 e^{\lambda(t-j\tau)} + x(t - \ell\tau)$$

or

$$x(t) = \frac{\lambda N(t)(1 - e^{-\ell\lambda\tau})}{1 - e^{-\lambda\tau}} + x(t - \ell\tau) .$$

As ℓ gets very large exp $(-\ell\lambda\tau)$ and $x(t - \ell\tau)$ get very small. Thus, in the limit $\ell \to \infty$ one has

$$x(t) = \frac{\lambda N(t)}{1 - e^{-\lambda\tau}} . \tag{7}$$

One is interested in the total inflow during a period of time Δ which is short in comparison to one year. (For example, later on, Δ will be set equal to 1/365 or 1 day.) The total inflow from t to t + Δ, denoted by $I_\Delta(t)$, is obtained from Eq. 7 as

$$I_\Delta(t) = \int_t^{t+\Delta} x(t)dt = \frac{\lambda N_o}{1 - e^{-\lambda\tau}} \int_t^{t+\Delta} e^{\lambda t}dt = N(t)\frac{(e^{\lambda\Delta} - 1)}{1 - e^{-\lambda\tau}} \tag{8}$$

Finally, the total inflow between time t and t + Δ per unit of product in use, a(t), is given by

$$a(t) = \frac{I_\Delta(t)}{N(t)} = \frac{e^{\lambda\Delta} - 1}{1 - e^{-\lambda\tau}} \tag{9}$$

The total outflow between t and t + Δ per unit of product in use, b(t), is given by Eqs. 5, 8 and 9 with x(t) replaced by x(t - τ) in Eq. 8. One then gets

$$b(t) = \frac{e^{-\lambda\tau}(e^{\lambda\Delta} - 1)}{1 - e^{-\lambda\tau}} \tag{10}$$

One notes that both a(t) and b(t), which are the desired final results, are independent of the time t. Thus, one can set a(t) = a and b(t) = b.

This independence of a(t) and b(t) from t, which is a consequence of the assumed exponential growth, has an important simplifying consequence. This is that, for each battery type, and value of λ, Δ, and τ, only one computation of flows, emissions and activity levels per unit capacity (e.g., MWh) at various points in the cycle is needed. To obtain total flows, emissions, and other activities at any time, t, one need only multiply the computed values by the total EV fleet size (e.g., in MWh) existing at time t. Without this independence, separate calculations of the cycle emissions, flows, and activity levels would be required for each time t.

Use of Eqs. 9 and 10 with the exponential assumption, gives the following results for the cycle: for the SOA lead/acid cycle, λ = 0.25/yr (25% annual growth); τ = 3.3 years, and Δ = 1/365 (i.e, 1 day). These give a(t) = a = 0.001220/day and b(t) = b = 0.0005346/day. Multiplying these numbers by 18,000 kg lead/MWh gives 21.96 (kg/day)/MWh inflow of lead and 9.62 (kg/day)/MWh outflow of lead. These are the numbers appearing in Table 2.2 and Figure 2.1.

APPENDIX B. CHANGES IN LEAD/ACID BATTERY COMPOSITION DURING DISCHARGE

The overall cell reaction is

$$Pb + PbO_2 + 2H_2SO_4 \rightarrow 2PbSO_4 + 2H_2O \ ,$$

with a cell voltage of 2.05 volts.[1] If E is the energy density of the battery in watt hours per kilogram (Wh/kg), then during complete discharge

$$E/(2.05)(26.8) - 0.0182E$$

moles of electrons are transferred per kilogram. The Faraday constant is 26.8 ampere hours/mole.

From the cell reaction, it is clear that for each mole of electrons transferred, 0.5 mole of Pb, 0.5 mole of PbO_2, and one mole of H_2SO_4 are consumed to give one mole of $PbSO_4$ and one mole of H_2O. Thus, per kilogram of battery weight,

1.89 E grams Pb are used up
2.18 E grams PbO_2 containing 0.29 E grams O_2 are used up
1.78 E grams H_2SO_4 are used up
5.53 E grams $PbSO_4$ are produced
0.33 E grams H_2O are produced

From a value of E and the weight percent composition of the lead/acid battery one can compute the amounts of active materials, oxygen, and electrolyte that are used, and the amounts of $PbSO_4$ and water produced during complete discharge per kilogram of battery weight.

Reference

1. *Review on Lead Acid Battery Science and Technology*, J. Power Sources 2: 3-120, 1977-78.

APPENDIX C. DISPERSION OF STACK AND FUGITIVE EMISSIONS FROM BATTERY-RELATED OPERATIONS

Concentrations and Deposition Rates of Constituents of Emissions from MODEL LEAD MINE-MILL COMPLEX

TABLE C.1. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	COPPER	ZINC	IRON	COBALT	NICKEL
1	2.7E-01	1.2E-02	5.4E-04	7.0E-03	4.3E-03	3.2E-05	4.6E-05
2	9.3E-02	4.2E-03	1.9E-04	2.5E-03	1.5E-03	1.1E-05	1.6E-05
4	3.2E-02	1.4E-03	6.6E-05	8.5E-04	5.2E-04	3.9E-06	5.6E-06
6	1.7E-02	7.8E-04	3.5E-05	4.6E-04	2.8E-04	2.1E-06	3.0E-06
10	8.0E-03	3.6E-04	1.6E-05	2.1E-04	1.3E-04	9.7E-07	1.4E-06
15	4.3E-03	1.9E-04	8.7E-06	1.1E-04	7.0E-05	5.2E-07	7.4E-07
20	2.8E-03	1.2E-04	5.6E-06	7.3E-05	4.5E-05	3.4E-07	4.8E-07
30	1.5E-03	6.6E-05	3.0E-06	3.9E-05	2.4E-05	1.8E-07	2.6E-07
50	6.8E-04	3.0E-05	1.4E-06	1.8E-05	1.1E-05	8.3E-08	1.2E-07

KM FROM MINE-MILL	CADMIUM	MANGANESE	CARBON MONOXIDE	SULFUR OXIDES	HYDRO-CARBONS	ALDEHYDES	ORGANIC ACIDS
1	3.2E-05	4.3E-04	8.7E-04	3.8E-04	4.9E-04	5.4E-05	3.8E-05
2	1.1E-05	1.5E-04	3.0E-04	1.3E-04	1.7E-04	1.9E-05	1.3E-05
4	3.9E-06	5.2E-05	1.0E-04	4.6E-05	5.9E-05	6.6E-06	4.6E-06
6	2.1E-06	2.8E-05	5.6E-05	2.5E-05	3.2E-05	3.5E-06	2.5E-06
10	9.7E-07	1.3E-05	2.6E-05	1.1E-05	1.5E-05	1.6E-06	1.1E-06
15	5.2E-07	7.0E-06	1.4E-05	6.1E-06	7.8E-06	8.7E-07	6.1E-07
20	3.4E-07	4.5E-06	9.0E-06	3.9E-06	5.0E-06	5.6E-07	3.9E-07
30	1.8E-07	2.4E-06	4.8E-06	2.1E-06	2.7E-06	3.0E-07	2.1E-07
50	8.3E-08	1.1E-06	2.2E-06	9.6E-07	1.2E-06	1.4E-07	9.6E-08

TABLE C.2. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	COPPER	ZINC	IRON	COBALT	NICKEL
0- 1	6.6E+05	2.9E+04	1.3E+03	1.7E+04	1.1E+04	8.0E+01	1.1E+02
1- 2	1.0E+05	4.5E+03	2.0E+02	2.6E+03	1.6E+03	1.2E+01	1.7E+01
2- 4	3.6E+04	1.6E+03	7.2E+01	9.4E+02	5.8E+02	4.3E+00	6.1E+00
4- 6	1.6E+04	7.3E+02	3.3E+01	4.3E+02	2.6E+02	2.0E+00	2.8E+00
6- 8	9.8E+03	4.3E+02	2.0E+01	2.6E+02	1.6E+02	1.2E+00	1.7E+00
8-10	6.7E+03	3.0E+02	1.3E+01	1.8E+02	1.1E+02	8.1E-01	1.1E+00
10-15	4.1E+03	1.8E+02	8.2E+00	1.1E+02	6.6E+01	4.9E-01	7.0E-01
15-20	2.4E+03	1.1E+02	4.9E+00	6.4E+01	3.9E+01	3.0E-01	4.2E-01
20-30	1.4E+03	6.3E+01	2.9E+00	3.7E+01	2.3E+01	1.7E-01	2.4E-01
30-50	7.0E+02	3.1E+01	1.4E+00	1.8E+01	1.1E+01	8.5E-02	1.2E-01

KM FROM MINE-MILL	CADMIUM	MANGANESE	CARBON MONOXIDE	SULFUR OXIDES	HYDRO-CARBONS	ALDEHYDES	ORGANIC ACIDS
0- 1	8.0E+01	1.1E+03	2.1E+03	9.3E+02	1.2E+03	1.3E+02	9.3E+01
1- 2	1.2E+01	1.6E+02	3.2E+02	1.4E+02	1.8E+02	2.0E+01	1.4E+01
2- 4	4.3E+00	5.8E+01	1.2E+02	5.0E+01	6.5E+01	7.2E+00	5.0E+00
4- 6	2.0E+00	2.6E+01	5.3E+01	2.3E+01	3.0E+01	3.3E+00	2.3E+00
6- 8	1.2E+00	1.6E+01	3.2E+01	1.4E+01	1.8E+01	2.0E+00	1.4E+00
8 10	8.1E-01	1.1E+01	2.2E+01	9.4E+00	1.2E+01	1.3E+00	9.4E-01
10-15	4.9E-01	6.6E+00	1.3E+01	5.8E+00	7.4E+00	8.2E-01	5.8E-01
15-20	3.0E-01	3.9E+00	7.9E+00	3.5E+00	4.4E+00	4.9E-01	3.5E-01
20-30	1.7E-01	2.3E+00	4.6E+00	2.0E+00	2.6E+00	2.9E-01	2.0E-01
30-50	8.5E-02	1.1E+00	2.3E+00	9.9E-01	1.3E+00	1.4E-01	9.9E-02

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL LEAD MINE-MILL COMPLEX

TABLE C.3. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	COPPER	ZINC	IRON	COBALT	NICKEL
0- 1	1.5E+01	6.5E-01	2.9E-02	3.8E-01	2.4E-01	1.8E-03	2.5E-03
1- 2	2.2E+00	9.9E-02	4.5E-03	5.8E-02	3.6E-02	2.7E-04	3.8E-04
2- 4	7.9E-01	3.5E-02	1.6E-03	2.1E-02	1.3E-02	9.6E-05	1.4E-04
4- 6	3.6E-01	1.6E-02	7.3E-04	9.5E-03	5.9E-03	4.4E-05	6.2E-05
6- 8	2.2E-01	9.7E-03	4.4E-04	5.7E-03	3.5E-03	2.6E-05	3.7E-05
8-10	1.5E-01	6.6E-03	3.0E-04	3.9E-03	2.4E-03	1.8E-05	2.5E-05
10-15	9.1E-02	4.0E-03	1.8E-04	2.4E-03	1.5E-03	1.1E-05	1.6E-05
15-20	5.4E-02	2.4E-03	1.1E-04	1.4E-03	8.8E-04	6.6E-06	9.3E-06
20-30	3.2E-02	1.4E-03	6.4E-05	8.3E-04	5.1E-04	3.8E-06	5.4E-06
30-50	1.5E-02	6.9E-04	3.1E-05	4.1E-04	2.5E-04	1.9E-06	2.7E-06

KM FROM MINE-MILL	CADMIUM	MANGANESE	CARBON MONOXIDE	SULFUR OXIDES	HYDRO-CARBONS	ALDEHYDES	ORGANIC ACIDS
0- 1	1.8E-03	2.4E-02	4.7E-02	2.1E-02	2.7E-02	2.9E-03	2.1E-03
1- 2	2.7E-04	3.6E-03	7.2E-03	3.1E-03	4.0E-03	4.5E-04	3.1E-04
2- 4	9.6E-05	1.3E-03	2.6E-03	1.1E-03	1.4E-03	1.6E-04	1.1E-04
4- 6	4.4E-05	5.9E-04	1.2E-03	5.1E-04	6.6E-04	7.3E-05	5.1E-05
6- 8	2.6E-05	3.5E-04	7.0E-04	3.1E-04	4.0E-04	4.4E-05	3.1E-05
8-10	1.8E-05	2.4E-04	4.8E-04	2.1E-04	2.7E-04	3.0E-05	2.1E-05
10-15	1.1E-05	1.5E-04	2.9E-04	1.3E-04	1.6E-04	1.8E-05	1.3E-05
15-20	6.6E-06	8.3E-05	1.8E-04	7.7E-05	9.8E-05	1.1E-05	7.7E-06
20-30	3.8E-06	5.1E-05	1.0E-04	4.5E-05	5.7E-05	6.4E-06	4.5E-06
30-50	1.9E-06	2.5E-05	5.0E-05	2.2E-05	2.8E-05	3.1E-06	2.2E-06

TABLE C.4. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	COPPER	ZINC	IRON	COBALT	NICKEL
0- 1	6.6E+02	2.9E+01	1.3E+00	1.7E+01	1.1E+01	8.0E-02	1.1E-01
1- 2	1.0E+02	4.5E+00	2.0E-01	2.6E+00	1.6E+00	1.2E-02	1.7E-02
2- 4	3.6E+01	1.6E+00	7.2E-02	9.4E-01	5.8E-01	4.3E-03	6.1E-03
4- 6	1.6E+01	7.3E-01	3.3E-02	4.3E-01	2.6E-01	2.0E-03	2.8E-03
6- 8	9.8E+00	4.3E-01	2.0E-02	2.6E-01	1.6E-01	1.2E-03	1.7E-03
8-10	6.7E+00	3.0E-01	1.3E-02	1.8E-01	1.1E-01	8.1E-04	1.1E-03
10 15	4.1E+00	1.8E-01	8.2E-03	1.1E-01	6.6E-02	4.9E-04	7.0E-04
15-20	2.4E+00	1.1E-01	4.9E-03	6.4E-02	3.9E-02	3.0E-04	4.2E-04
20-30	1.4E+00	6.3E-02	2.9E-03	3.7E-02	2.3E-02	1.7E-04	2.4E-04
30-50	7.0E-01	3.1E-02	1.4E-03	1.8E-02	1.1E-02	8.5E-05	1.2E-04

KM FROM MINE-MILL	CADMIUM	MANGANESE	CARBON MONOXIDE	SULFUR OXIDES	HYDRO-CARBONS	ALDEHYDES	ORGANIC ACIDS
0- 1	8.0E-02	1.1E+00	2.1E+00	9.3E-01	1.2E+00	1.3E-01	9.3E-02
1- 2	1.2E-02	1.6E-01	3.2E 01	1.4E+01	1.8E-01	2.0E-02	1.4E-02
2- 4	4.3E-03	5.8E-02	1.2E-01	5.0E-02	6.5E-02	7.2E-03	5.0E-03
4- 6	2.0E-03	2.6E-02	5.3E-02	2.3E-02	3.0E-02	3.3E-03	2.3E-03
6- 8	1.2E-03	1.6E-02	3.2E-02	1.4E-02	1.8E-02	2.0E-03	1.4E-03
8-10	8.1E-04	1.1E-02	2.2E-02	9.4E-03	1.2E-02	1.3E-03	9.4E-04
10-15	4.9E-04	6.6E-03	1.3E-02	5.8E-03	7.4E-03	8.2E-04	5.8E-04
15-20	3.0E-04	3.9E-03	7.9E-03	3.5E-03	4.4E-03	4.9E-04	3.5E-04
20-30	1.7E-04	2.3E-03	4.6E-03	2.0E-03	2.6E-03	2.9E-04	2.0E-04
30-50	8.5E-05	1.1E-03	2.3E-03	9.9E-04	1.3E-03	1.4E-04	9.9E-05

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL PRIMARY LEAD SMELTING AND REFINING COMPLEX

TABLE C.5. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)/MMH

KM FROM SMELTER	PARTICULATES	LEAD	SULFUR DIOXIDE
1	1.4E-03	4.2E-04	3.2E-05
2	1.9E-03	5.8E-04	4.5E-05
4	1.2E-03	3.6E-04	2.8E-05
6	7.6E-04	2.3E-04	1.8E-05
10	3.9E-04	1.2E-04	9.1E-06
15	2.2E-04	6.7E-05	5.2E-06
20	1.5E-04	4.4E-05	3.4E-06
30	8.1E-05	2.4E-05	1.9E-06
50	3.8E-05	1.1E-05	8.7E-07

TABLE C.6. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MMH

KM FROM SMELTER	PARTICULATES	LEAD	SULFUR DIOXIDE
0- 1	2.6E+04	6.9E+03	1.5E+02
1- 2	4.9E+03	1.4E+03	8.6E+01
2- 4	1.9E+03	5.7E+02	4.1E+01
4- 6	9.2E+02	2.8E+02	2.2E+01
6- 8	5.7E+02	1.7E+02	1.4E+01
8-10	3.9E+02	1.2E+02	1.0E+01
10-15	2.4E+02	7.5E+01	6.4E+00
15-20	1.5E+02	4.6E+01	4.0E+00
20-30	8.8E+01	2.7E+01	2.4E+00
30-50	4.4E+01	1.4E+01	1.2E+00

TABLE C.7. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MMH

KM FROM SMELTER	PARTICULATES	LEAD	SULFUR DIOXIDE
0- 1	5.8E-01	1.5E-01	3.3E-03
1- 2	1.1E-01	3.2E-02	1.9E-03
2- 4	4.2E-02	1.3E-02	9.0E-04
4- 6	2.0E-02	6.2E-03	4.8E-04
6- 8	1.3E-02	3.8E-03	3.1E-04
8-10	8.7E-03	2.7E-03	2.2E-04
10-15	5.4E-03	1.7E-03	1.4E-04
15-20	3.3E-03	1.0E-03	8.8E-05
20-30	1.9E-03	6.0E-04	5.3E-05
30-50	9.7E-04	3.0E-04	2.7E-05

TABLE C.8. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MMH

KM FROM SMELTER	PARTICULATES	LEAD	SULFUR DIOXIDE
0- 1	2.6E+01	6.9E+00	1.5E-01
1- 2	4.9E+00	1.4E+00	8.6E-02
2- 4	1.9E+00	5.7E-01	4.1E-02
4- 6	9.2E-01	2.8E-01	2.2E-02
6- 8	5.7E-01	1.7E-01	1.4E-02
8-10	3.9E-01	1.2E-01	1.0E-02
10-15	2.4E-01	7.5E-02	6.4E-03
15-20	1.5E-01	4.6E-02	4.0E-03
20-30	8.8E-02	2.7E-02	2.4E-03
30-50	4.4E-02	1.4E-02	1.2E-03

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL SECONDARY LEAD SMELTER

TABLE C.9. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)/MWH

KM FROM SMELTER	PARTICULATES	LEAD	ANTIMONY	ARSENIC	SULFUR DIOXIDE
1	1.3E-03	2.9E-04	5.9E-06	1.0E-07	1.6E-03
2	1.8E-03	4.1E-04	8.2E-06	1.4E-07	2.2E-03
4	1.1E-03	2.5E-04	5.1E-06	8.8E-08	1.4E-03
6	7.0E-04	1.6E-04	3.2E-06	5.6E-08	8.7E-04
10	3.6E-04	8.3E-05	1.7E-06	2.9E-08	4.5E-04
15	2.1E-04	4.7E-05	9.4E-07	1.6E-08	2.5E-04
20	1.4E-04	3.1E-05	6.2E-07	1.1E-08	1.7E-04
30	7.5E-05	1.7E-05	3.4E-07	5.9E-09	9.2E-05
50	3.5E-05	8.0E-06	1.6E-07	2.8E-09	4.3E-05

TABLE C.10. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM SMELTER	PARTICULATES	LEAD	ANTIMONY	ARSENIC	SULFUR DIOXIDE
0- 1	3.2E+04	7.4E+03	1.5E+02	2.6E+00	7.2E+03
1- 2	5.1E+03	1.2E+03	2.3E+01	4.0E-01	4.2E+03
2- 4	1.8E+03	4.2E+02	8.4E+00	1.4E-01	2.0E+03
4- 6	8.5E+02	1.9E+02	3.9E+00	6.7E-02	1.1E+03
6- 8	5.1E+02	1.2E+02	2.3E+00	4.0E-02	6.8E+02
8-10	3.5E+02	8.0E+01	1.6E+00	2.8E-02	4.9E+02
10-15	2.1E+02	4.9E+01	9.8E-01	1.7E-02	3.1E+02
15-20	1.3E+02	2.9E+01	5.9E-01	1.0E-02	1.9E+02
20-30	7.5E+01	1.7E+01	3.4E-01	5.9E-03	1.2E+02
30-50	3.7E+01	8.5E+00	1.7E-01	2.9E-03	5.9E+01

TABLE C.11. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM SMELTER	PARTICULATES	LEAD	ANTIMONY	ARSENIC	SULFUR DIOXIDE
0- 1	7.2E-01	1.6E-01	3.3E-03	5.7E-05	1.6E-01
1- 2	1.1E-01	2.6E-02	5.2E-04	8.9E-06	9.3E-02
2- 4	4.1E-02	9.3E-03	1.9E-04	3.2E-06	4.4E-02
4- 6	1.9E-02	4.3E-03	8.6E-05	1.5E-06	2.3E-02
6- 8	1.1E-02	2.6E-03	5.2E-05	9.0E-07	1.5E-02
8-10	7.7E-03	1.8E-03	3.6E-05	6.1E-07	1.1E-02
10-15	4.7E-03	1.1E-03	2.2E-05	3.8E-07	6.9E-03
15-20	2.8E-03	6.5E-04	1.3E-05	2.3E-07	4.3E-03
20-30	1.7E-03	3.8E-04	7.6E-06	1.3E-07	2.6E-03
30-50	8.2E-04	1.9E-04	3.8E-06	6.5E-08	1.3E-03

TABLE C.12. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM SMELTER	PARTICULATES	LEAD	ANTIMONY	ARSENIC	SULFUR DIOXIDE
0- 1	3.2E+01	7.4E+00	1.5E-01	2.6E-03	7.2E+00
1- 2	5.1E+00	1.2E+00	2.3E-02	4.0E-04	4.2E+00
2- 4	1.8E+00	4.2E-01	8.4E-03	1.4E-04	2.0E+00
4- 6	8.5E-01	1.9E-01	3.9E-03	6.7E-05	1.1E+00
6- 8	5.1E-01	1.2E-01	2.3E-03	4.0E-05	6.8E-01
8-10	3.5E-01	8.0E-02	1.6E-03	2.8E-05	4.9E-01
10-15	2.1E-01	4.9E-02	9.8E-04	1.7E-05	3.1E-01
15-20	1.3E-01	2.9E-02	5.9E-04	1.0E-05	1.9E-01
20-30	7.5E-02	1.7E-02	3.4E-04	5.9E-06	1.2E-01
30-50	3.7E-02	8.5E-03	1.7E-04	2.9E-06	5.9E-02

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL LEAD/ACID BATTERY MANUFACTURING PLANT

TABLE C.13. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)/MWH

KM FROM PLANT	PARTICULATES	LEAD	LEAD FUMES	LEAD OXIDES	LEAD SULFATES
1	7.6E-04	3.8E-04	2.6E-05	3.0E-04	4.3E-05
2	2.6E-04	1.3E-04	9.1E-06	1.0E-04	1.5E-05
4	9.2E-05	4.6E-05	3.1E-06	3.6E-05	5.2E-06
6	4.9E-05	2.5E-05	1.7E-06	1.9E-05	2.8E-06
10	2.3E-05	1.1E-05	7.8E-07	8.9E-06	1.3E-06
15	1.2E-05	6.1E-06	4.2E-07	4.8E-06	7.0E-07
20	7.8E-06	3.9E-06	2.7E-07	3.1E-06	4.5E-07
30	4.2E-06	2.1E-06	1.4E-07	1.7E-06	2.4E-07
50	1.9E-06	9.6E-07	6.6E-08	7.6E-07	1.1E-07

KM FROM PLANT	ANTIMONY	ARSENIC	EXPANDER	SEPARATOR	SULFURIC ACID
1	5.1E-07	9.2E-09	7.0E-07	1.9E-07	2.1E-05
2	1.8E-07	3.2E-09	2.5E-07	6.8E-08	7.2E-06
4	6.2E-08	1.1E-09	8.5E-08	2.4E-08	2.5E-06
6	3.4E-08	6.0E-10	4.6E-08	1.3E-08	1.3E-06
10	1.5E-08	2.7E-10	2.1E-08	5.8E-09	6.1E-07
15	8.3E-09	1.5E-10	1.1E-08	3.1E-09	3.3E-07
20	5.3E-09	9.5E-11	7.3E-09	2.0E-09	2.1E-07
30	2.9E-09	5.1E-11	3.9E-09	1.1E-09	1.1E-07
50	1.3E-09	2.3E-11	1.8E-09	5.0E-10	5.2E-08

TABLE C.14. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM PLANT	PARTICULATES	LEAD	LEAD FUMES	LEAD OXIDES	LEAD SULFATES
0- 1	1.9E+03	9.3E+02	6.4E+01	7.3E+02	1.1E+02
1- 2	2.8E+02	1.4E+02	9.7E+00	1.1E+02	1.6E+01
2- 4	1.0E+02	5.0E+01	3.5E+00	4.0E+01	5.8E+00
4- 6	4.6E+01	2.3E+01	1.6E+00	1.8E+01	2.6E+00
6- 8	2.8E+01	1.4E+01	9.5E-01	1.1E+01	1.6E+00
8-10	1.9E+01	9.4E+00	6.5E-01	7.4E+00	1.1E+00
10-15	1.2E+01	5.8E+00	4.0E-01	4.5E+00	6.6E-01
15-20	6.9E+00	3.5E+00	2.4E-01	2.7E+00	3.9E-01
20-30	4.0E+00	2.0E+00	1.4E-01	1.6E+00	2.3E-01
30-50	2.0E+00	9.9E-01	6.8E-02	7.7E-01	1.1E-01

KM FROM PLANT	ANTIMONY	ARSENIC	EXPANDER	SEPARATOR	SULFURIC ACID
0- 1	1.3E+00	2.3E-02	1.7E+00	4.8E-01	5.0E+01
1- 2	1.9E-01	3.4E-03	2.6E-01	7.3E-02	7.7E+00
2- 4	6.8E-02	1.2E-03	9.4E-02	2.6E-02	2.7E+00
4- 6	3.1E-02	5.6E-04	4.3E-02	1.2E-02	1.3E+00
6- 8	1.9E-02	3.4E-04	2.6E-02	7.1E-03	7.5E-01
8-10	1.3E-02	2.3E-04	1.8E-02	4.9E-03	5.1E-01
10-15	7.8E-03	1.4E-04	1.1E-02	3.0E-03	3.1E-01
15-20	4.7E-03	8.4E-05	6.4E-03	1.8E-03	1.9E-01
20-30	2.7E-03	4.9E-05	3.7E-03	1.0E-03	1.1E-01
30-50	1.3E-03	2.4E-05	1.8E-03	5.1E-04	5.4E-02

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL LEAD/ACID BATTERY MANUFACTURING PLANT

TABLE C.15. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM PLANT	PARTICULATES	LEAD	LEAD FUMES	LEAD OXIDES	LEAD SULFATES
0- 1	4.1E-02	2.1E-02	1.4E-03	1.6E-02	2.4E-03
1- 2	6.3E-03	3.1E-03	2.2E-04	2.5E-03	3.6E-04
2- 4	2.2E-03	1.1E-03	7.7E-05	8.8E-04	1.3E-04
4- 6	1.0E-03	5.1E-04	3.5E-05	4.0E-04	5.9E-05
6- 8	6.1E-04	3.1E-04	2.1E-05	2.4E-04	3.5E-05
8-10	4.2E-04	2.1E-04	1.4E-05	1.6E-04	2.4E-05
10-15	2.6E-04	1.3E-04	8.8E-06	1.0E-04	1.5E-05
15-20	1.5E-04	7.7E-05	5.3E-06	6.0E-05	8.8E-06
20-30	8.9E-05	4.5E-05	3.1E-06	3.5E-05	5.1E-06
30-50	4.4E-05	2.2E-05	1.5E-06	1.7E-05	2.5E-06

KM FROM PLANT	ANTIMONY	ARSENIC	EXPANDER	SEPARATOR	SULFURIC ACID
0- 1	2.8E-05	5.0E-07	3.8E-05	1.1E-05	1.1E-03
1- 2	4.3E-06	7.6E-08	5.8E-06	1.6E-06	1.7E-04
2- 4	1.5E-06	2.7E-08	2.1E-06	5.8E-07	6.1E-05
4- 6	7.0E-07	1.2E-08	9.5E-07	2.6E-07	2.8E-05
6- 8	4.2E-07	7.5E-09	5.7E-07	1.6E-07	1.7E-05
8-10	2.8E-07	5.1E-09	3.9E-07	1.1E-07	1.1E-05
10-15	1.7E-07	3.1E-09	2.4E-07	6.6E-08	7.0E-06
15-20	1.0E-07	1.9E-09	1.4E-07	3.9E-08	4.2E-06
20-30	6.1E-08	1.1E-09	8.3E-08	2.3E-08	2.4E-06
30-50	3.0E-08	5.3E-10	4.1E-08	1.1E-08	1.2E-06

TABLE C.16. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM PLANT	PARTICULATES	LEAD	LEAD FUMES	LEAD OXIDES	LEAD SULFATES
0- 1	1.9E+00	9.3E-01	6.4E-02	7.3E-01	1.1E-01
1- 2	2.8E-01	1.4E-01	9.7E-03	1.1E-01	1.6E-02
2- 4	1.0E-01	5.0E-02	3.5E-03	4.0E-02	5.8E-03
4- 6	4.6E-02	2.3E-02	1.6E-03	1.8E-02	2.6E-03
6- 8	2.8E-02	1.4E-02	9.5E-04	1.1E-02	1.6E-03
8-10	1.9E-02	9.4E-03	6.5E-04	7.4E-03	1.1E-03
10-15	1.2E-02	5.8E-03	4.0E-04	4.5E-03	6.6E-04
15-20	6.9E-03	3.5E-03	2.4E-04	2.7E-03	3.9E-04
20-30	4.0E-03	2.0E-03	1.4E-04	1.6E-03	2.3E-04
30-50	2.0E-03	9.9E-04	6.8E-05	7.7E-04	1.1E-04

KM FROM PLANT	ANTIMONY	ARSENIC	EXPANDER	SEPARATOR	SULFURIC ACID
0- 1	1.3E-03	2.3E-05	1.7E-03	4.8E-04	5.0E-02
1- 2	1.9E-04	3.4E-06	2.6E-04	7.3E-05	7.7E-03
2- 4	6.8E-05	1.2E-06	9.4E-05	2.6E-05	2.7E-03
4- 6	3.1E-05	5.6E-07	4.3E-05	1.2E-05	1.3E-03
6- 8	1.9E-05	3.4E-07	2.6E-05	7.1E-06	7.5E-04
8-10	1.3E-05	2.3E-07	1.8E-05	4.9E-06	5.1E-04
10-15	7.8E-06	1.4E-07	1.1E-05	3.0E-06	3.1E-04
15-20	4.7E-06	8.4E-08	6.4E-06	1.8E-06	1.9E-04
20-30	2.7E-06	4.9E-08	3.7E-06	1.0E-06	1.1E-04
30-50	1.3E-06	2.4E-08	1.8E-06	5.1E-07	5.4E-05

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL POLYPROPYLENE MANUFACTURING PLANT

TABLE C.17. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)/MMH

KM FROM PLANT	PARTICULATES	PROPYLENE
1	1.0E-03	2.4E-04
2	3.6E-04	8.2E-05
4	1.2E-04	2.9E-05
6	6.7E-05	1.5E-05
10	3.1E-05	7.0E-06
15	1.7E-05	3.8E-06
20	1.1E-05	2.4E-06
30	5.7E-06	1.3E-06
50	2.6E-06	6.0E-07

TABLE C.18. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MMH

KM FROM PLANT	PARTICULATES	PROPYLENE
0- 1	2.5E+03	5.8E+02
1- 2	3.8E+02	8.8E+01
2- 4	1.4E+02	3.1E+01
4- 6	6.3E+01	1.4E+01
6- 8	3.8E+01	8.6E+00
8-10	2.6E+01	5.9E+00
10-15	1.6E+01	3.6E+00
15-20	9.4E+00	2.1E+00
20-30	5.5E+00	1.3E+00
30-50	2.7E+00	6.1E-01

TABLE C.19. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MMH

KM FROM PLANT	PARTICULATES	PROPYLENE
0- 1	5.6E-02	1.3E-02
1- 2	8.5E-03	2.0E-03
2- 4	3.0E-03	7.0E-04
4- 6	1.4E-03	3.2E-04
6- 8	8.3E-04	1.9E-04
8-10	5.7E-04	1.3E-04
10-15	3.5E-04	8.0E-05
15-20	2.1E-04	4.8E-05
20-30	1.2E-04	2.8E-05
30-50	5.9E-05	1.4E-05

TABLE C.20. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MMH

KM FROM PLANT	PARTICULATES	PROPYLENE
0- 1	2.5E+00	5.8E-01
1- 2	3.8E-01	8.8E-02
2- 4	1.4E-01	3.1E-02
4- 6	6.3E-02	1.4E-02
6- 8	3.8E-02	8.6E-03
8-10	2.6E-02	5.9E-03
10-15	1.6E-02	3.6E-03
15-20	9.4E-03	2.1E-03
20-30	5.5E-03	1.3E-03
30-50	2.7E-03	6.1E-04

Concentrations and Deposition Rates of Constituents of Emissions from MODEL ZINC MINE-MILL COMPLEX

TABLE C.21. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	ZINC	COBALT	NICKEL	CADMIUM	MANGANESE
1	2.7E-04	1.6E-05	1.1E-05	5.4E-08	5.4E-08	5.4E-08	5.4E-07
2	9.4E-05	5.7E-06	3.8E-06	1.9E-08	1.9E-08	1.9E-08	1.9E-07
4	3.3E-05	2.0E-06	1.3E-06	6.6E-09	6.6E-09	6.6E-09	6.6E-08
6	1.8E-05	1.1E-06	7.1E-07	3.5E-09	3.5E-09	3.5E-09	3.5E-08
10	8.1E-06	4.8E-07	3.2E-07	1.6E-09	1.6E-09	1.6E-09	1.6E-08
15	4.3E-06	2.6E-07	1.7E-07	8.7E-10	8.7E-10	8.7E-10	8.7E-09
20	2.8E-06	1.7E-07	1.1E-07	5.6E-10	5.6E-10	5.6E-10	5.6E-09
30	1.5E-06	9.0E-08	6.0E-08	3.0E-10	3.0E-10	3.0E-10	3.0E-09
50	6.9E-07	4.1E-08	2.8E-08	1.4E-10	1.4E-10	1.4E-10	1.4E-09

TABLE C.22. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	ZINC	COBALT	NICKEL	CADMIUM	MANGANESE
0- 1	6.6E+02	4.0E+01	2.7E+01	1.3E-01	1.3E-01	1.3E-01	1.3E+00
1- 2	1.0E+02	6.1E+00	4.0E+00	2.0E-02	2.0E-02	2.0E-02	2.0E-01
2- 4	3.6E+01	2.2E+00	1.4E+00	7.2E-03	7.2E-03	7.2E-03	7.2E-02
4- 6	1.6E+01	9.9E-01	6.6E-01	3.3E-03	3.3E-03	3.3E-03	3.3E-02
6- 8	9.9E+00	5.9E-01	4.0E-01	2.0E-03	2.0E-03	2.0E-03	2.0E-02
8-10	6.7E+00	4.0E-01	2.7E-01	1.3E-03	1.3E-03	1.3E-03	1.3E-02
10-15	4.1E+00	2.5E-01	1.6E-01	8.2E-04	8.2E-04	8.2E-04	8.2E-03
15-20	2.5E+00	1.5E-01	9.9E-02	4.9E-04	4.9E-04	4.9E-04	4.9E-03
20-30	1.4E+00	8.6E-02	5.7E-02	2.9E-04	2.9E-04	2.9E-04	2.9E-03
30-50	7.0E-01	4.2E-02	2.8E-02	1.4E-04	1.4E-04	1.4E-04	1.4E-03

TABLE C.23. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	ZINC	COBALT	NICKEL	CADMIUM	MANGANESE
0- 1	1.5E-02	8.8E-04	5.9E-04	2.9E-06	2.9E-06	2.9E-06	2.9E-05
1- 2	2.2E-03	1.3E-04	9.0E-05	4.5E-07	4.5E-07	4.5E-07	4.5E-06
2- 4	8.0E-04	4.8E-05	3.2E-05	1.6E-07	1.6E-07	1.6E-07	1.6E-06
4- 6	3.7E-04	2.2E-05	1.5E-05	7.3E-08	7.3E-08	7.3E-08	7.3E-07
6- 8	2.2E-04	1.3E-05	8.8E-06	4.4E-08	4.4E-08	4.4E-08	4.4E-07
8-10	1.5E-04	9.0E-06	6.0E-06	3.0E-08	3.0E-08	3.0E-08	3.0E-07
10-15	9.2E-05	5.5E-06	3.7E-06	1.8E-08	1.8E-08	1.8E-08	1.8E-07
15-20	5.5E-05	3.3E-06	2.2E-06	1.1E-08	1.1E-08	1.1E-08	1.1E-07
20-30	3.2E-05	1.9E-06	1.3E-06	6.4E-09	6.4E-09	6.4E-09	6.4E-08
30-50	1.6E-05	9.4E-07	6.3E-07	3.1E-09	3.1E-09	3.1E-09	3.1E-08

TABLE C.24. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM MINE-MILL	PARTICULATES	LEAD	ZINC	COBALT	NICKEL	CADMIUM	MANGANESE
0- 1	6.6E-01	4.0E-02	2.7E-02	1.3E-04	1.3E-04	1.3E-04	1.3E-03
1- 2	1.0E-01	6.1E-03	4.0E-03	2.0E-05	2.0E-05	2.0E-05	2.0E-04
2- 4	3.6E-02	2.2E-03	1.4E-03	7.2E-06	7.2E-06	7.2E-06	7.2E-05
4- 6	1.6E-02	9.9E-04	6.6E-04	3.3E-06	3.3E-06	3.3E-06	3.3E-05
6- 8	9.9E-03	5.9E-04	4.0E-04	2.0E-06	2.0E-06	2.0E-06	2.0E-05
8-10	6.7E-03	4.0E-04	2.7E-04	1.3E-06	1.3E-06	1.3E-06	1.3E-05
10-15	4.1E-03	2.5E-04	1.6E-04	8.2E-07	8.2E-07	8.2E-07	8.2E-06
15-20	2.5E-03	1.5E-04	9.9E-05	4.9E-07	4.9E-07	4.9E-07	4.9E-06
20-30	1.4E-03	8.6E-05	5.7E-05	2.9E-07	2.9E-07	2.9E-07	2.9E-06
30-50	7.0E-04	4.2E-05	2.8E-05	1.4E-07	1.4E-07	1.4E-07	1.4E-06

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL PRIMARY ZINC SMELTER

TABLE C.25. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM SMELTER	ARSENIC	ANTIMONY	CADMIUM	LEAD	ZINC	SULFUR DIOXIDE
1	1.8E-08	1.8E-08	6.3E-07	8.3E-08	1.0E-05	2.4E-03
2	2.4E-08	2.4E-08	8.7E-07	1.2E-07	1.4E-05	3.4E-03
4	1.5E-08	1.5E-08	5.4E-07	7.1E-08	8.7E-06	2.1E-03
6	9.6E-09	9.6E-09	3.4E-07	4.5E-08	5.5E-06	1.3E-03
10	5.0E-09	5.0E-09	1.8E-07	2.3E-08	2.8E-06	6.8E-04
15	2.8E-09	2.8E-09	1.0E-07	1.3E-08	1.6E-06	3.9E-04
20	1.9E-09	1.9E-09	6.6E-08	8.7E-09	1.1E-06	2.5E-04
30	1.0E-09	1.0E-09	3.6E-08	4.8E-09	5.8E-07	1.4E-04
50	4.7E-10	4.7E-10	1.7E-08	2.2E-09	2.7E-07	6.5E-05

TABLE C.26. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM SMELTER	ARSENIC	ANTIMONY	CADMIUM	LEAD	ZINC	SULFUR DIOXIDE
0- 1	8.0E-02	8.0E-02	2.9E+00	3.8E-01	4.6E+01	1.1E+04
1- 2	4.7E-02	4.7E-02	1.7E+00	2.2E-01	2.7E+01	6.4E+03
2- 4	2.2E-02	2.2E-02	7.9E-01	1.0E-01	1.3E+01	3.0E+03
4- 6	1.2E-02	1.2E-02	4.2E-01	5.5E-02	6.7E+00	1.6E+03
6- 8	7.6E-03	7.6E-03	2.7E-01	3.6E-02	4.3E+00	1.0E+03
8-10	5.4E-03	5.4E-03	1.9E-01	2.5E-02	3.1E+00	7.4E+02
10-15	3.5E-03	3.5E-03	1.2E-01	1.6E-02	2.0E+00	4.7E+02
15-20	2.2E-03	2.2E-03	7.7E-02	1.0E-02	1.2E+00	3.0E+02
20-30	1.3E-03	1.3E-03	4.6E-02	6.1E-03	7.4E-01	1.8E+02
30-50	6.6E-04	6.6E-04	2.4E-02	3.1E-03	3.8E-01	9.0E+01

TABLE C.27. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM SMELTER	ARSENIC	ANTIMONY	CADMIUM	LEAD	ZINC	SULFUR DIOXIDE
0- 1	1.0E-06	1.0E-06	6.4E-05	8.4E-06	1.0E-03	2.4E-01
1- 2	1.0E-06	1.0E-06	3.7E-05	4.9E-06	5.9E-04	1.4E-01
2- 4	4.9E-07	4.9E-07	1.7E-05	2.3E-06	2.8E-04	6.7E-02
4- 6	2.6E-07	2.6E-07	9.3E-06	1.2E-06	1.5E-04	3.6E-02
6- 8	1.7E-07	1.7E-07	6.0E-06	7.9E-07	9.6E-05	2.3E-02
8-10	1.2E-07	1.2E-07	4.3E-06	5.7E-07	6.9E-05	1.6E-02
10-15	7.7E-08	7.7E-08	2.7E-06	3.6E-07	4.4E-05	1.1E-02
15-20	4.8E-08	4.8E-08	1.7E-06	2.3E-07	2.7E-05	6.6E-03
20-30	2.9E-08	2.9E-08	1.0E-06	1.4E-07	1.6E-05	4.0E-03
30-50	1.5E-08	1.5E-08	5.2E-07	6.9E-08	8.4E-06	2.0E-03

TABLE C.28. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM SMELTER	ARSENIC	ANTIMONY	CADMIUM	LEAD	ZINC	SULFUR DIOXIDE
0- 1	8.0E-05	8.0E-05	2.9E-03	3.8E-04	4.6E-02	1.1E+01
1- 2	4.7E-05	4.7E-05	1.7E-03	2.2E-04	2.7E-02	6.4E+00
2- 4	2.2E-05	2.2E-05	7.9E-04	1.0E-04	1.3E-02	3.0E+00
4- 6	1.2E-05	1.2E-05	4.2E-04	5.5E-05	6.7E-03	1.6E+00
6- 8	7.6E-06	7.6E-06	2.7E-04	3.6E-05	4.3E-03	1.0E+00
8-10	5.4E-06	5.4E-06	1.9E-04	2.5E-05	3.1E-03	7.4E-01
10-15	3.5E-06	3.5E-06	1.2E-04	1.6E-05	2.0E-03	4.7E-01
15-20	2.2E-06	2.2E-06	7.7E-05	1.0E-05	1.2E-03	3.0E-01
20-30	1.3E-06	1.3E-06	4.6E-05	6.1E-06	7.4E-04	1.8E-01
30-50	6.6E-07	6.6E-07	2.4E-05	3.1E-06	3.8E-04	9.0E-02

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL PRIMARY NICKEL SMELTER

TABLE C.29. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM SMELTER	PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
1	2.0E-03	2.3E-04	5.0E-06	2.0E-05	7.5E-02
2	2.8E-03	3.2E-04	7.0E-06	2.8E-05	1.0E-01
4	1.8E-03	2.0E-04	4.3E-06	1.7E-05	6.5E-02
6	1.1E-03	1.3E-04	2.8E-06	1.1E-05	4.1E-02
10	5.7E-04	6.6E-05	1.4E-06	5.7E-06	2.1E-02
15	3.3E-04	3.7E-05	8.0E-07	3.2E-06	1.2E-02
20	2.1E-04	2.5E-05	5.3E-07	2.1E-06	8.0E-03
30	1.2E-04	1.4E-05	2.9E-07	1.2E-06	4.4E-03
50	5.5E-05	6.3E-06	1.4E-07	5.4E-07	2.0E-03

TABLE C.30. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM SMELTER	PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
0- 1	9.3E+03	1.1E+03	2.3E+01	9.2E+01	3.4E+05
1- 2	5.4E+03	6.2E+02	1.3E+01	5.3E+01	2.0E+05
2- 4	2.6E+03	2.9E+02	6.3E+00	2.5E+01	9.5E+04
4- 6	1.4E+03	1.6E+02	3.4E+00	1.3E+01	5.0E+04
6- 8	8.8E+02	1.0E+02	2.2E+00	8.7E+00	3.2E+04
8-10	6.3E+02	7.2E+01	1.5E+00	6.2E+00	2.3E+04
10-15	4.0E+02	4.6E+01	9.9E-01	4.0E+00	1.5E+04
15-20	2.5E+02	2.9E+01	6.2E-01	2.5E+00	9.2E+03
20-30	1.5E+02	1.7E+01	3.7E-01	1.5E+00	5.6E+03
30-50	7.6E+01	8.7E+00	1.9E-01	7.5E-01	2.8E+03

TABLE C.31. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM SMELTER	PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
0- 1	2.1E-01	2.4E-02	5.1E-04	2.0E-03	7.6E+00
1- 2	1.2E-01	1.4E-02	3.0E-04	1.2E-03	4.4E+00
2- 4	5.7E-02	6.5E-03	1.4E-04	5.6E-04	2.1E+00
4- 6	3.0E-02	3.5E-03	7.5E-05	3.0E-04	1.1E+00
6- 8	1.9E-02	2.2E-03	4.8E-05	1.9E-04	7.2E-01
8-10	1.4E-02	1.6E-03	3.4E-05	1.4E-04	5.1E-01
10-15	8.9E-03	1.0E-03	2.2E-05	8.8E-05	3.3E-01
15-20	5.5E-03	6.4E-04	1.4E-05	5.5E-05	2.1E-01
20-30	3.3E-03	3.8E-04	8.2E-06	3.3E-05	1.2E-01
30-50	1.7E-03	1.9E-04	4.2E-06	1.7E-05	6.3E-02

TABLE C.32. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM SMELTER	PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
0- 1	9.3E+00	1.1E+00	2.3E-02	9.2E-02	3.4E+02
1- 2	5.4E+00	6.2E-01	1.3E-02	5.3E-02	2.0E+02
2- 4	2.6E+00	2.9E-01	6.3E-03	2.5E-02	9.5E+01
4- 6	1.4E+00	1.6E-01	3.4E-03	1.3E-02	5.0E+01
6- 8	8.8E-01	1.0E-01	2.2E-03	8.7E-03	3.2E+01
8-10	6.3E-01	7.2E-02	1.5E-03	6.2E-03	2.3E+01
10-15	4.0E-01	4.6E-02	9.9E-04	4.0E-03	1.5E+01
15-20	2.5E-01	2.9E-02	6.2E-04	2.5E-03	9.2E+00
20-30	1.5E-01	1.7E-02	3.7E-04	1.5E-03	5.6E+00
30-50	7.6E-02	8.7E-03	1.9E-04	7.5E-04	2.8E+00

Concentrations and Deposition Rates of Constituents of Emissions from MODEL CAUSTIC POTASH PLANT

TABLE C.33. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM PRODUCTION COMPLEX	CHLORINE	CARBON MONOXIDE
1	1.0E-07	1.8E-06
2	1.4E-07	2.4E-06
4	8.7E-08	1.5E-06
6	5.5E-08	9.6E-07
10	2.8E-08	5.0E-07
15	1.6E-08	2.8E-07
20	1.1E-08	1.9E-07
30	5.8E-09	1.0E-07
50	2.7E-09	4.7E-08

Concentrations and Deposition Rates of Constituents of Emissions from MODEL NICKEL/ZINC BATTERY MANUFACTURING PLANT

TABLE C.34. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM PLANT	NICKEL	ZINC OXIDE	COBALT	ELECTROLYTE
1	1.3E-03	1.2E-03	6.8E-05	6.8E-06
2	4.5E-04	4.1E-04	2.4E-05	2.4E-06
4	1.6E-04	1.4E-04	8.2E-06	8.2E-07
6	8.5E-05	7.6E-05	4.4E-06	4.4E-07
10	3.9E-05	3.5E-05	2.0E-06	2.0E-07
15	2.1E-05	1.9E-05	1.1E-06	1.1E-07
20	1.3E-05	1.2E-05	7.0E-07	7.0E-08
30	7.2E-06	6.5E-06	3.8E-07	3.8E-08
50	3.3E-06	3.0E-06	1.7E-07	1.7E-08

TABLE C.35. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM PLANT	NICKEL	ZINC OXIDE	COBALT	ELECTROLYTE
0- 1	3.2E+03	2.9E+03	1.7E+02	1.7E+01
1- 2	4.9E+02	4.4E+02	2.5E+01	2.5E+00
2- 4	1.7E+02	1.5E+02	9.0E+00	9.0E-01
4- 6	7.9E+01	7.1E+01	4.1E+00	4.1E-01
6- 8	4.7E+01	4.3E+01	2.5E+00	2.5E-01
8-10	3.2E+01	2.9E+01	1.7E+00	1.7E-01
10-15	2.0E+01	1.8E+01	1.0E+00	1.0E-01
15-20	1.2E+01	1.1E+01	6.2E-01	6.2E-02
20-30	6.9E+00	6.2E+00	3.6E-01	3.6E-02
30-50	3.4E+00	3.0E+00	1.8E-01	1.8E-02

TABLE C.36. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM PLANT	NICKEL	ZINC OXIDE	COBALT	ELECTROLYTE
0- 1	7.1E-02	6.3E-02	3.7E-03	3.7E-04
1- 2	1.1E-02	9.7E-03	5.6E-04	5.6E-05
2- 4	3.8E-03	3.4E-03	2.0E-04	2.0E-05
4- 6	1.8E-03	1.6E-03	9.2E-05	9.2E-06
6- 8	1.1E-03	9.4E-04	5.5E-05	5.5E-06
8-10	7.2E-04	6.4E-04	3.7E-05	3.7E-06
10-15	4.4E-04	3.9E-04	2.3E-05	2.3E-06
15-20	2.6E-04	2.4E-04	1.4E-05	1.4E-06
20-30	1.5E-04	1.4E-04	8.0E-06	8.0E-07
30-50	7.5E-05	6.7E-05	3.9E-06	3.9E-07

TABLE C.37. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM PLANT	NICKEL	ZINC OXIDE	COBALT	ELECTROLYTE
0- 1	3.2E+00	2.9E+00	1.7E-01	1.7E-02
1- 2	4.9E-01	4.4E-01	2.5E-02	2.5E-03
2- 4	1.7E-01	1.5E-01	9.0E-03	9.0E-04
4- 6	7.9E-02	7.1E-02	4.1E-03	4.1E-04
6- 8	4.7E-02	4.3E-02	2.5E-03	2.5E-04
8-10	3.2E-02	2.9E-02	1.7E-03	1.7E-04
10-15	2.0E-02	1.8E-02	1.0E-03	1.0E-04
15-20	1.2E-02	1.1E-02	6.2E-04	6.2E-05
20-30	6.9E-03	6.2E-03	3.6E-04	3.6E-05
30-50	3.4E-03	3.0E-03	1.8E-04	1.8E-05

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL POLYPROPYLENE MANUFACTURING PLANT

TABLE C.38. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM PLANT	PARTICULATES	PROPYLENE
1	3.5E-04	8.4E-05
2	1.2E-04	2.9E-05
4	4.3E-05	1.0E-05
6	2.3E-05	5.5E-06
10	1.1E-05	2.5E-06
15	5.7E-06	1.3E-06
20	3.6E-06	8.7E-07
30	2.0E-06	4.7E-07
50	9.0E-07	2.1E-07

TABLE C.39. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM PLANT	PARTICULATES	PROPYLENE
0- 1	8.6E+02	2.1E+02
1- 2	1.3E+02	3.1E+01
2- 4	4.7E+01	1.1E+01
4- 6	2.1E+01	5.1E+00
6- 8	1.3E+01	3.1E+00
8-10	8.8E+00	2.1E+00
10-15	5.4E+00	1.3E+00
15-20	3.2E+00	7.6E-01
20-30	1.9E+00	4.5E-01
30-50	9.2E-01	2.2E-01

TABLE C.40. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM PLANT	PARTICULATES	PROPYLENE
0- 1	1.9E-02	4.6E-03
1- 2	2.9E-03	7.0E-04
2- 4	1.0E-03	2.5E-04
4- 6	4.8E-04	1.1E-04
6- 8	2.9E-04	6.8E-05
8-10	1.9E-04	4.6E-05
10-15	1.2E-04	2.8E-05
15-20	7.1E-05	1.7E-05
20-30	4.1E-05	9.9E-06
30-50	2.0E-05	4.8E-06

TABLE C.41. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM PLANT	PARTICULATES	PROPYLENE
0- 1	8.6E-01	2.1E-01
1- 2	1.3E-01	3.1E-02
2- 4	4.7E-02	1.1E-02
4- 6	2.1E-02	5.1E-03
6- 8	1.3E-02	3.1E-03
8-10	8.8E-03	2.1E-03
10-15	5.4E-03	1.3E-03
15-20	3.2E-03	7.6E-04
20-30	1.9E-03	4.5E-04
30-50	9.2E-04	2.2E-04

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL IRON AND STEEL COMPLEX

TABLE C.42. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM PLANT	PARTICULATES	CHROMIUM	COPPER	MANGANESE	NICKEL	LEAD	ZINC
1	2.5E-05	2.5E-08	5.0E-08	1.0E-06	5.0E-09	5.0E-07	2.5E-06
2	3.5E-05	3.5E-08	7.0E-08	1.4E-06	7.0E-09	7.0E-07	3.5E-06
4	2.2E-05	2.2E-08	4.3E-08	8.7E-07	4.3E-09	4.3E-07	2.2E-06
6	1.4E-05	1.4E-08	2.8E-08	5.5E-07	2.8E-09	2.8E-07	1.4E-06
10	7.1E-06	7.1E-09	1.4E-08	2.8E-07	1.4E-09	1.4E-07	7.1E-07
15	4.0E-06	4.0E-09	8.0E-09	1.6E-07	8.0E-10	8.0E-08	4.0E-07
20	2.7E-06	2.7E-09	5.3E-09	1.1E-07	5.3E-10	5.3E-08	2.7E-07
30	1.5E-06	1.5E-09	2.9E-09	5.8E-08	2.9E-10	2.9E-08	1.5E-07
50	6.8E-07	6.8E-10	1.4E-09	2.7E-08	1.4E-10	1.4E-08	6.8E-08

TABLE C.43. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM PLANT	PARTICULATES	CHROMIUM	COPPER	MANGANESE	NICKEL	LEAD	ZINC
0- 1	1.1E+02	1.1E-01	2.3E-01	4.6E+00	2.3E-02	2.3E+00	1.1E+01
1- 2	6.7E+01	6.7E-02	1.3E-01	2.7E+00	1.3E-02	1.3E+00	6.7E+00
2- 4	3.2E+01	3.2E-02	6.3E-02	1.3E+00	6.3E-03	6.3E-01	3.2E+00
4- 6	1.7E+01	1.7E-02	3.4E-02	6.7E-01	3.4E-03	3.4E-01	1.7E+00
6- 8	1.1E+01	1.1E-02	2.2E-02	4.3E-01	2.2E-03	2.2E-01	1.1E+00
8-10	7.7E+00	7.7E-03	1.5E-02	3.1E-01	1.5E-03	1.5E-01	7.7E-01
10-15	4.9E+00	4.9E-03	9.9E-03	2.0E-01	9.9E-04	9.9E-02	4.9E-01
15-20	3.1E+00	3.1E-03	6.2E-03	1.2E-01	6.2E-04	6.2E-02	3.1E-01
20-30	1.9E+00	1.9E-03	3.7E-03	7.4E-02	3.7E-04	3.7E-02	1.9E-01
30-50	9.4E-01	9.4E-04	1.9E-03	3.8E-02	1.9E-04	1.9E-02	9.4E-02

TABLE C.44. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM PLANT	PARTICULATES	CHROMIUM	COPPER	MANGANESE	NICKEL	LEAD	ZINC
0- 1	2.5E-03	2.5E-06	5.1E-06	1.0E-04	5.1E-07	5.1E-05	2.5E-04
1- 2	1.5E-03	1.5E-06	3.0E-06	5.9E-05	3.0E-07	3.0E-05	1.5E-04
2- 4	7.0E-04	7.0E-07	1.4E-06	2.8E-05	1.4E-07	1.4E-05	7.0E-05
4- 6	3.7E-04	3.7E-07	7.5E-07	1.5E-05	7.5E-08	7.5E-06	3.7E-05
6- 8	2.4E-04	2.4E-07	4.8E-07	9.6E-06	4.8E-08	4.8E-06	2.4E-05
8-10	1.7E-04	1.7E-07	3.4E-07	6.9E-06	3.4E-08	3.4E-06	1.7E-05
10-15	1.1E-04	1.1E-07	2.2E-07	4.4E-06	2.2E-08	2.2E-06	1.1E-05
15-20	6.8E-05	6.8E-08	1.4E-07	2.7E-06	1.4E-08	1.4E-06	6.8E-06
20-30	4.1E-05	4.1E-08	8.2E-08	1.6E-06	8.2E-09	8.2E-07	4.1E-06
30-50	2.1E-05	2.1E-08	4.2E-08	8.4E-07	4.2E-09	4.2E-07	2.1E-06

TABLE C.45. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM PLANT	PARTICULATES	CHROMIUM	COPPER	MANGANESE	NICKEL	LEAD	ZINC
0- 1	1.1E-01	1.1E-04	2.3E-04	4.6E-03	2.3E-05	2.3E-03	1.1E-02
1- 2	6.7E-02	6.7E-05	1.3E-04	2.7E-03	1.3E-05	1.3E-03	6.7E-03
2- 4	3.2E-02	3.2E-05	6.3E-05	1.3E-03	6.3E-06	6.3E-04	3.2E-03
4- 6	1.7E-02	1.7E-05	3.4E-05	6.7E-04	3.4E-06	3.4E-04	1.7E-03
6- 8	1.1E-02	1.1E-05	2.2E-05	4.3E-04	2.2E-06	2.2E-04	1.1E-03
8-10	7.7E-03	7.7E-06	1.5E-05	3.1E-04	1.5E-06	1.5E-04	7.7E-04
10-15	4.9E-03	4.9E-06	9.9E-06	2.0E-04	9.9E-07	9.9E-05	4.9E-04
15-20	3.1E-03	3.1E-06	6.2E-06	1.2E-04	6.2E-07	6.2E-05	3.1E-04
20-30	1.9E-03	1.9E-06	3.7E-06	7.4E-05	3.7E-07	3.7E-05	1.9E-04
30-50	9.4E-04	9.4E-07	1.9E-06	3.8E-05	1.9E-07	1.9E-05	9.4E-05

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL LITHIUM HYDROXIDE PLANT

TABLE C.46. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS, (MICROGRAMS/M**3)/MWH

KM FROM PLANT	PARTICULATES
1	2.0E-05
2	2.8E-05
4	1.7E-05
6	1.1E-05
10	5.7E-06
15	3.2E-06
20	2.1E-06
30	1.2E-06
50	5.4E-07

TABLE C.47. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM PLANT	PARTICULATES
0- 1	9.2E+01
1- 2	5.3E+01
2- 4	2.5E+01
4- 6	1.3E+01
6- 8	8.7E+00
8-10	6.2E+00
10-15	4.0E+00
15-20	2.5E+00
20-30	1.5E+00
30-50	7.5E-01

TABLE C.48. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM PLANT	PARTICULATES
0- 1	2.0E-03
1- 2	1.2E-03
2- 4	5.6E-04
4- 6	3.0E-04
6- 8	1.9E-04
8-10	1.4E-04
10-15	8.8E-05
15-20	5.5E-05
20-30	3.3E-05
30-50	1.7E-05

TABLE C.49. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM PLANT	PARTICULATES
0- 1	9.2E-02
1- 2	5.3E-02
2- 4	2.5E-02
4- 6	1.3E-02
6- 8	8.7E-03
8-10	6.2E-03
10-15	4.0E-03
15-20	2.5E-03
20-30	1.5E-03
30-50	7.5E-04

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL PRIMARY NICKEL SMELTER

TABLE C.50. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MMH

KM FROM SMELTER	TOTAL PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
1	1.3E-03	1.5E-04	2.5E-06	1.3E-05	4.8E-02
2	1.8E-03	2.1E-04	3.5E-06	1.7E-05	6.6E-02
4	1.1E-03	1.3E-04	2.2E-06	1.1E-05	4.1E-02
6	7.0E-04	8.3E-05	1.4E-06	6.9E-06	2.6E-02
10	3.6E-04	4.3E-05	7.1E-07	3.5E-06	1.3E-02
15	2.0E-04	2.4E-05	4.0E-07	2.0E-06	7.6E-03
20	1.4E-04	1.6E-05	2.7E-07	1.3E-06	5.0E-03
30	7.4E-05	8.7E-06	1.5E-07	7.3E-07	2.8E-03
50	3.5E-05	4.1E-06	6.8E-08	3.4E-07	1.3E-03

TABLE C.51. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MMH

KM FROM SMELTER	TOTAL PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
0- 1	5.9E+03	6.9E+02	1.1E+01	5.7E+01	2.2E+05
1- 2	3.4E+03	4.0E+02	6.7E+00	3.3E+01	1.3E+05
2- 4	1.6E+03	1.9E+02	3.2E+00	1.6E+01	6.0E+04
4- 6	8.6E+02	1.0E+02	1.7E+00	8.4E+00	3.2E+04
6- 8	5.5E+02	6.5E+01	1.1E+00	5.4E+00	2.1E+04
8-10	3.9E+02	4.6E+01	7.7E-01	3.9E+00	1.5E+04
10-15	2.5E+02	3.0E+01	4.9E-01	2.5E+00	9.4E+03
15-20	1.6E+02	1.8E+01	3.1E-01	1.5E+00	5.8E+03
20-30	9.5E+01	1.1E+01	1.9E-01	9.3E-01	3.5E+03
30-50	4.8E+01	5.6E+00	9.4E-02	4.7E-01	1.8E+03

TABLE C.52. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MMH

KM FROM SMELTER	TOTAL PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
0- 1	1.3E-01	1.5E-02	2.5E-04	1.3E-03	4.8E+00
1- 2	7.5E-02	8.9E-03	1.5E-04	7.4E-04	2.8E+00
2- 4	3.6E-02	4.2E-03	7.0E-05	3.5E-04	1.3E+00
4- 6	1.9E-02	2.2E-03	3.7E-05	1.9E-04	7.1E-01
6- 8	1.2E-02	1.4E-03	2.4E-05	1.2E-04	4.6E-01
8-10	8.7E-03	1.0E-03	1.7E-05	8.6E-05	3.3E-01
10-15	5.6E-03	6.6E-04	1.1E-05	5.5E-05	2.1E-01
15-20	3.5E-03	4.1E-04	6.8E-06	3.4E-05	1.3E-01
20-30	2.1E-03	2.5E-04	4.1E-06	2.1E-05	7.8E-02
30-50	1.1E-03	1.3E-04	2.1E-06	1.0E-05	4.0E-02

TABLE C.53. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MMH

KM FROM SMELTER	TOTAL PARTICULATES	NICKEL	COBALT	LEAD	SULFUR DIOXIDE
0- 1	5.9E+00	6.9E-01	1.1E-02	5.7E-02	2.2E+02
1- 2	3.4E+00	4.0E-01	6.7E-03	3.3E-02	1.3E+02
2- 4	1.6E+00	1.9E-01	3.2E-03	1.6E-02	6.0E+01
4- 6	8.6E-01	1.0E-01	1.7E-03	8.4E-03	3.2E+01
6- 8	5.5E-01	6.5E-02	1.1E-03	5.4E-03	2.1E+01
8-10	3.9E-01	4.6E-02	7.7E-04	3.9E-03	1.5E+01
10-15	2.5E-01	3.0E-02	4.9E-04	2.5E-03	9.4E+00
15-20	1.6E-01	1.8E-02	3.1E-04	1.5E-03	5.8E+00
20-30	9.5E-02	1.1E-02	1.9E-04	9.3E-04	3.5E+00
30-50	4.8E-02	5.6E-03	9.4E-05	4.7E-04	1.8E+00

Concentrations and Deposition Rates of Constituents of Emissions from MODEL CAUSTIC POTASH PLANT

TABLE C.54. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM REFINERY	CHLORINE	CARBON MONOXIDE
1	7.5E-08	1.5E-06
2	1.0E-07	2.1E-06
4	6.5E-08	1.3E-06
6	4.1E-08	8.3E-07
10	2.1E-08	4.3E-07
15	1.2E-08	2.4E-07
20	8.0E-09	1.6E-07
30	4.4E-09	8.7E-08
50	2.0E-09	4.1E-08

Concentrations and Deposition Rates of Constituents of Emissions from MODEL NICKEL/IRON BATTERY MANUFACTURING PLANT

TABLE C.55. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM PLANT	STEEL	FERRIC OXIDE	ELECTROLYTE
1	1.9E-03	1.4E-03	1.4E-05
2	6.6E-04	4.7E-04	4.7E-06
4	2.3E-04	1.6E-04	1.6E-06
6	1.2E-04	8.8E-05	8.8E-07
10	5.7E-05	4.0E-05	4.0E-07
15	3.0E-05	2.2E-05	2.2E-07
20	2.0E-05	1.4E-05	1.4E-07
30	1.1E-05	7.5E-06	7.5E-08
50	4.8E-06	3.4E-06	3.4E-08

TABLE C.56. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM PLANT	STEEL	FERRIC OXIDE	ELECTROLYTE
0- 1	4.6E+03	3.3E+03	3.3E+01
1- 2	7.1E+02	5.1E+02	5.1E+00
2- 4	2.5E+02	1.8E+02	1.8E+00
4- 6	1.2E+02	8.2E+01	8.2E-01
6- 8	6.9E+01	4.9E+01	4.9E-01
8-10	4.7E+01	3.4E+01	3.4E-01
10-15	2.9E+01	2.1E+01	2.1E-01
15-20	1.7E+01	1.2E+01	1.2E-01
20-30	1.0E+01	7.2E+00	7.2E-02
30-50	4.9E+00	3.5E+00	3.5E-02

TABLE C.57. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM PLANT	STEEL	FERRIC OXIDE	ELECTROLYTE
0- 1	1.0E-01	7.4E-02	7.4E-04
1- 2	1.6E-02	1.1E-02	1.1E-04
2- 4	5.6E-03	4.0E-03	4.0E-05
4- 6	2.6E-03	1.8E-03	1.8E-05
6- 8	1.5E-03	1.1E-03	1.1E-05
8-10	1.0E-03	7.5E-04	7.5E-06
10-15	6.4E-04	4.6E-04	4.6E-06
15-20	3.8E-04	2.7E-04	2.7E-06
20-30	2.2E-04	1.6E-04	1.6E-06
30-50	1.1E-04	7.8E-05	7.8E-07

TABLE C.58. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM PLANT	STEEL	FERRIC OXIDE	ELECTROLYTE
0- 1	4.6E+00	3.3E+00	3.3E-02
1- 2	7.1E-01	5.1E-01	5.1E-03
2- 4	2.5E-01	1.8E-01	1.8E-03
4- 6	1.2E-01	8.2E-02	8.2E-04
6- 8	6.9E-02	4.9E-02	4.9E-04
8-10	4.7E-02	3.4E-02	3.4E-04
10-15	2.9E-02	2.1E-02	2.1E-04
15-20	1.7E-02	1.2E-02	1.2E-04
20-30	1.0E-02	7.2E-03	7.2E-05
30-50	4.9E-03	3.5E-03	3.5E-05

Concentrations and Deposition Rates of Constituents of Emissions from
MODEL POLYPROPYLENE MANUFACTURING PLANT

TABLE C.59. GROUND-LEVEL ATMOSPHERIC CONCENTRATIONS,(MICROGRAMS/M**3)/MWH

KM FROM COMPLEX	PARTICULATES	PROPYLENE
1	5.4E-04	1.2E-04
2	1.9E-04	4.2E-05
4	6.6E-05	1.5E-05
6	3.5E-05	7.9E-06
10	1.6E-05	3.6E-06
15	8.7E-06	2.0E-06
20	5.6E-06	1.3E-06
30	3.0E-06	6.8E-07
50	1.4E-06	3.1E-07

TABLE C.60. DEPOSITION RATES, (MICROGRAMS/M**2/YR)/MWH

KM FROM COMPLEX	PARTICULATES	PROPYLENE
0- 1	1.3E+03	3.0E+02
1- 2	2.0E+02	4.6E+01
2- 4	7.2E+01	1.6E+01
4- 6	3.3E+01	7.4E+00
6- 8	2.0E+01	4.4E+00
8-10	1.3E+01	3.0E+00
10-15	8.2E+00	1.9E+00
15-20	4.9E+00	1.1E+00
20-30	2.9E+00	6.5E-01
30-50	1.4E+00	3.2E-01

TABLE C.61. INCREMENTAL SOIL CONCENTRATIONS, (MICROGRAMS/G/YR)/MWH

KM FROM COMPLEX	PARTICULATES	PROPYLENE
0- 1	2.9E-02	6.6E-03
1- 2	4.5E-03	1.0E-03
2- 4	1.6E-03	3.6E-04
4- 6	7.3E-04	1.6E-04
6- 8	4.4E-04	9.9E-05
8-10	3.0E-04	6.7E-05
10-15	1.8E-04	4.1E-05
15-20	1.1E-04	2.5E-05
20-30	6.4E-05	1.4E-05
30-50	3.1E-05	7.0E-06

TABLE C.62. RUNOFF CONCENTRATIONS, (MICROGRAMS/L)/MWH

KM FROM COMPLEX	PARTICULATES	PROPYLENE
0- 1	1.3E+00	3.0E-01
1- 2	2.0E-01	4.6E-02
2- 4	7.2E-02	1.6E-02
4- 6	3.3E-02	7.4E-03
6- 8	2.0E-02	4.4E-03
8-10	1.3E-02	3.0E-03
10-15	8.2E-03	1.9E-03
15-20	4.9E-03	1.1E-03
20-30	2.9E-03	6.5E-04
30-50	1.4E-03	3.2E-04

APPENDIX D. METHODOLOGY FOR ESTIMATING POPULATION EXPOSURE TO TOTAL SUSPENDED PARTICULATES (TSP) AND SO_2 RESULTING FROM CENTRAL POWER STATION EMISSIONS FOR THE DAILY BATTERY CHARGING DEMAND OF 10,000 ELECTRIC VEHICLES

Nelson et al.[1] define the energy requirement of the wheels of an electric vehicle as 0.22 kWh/ton·mile. Such a vehicle conforms to the profile of a small four-passenger commuter car weighing about 2800 lb and driven 10,000 miles per year. The daily energy requirements for six different four-passenger electric vehicles are presented in Table D.1, together with pertinent characteristics for each vehicle.

Energy is always transferred at less than 100% efficiency. The transfer efficiencies between various steps in an electric vehicle system are shown in Table D.2. As can be seen from this table, the energy delivered to the wheels is only a fraction ($\sim$ 20%) of the overall energy demand of the system. Expressing this result in terms of total demand, the system energy requirement is between 1.5 to 2.3 times the energy required for the wheels of the vehicle. The overall energy demand, which includes efficiency factors for power generation, can be shown to be between 3.8 and 7.8 times the final energy requirement. Table D.3 describes the daily demand for electric vehicles of the types defined in Table D.1.

For a fleet of 10,000 vehicles of the types in Table D.3 the daily electrical power demand is expected to be on the order of 200 MWh. Assuming a six- to eight-hour recharging period each day, this energy demand represents a constant power output of 29 MWe (22.3-37.1). In Table D.4 the electrical power production required to meet the daily energy demand of 10,000 electric vehicles is indicated, assuming six- and eight-hour recharging periods.

In this analysis it is assumed that a single power station will supply the electrical energy to meet the daily demand. Further, we assume that the plant is coal-fired and is operated so as to conform to new source performance standards (NSPS) for the emission of SO_2 and TSP (1.2 lb SO_2 and 0.1 lb TSP per 10^6 Btu input heat). In Table D.3 the second column, "overall energy demand," is calculated from the daily energy requirement by incorporating the conversion efficiency of the power plant (20% to 40%) into the daily demand estimate. Input heat for a plant with a heat rate of 9850 Btu/kWh is shown in Table D.5. The allowable emission rates, under NSPS, for SO_2 and TSP are given in Table D.6.

Determinations of the maximum ground-level concentrations for the emission rates in Table D.6 were carried out using Ranchoux's technique for evaluating Pasquill-Gifford's and Holland's equations.[2] Estimates for the maximum ground-level concentrations are given in Table D.7. These estimates were calculated under the assumptions of C and D class atmospheric stability, an effective stack height of 400 m (stack = 150 m, plume rise = 250 m), and a wind velocity of 2 m/s.

Table D.1. Four-Passenger Electric Vehicle Characteristics

Electric Vehicle Type			Battery		
	Weight, tons	Range, miles	System	Energy Density, Wh/kg	Daily Energy Requirements, kWh/day[a]
A	1.547	50	Pb/Acid	44	13.50
B	1.547	50	Pb/Acid	47	13.09
C	1.409	50	Ni/Fe	60	11.92
D	1.375	50	Ni/Fe	65	11.63
E	1.649	90	Ni/Zn	81	13.95
F	1.716	100	Ni/Zn	85	14.52

Data from M. Singh et al., "EHV Programmatic Environmental Assessment; Future EV Weight and Range Characteristics," Draft Report, Argonne National Laboratory, 1978.

[a]*Assumes ∿ 40 miles/day use and 0.22 kWh/ton·mile energy requirement.*

Table D.2. Energy Conversion Efficiencies between Steps in an Electric Vehicle Charging System, % Input

Charge System	Low	High
Generating Plant	30	40
Electrical Transmission	90	95
Battery		
Charge	86	95
Discharge	75	85
Electrical to Mechanical	75	85
Overall	12.9	26.1
Average	19.5	

Data from P. A. Nelson et al., "The Need for Developmental High-energy Batteries for Electric Automobiles," ANL 8075, 1974.

Table D.3. Daily Energy Demand for an Electric Vehicle

Vehicle Type[a]	Daily Electrical Demand, kWh/day	Overall Energy Demand, kWh/day
A	20.70 - 31.37	51.72 - 104.65
B	20.07 - 30.41	50.14 - 101.44
C	18.29 - 27.71	45.68 - 92.42
D	17.85 - 27.04	44.58 - 90.19
E	21.39 - 32.42	53.45 - 108.15
F	22.27 - 33.74	55.62 - 112.54

[a]*Defined in Table D.1.*

Table D.4. Daily Electrical Power Level to Meet the Energy Demand of 10,000 Electric Vehicles (high-system efficiency)

Vehicle Type[a]	Eight-Hour Recharge, MWe	Six-Hour Recharge, MWe
A	25.9	34.5
B	25.1	33.4
C	22.9	30.4
D	22.3	29.7
E	26.7	35.7
F	27.9	37.1

[a]*Defined in Table D.1.*

Table D.5. Input Heat Rates for a Coal-fired Power Plant with a Heat Rate of 9850 Btu/kWh Supplying Energy to 10,000 Electric Vehicles at Two Battery Charging Rates

Vehicle Type[a]	Eight-Hour Charge, 10^6 Btu/hr	Six-Hour Charge, 10^6 Btu/hr
A	254.9	339.8
B	247.1	329.4
C	225.2	300.3
D	219.8	293.0
E	263.4	351.2
F	274.2	365.6

[a] Defined in Table D.1.

Table D.6. Allowable Emission Rates under New Source Performance Standards for TSP (0.1 lb/10^6 Btu) and SO_2 (1.2 lb/10^6 Btu)

	Eight-hour Charge		Six-hour Charge	
Vehicle Type[a]	TSP, lb/hr	SO_2, lb/hr	TSP, lb/hr	SO_2, lb/hr
A	25.4	305.9	34.0	407.8
B	24.7	296.5	32.9	395.3
C	22.5	270.2	30.0	360.4
D	22.0	263.8	29.3	351.6
E	26.3	316.1	35.1	421.4
F	27.4	329.0	36.6	438.7

[a] Defined in Table D.1.

Table D.7. Maximum Ground-level Concentrations ($\mu g/m^3$) of TSP and SO_2 Corresponding to the Emission Rates in Table D.6[a]

	Stability Class C				Stability Class D			
	Low		High		Low		High	
Vehicle Type[b]	TSP	SO_2	TSP	SO_2	TSP	SO_2	TSP	SO_2
A	1.40	16.9	1.88	22.6	0.40	4.80	0.53	6.40
B	1.37	16.4	1.82	21.9	0.39	4.65	0.52	6.21
C	1.24	14.9	1.66	19.9	0.35	4.23	0.47	5.64
D	1.22	15.0	1.62	19.4	0.34	4.24	0.46	5.51
E	1.46	17.5	1.94	23.3	0.41	4.96	0.55	6.61
F	1.52	18.2	2.03	24.3	0.43	5.15	0.57	6.88

[a] Effective stack height = 400 m; wind velocity = 2 mph.

[b] Defined in Table D.1.

The concentration estimates in Table D.7 represent the maximum ground-level concentrations at the center line of plume with dispersion coefficients valid for sampling times of ten minutes. For the present analysis, a compensation factor of 0.01 was applied to the estimates to allow for off-centerline population exposures (within 90% of the maximum value), which were averaged over a yearly exposure.

References

1. P. A. Nelson et al., *The Need for Developmental High-energy Batteries for Electric Automobiles*, ANL 8075, Argonne National Laboratory, Argonne, Illinois, 1974.

2. R. J. P. Ranchoux, *Determination of Maximum Ground Level Concentration*, J. Air Pollu. Control Assoc. 26(11): 1088-1089, 1976.

APPENDIX E. DETERMINATION OF ARSENIC AIR EMISSIONS FROM ZINC SMELTING

A. OPTIMISTIC ESTIMATE

1. Assumptions
   - 90% of the zinc is recycled after battery use.
   - The burden is spread over the lifetime of the battery (9.4 yrs).
   - 99.85% of the particulates are removed.
   - Electrolytic process is used exclusively to produce zine for batteries.

2. Calculation
   - Flow is 7×10^{-7} kg As/MWh per day, as in Table 3.5, or 0.0003 kg/MT Zn, given an annual total of 2.6×10^{-4} kg As/MWh (365 days $\times$ 7×10^{-7} kg As/MWh).
   - 200,000 MWh produced in year 2000 (Table 3.7); 200,000 MWh $\times$ 2.6×10^{-4} kg As/MWh = 52 kg As in 2000.

B. CONSERVATIVE ESTIMATE

1. Assumptions
   - No recycling, as industry does not yet exist.
   - Arsenic burden is for specific years in which the battery is produced, not spread over battery lifetime.
   - Emission factor is 0.65 kg As/MT zinc, the estimated emission rate for existing smelters in 1968. This is not an unreasonable assumption, as the mandatory compliance date for nonferrous smelters has recently been set back to 1983 and the possibility exists for the extension of the compliance deadline to 1988.[1] Additionally, as the zinc smelting industry is not anticipated to be seriously impacted and only two new zinc smelters may be necessary (p. 3-11), standards for existing nonferrous smelters, and not new source performance standards, will affect most of the zine smeltered for batteries.

2. Calculations for the Year 2000
   - From Table 3.7: $(2 \times 10^5$ MWh installed batteries$)/(8 \times 10^6$ EV) = 25 kWh/EV.
   - (25 kWh/EV)/(77 Wh/kg) = $\frac{25}{77} \cdot \frac{\text{kWh}}{\text{EV}} \cdot \frac{\text{kg}}{\text{Wh}}$ = 0.325 MT battery/EV.
   - (0.325 MT battery/EV) $\times$ 18.47% Zn/battery = 0.06 MT Zn/EV.
   - (0.06 MT Zn/EV) $\times$ 8×10^6 EV = 4.8×10^5 MT Zn.
   - $(4.8 \times 10^5$ MT Zn) $\times$ (0.65 kg As/MT Zn) = 312 MT As.

Reference

1. L.J. Lundquist, *Who is Winning the Race for Clean Air? An Evaluation of the Impacts of the U.S. and Swedish Approaches to Air Pollution Control*, Ambio 8: 144-151, 1979.

APPENDIX F. DETERMINATION OF ARSENIC AIR EMISSIONS FROM PRIMARY LEAD SMELTING

A. OPTIMISTIC ESTIMATE - insufficient data.

B. CONSERVATIVE ESTIMATE

1. Assumptions
   - 80% of lead is recycled, so only 20% is primary-smelted as is the case at present.
   - Arsenic burden is for year in which the lead is smelted and not carried for lifetime of battery.
   - Emission factor is 0.40 kg As/MT Pb.
   - Cumulative number of batteries produced is as given in Section 2.2.

2. Calculations for the Year 2000
   - From page 8: $(25 \text{ kWh/EV})/35 \text{ Wh/kg} = \frac{25}{35} \times \frac{\text{kWh}}{\text{EV}} \times \frac{\text{kg}}{\text{Wh}} = 0.714 \text{ MT battery/EV}$.
   - (0.714 MT battery/EV) × (63% Pb/battery) = 0.450 MT Pb/EV.
   - (0.450 MT Pb/EV) × (3×10^6 EVs in 2000 = 1,350,000 MT Pb.
   - 1,350,000 MT Pb × 20% = 270,000 MT primary smelted Pb.
   - 270,000 MT Pb × (0.4 kg As/MT Pb) = 108 MT As.

APPENDIX G. HEALTH EFFECTS: RESEARCH RECOMMENDATIONS RELATED TO EV BATTERY TECHNOLOGIES

Analysis of the health effects assessment has identified research projects or areas of investigation which are uniquely related to some aspect of EV production or to those agents whose use level in the U.S. would significantly increase with projected increases in EV production. The identified projects are listed below.

A. LEAD/ACID BATTERY SYSTEM (Pb, As, Sb)

1. Measurements of (a) ambient levels of stibine produced and (b) amounts of antimony trioxide and arsenic trioxide released and accumulated as a result of normal and equalization charging of lead/acid EV batteries containing antimony: Measurements have so far been reported only for load-leveling batteries, and the total amount of stibine produced inside the battery rather than that amount actually released during charging was measured.

2. Stibine toxicity under low-level exposure conditions: The TLV for stibine has been set based on its analogy to arsine. Data on the toxicity of stibine itself are extremely limited. Levels studied should include those measured in project 1 above.

3. Antimony trioxide toxicity in subsections of the general population, e.g., the young, pregnant, and old: Generation of stibine in the garage with its subsequent decomposition to antimony trioxide would result in exposure of the general public to antimony trioxide. Toxic responses in subsections of the population which might be most sensitive to its effects should be analyzed.

4. Animal studies on the effects of lead on the developing nervous system: These effects are of most concern with respect to increases in environmental exposure to lead which might result from significant expansion of lead production for use in EV batteries.

5. Epidemiological studies to investigate the health effects of inorganic lead exposure, with special reference to those effects of a neurological nature (related to project 4 above).

6. Improved means for therapeutic removal of lead from animals, especially with respect to removal of lead from young animals: Treatment with ethylenediaminepentaacetic acid (EDTA) has proven beneficial for prevention of encephalopathy and related disorders in children due to heavy exposure to lead, but unwanted side effects are a complicating problem.

B. Ni/Fe AND Ni/Zn BATTERY SYSTEMS (Ni, Co, Cd)

1. Reevaluation of the OSHA standard for exposure to nickel compounds: Data indicate (a) a 14% lung tumor incidence in rats exposed to nickel subsulfide (Ni_3S_2) at the OSHA standard concentration (1 mg/m^3) compared to 1% in controls, and (b) lung alterations observed in rats exposed to nickel oxide (NiO) and nickel chloride ($NiCl_2$) at levels ten-fold below the OSHA standard.

2. Identification of nickel compounds to be involved in newly developing nickel recycling processes and scrutiny for their potential effects on the lung, particularly with respect to respiratory system carcinogenicity.

3. Accurate characterization of workplace exposures to nickel, cobalt, and cadmium with particular reference to battery manufacture: Levels of exposure, chemical composition, and size distribution of particles should be determined in the workplace.

4. Investigation of potential synergistic effects of nickel plus cobalt or nickel plus cadmium on the respiratory system: Manufacture of Ni/Fe and Ni/Zn EV batteries includes steps where occupational exposure to vapors containing these metal combinations could occur.

5. Clarification of the relationship between nickel sensitization and proxymal asthma response: This project would require (a) further laboratory study to understand the underlying pathophysiology, (b) clinical investigation to evaluate the medical significance of the response, and (c) epidemiological study to evaluate the potential population impact.

6. Investigation of a possible fetotoxic response to nickel: Increased participation of women in the workplace implies increased exposure of susceptible populations, especially for women during early pregnancy. Further basic biological research efforts will be required to determine the applicability of observed experimental animal responses to a clearly defined human situation.

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