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Lawrence Livermore National Laboratory

RCRA Part B Health Risk Assessment Phase II

Hazardous Waste Management Units

February 1990

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LLNL RCRA PART B
HEALTH RISK ASSESSMENT

Phase II - Hazardous Waste Management Units

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RADIAN CORPORATION

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LIST OF ACRONYMS

ABAG - Association of Bay Area Governments

ACGIH - American Conference of Governmental Industrial Hygienists

AIRDOS - Computer model that estimates concentration in air, rates of deposition on ground surfaces, ground surface concentrations, intake rates by man via food ingestion and air inhalation, and radiation dose received by man.

AMAD - Activity median aerodynamic diameter

AOD - Argon - oxygen decarburization

BAAQMD - Bay Area Air Quality Management District

BCF - Bioconcentration factor

CAG - Carcinogen assessment group

cal/sec - Calories per second

CAPCOA - California Air Pollution Control Officers Association

CARB - California Air Resources Board

CEQA - California Environmental Quality Act

cm - Centimeter

cm/sec - Centimeter per second

CNS - Central nervous system

CPF - Cancer potency factor

DARTAB - A computer model that takes exposure estimations from AIRDOS and estimates health risks or genetic damage.

DHS - California Department of Health Services

DOE - U. S. Department of Energy

EDTA - Ethylenediamine tetraacetic acid

EPA - U. S. Environmental Protection Agency

GI - Gastrointestinal factor (absorption factor)

GLC - Ground-level concentration

g/sec - Gram per second

HI - Hazard index

LIST OF ACRONYMS (Continued)

HRA - Health risk assessment
ICRP - International Commission on Radiological Protection and Measurement
IRIS - Integrated risk information system
ISCLT - Industrial source complex long-term
kg/day - Kilograms per day
kg/m² - Kilograms per square meter
km - Kilometer
Kow - Octanol-water partition coefficient
kW - Kilowatt
lb - Pound
lb/day - Pounds per day
lb/hr - Pounds per hour
lb/yr - Pounds per year
ℓ/day - Liters per day
LD_{50} - Lethal dose 50
LET - Linear energy transfer
LLNL - Lawrence Livermore National Laboratory
m - Meter
m^3 - Cubic meters
MEI - Maximally-exposed individual
mg/day - Milligrams per day
mg/kg - Milligrams per kilogram
mg/kg/day - Milligrams per kilogram per day
mg/m^3 - Milligrams per cubic meter
MMBtu/hr - Million British thermal units per hour
mrad/year - Millirads per year
MSDS - Material safety data sheet
m/sec - Meters per second

LIST OF ACRONYMS (Continued)

NCDC - National climatic data center
ng/kJ - Nanograms per kilojoule
ng/Nm^3 - Nanograms per normal cubic meter
NRC - U. S. Nuclear Regulatory Commission
ORNL - Oak Ridge National Laboratory
PAH - Polycyclic aromatic hydrocarbons
PCB - Polychlorinated biphenyl
pCi/year - Picocuries per year
ppm - Parts per million
RADRISK - Computer program that gathers data on the nuclides from a larger database.
RCRA - Resource Conservation and Recovery Act
RfD - Reference dose
R-RAM® - Radian Risk Assessment Model
RTECs - Registry of toxic effects
RUF - Root uptake factor
TAC - Toxic air contaminant
TEF - Toxic equivalency factor
TLV® - Threshold limit value (as established by the American Conference of Governmental Industrial Hygienists)
TPY - Tons per year
μg - Microgram
μg/m^3 - Micrograms per cubic meter
μg/m^3/g/sec - Micrograms per cubic meter per gram per second
μm - Micrometer
U.S. EPA - see EPA
U.S. NRC - see NRC
VOC - Volatile organic compound

1.0 EXECUTIVE SUMMARY

The Lawrence Livermore National Laboratory (LLNL) operates several hazardous waste storage and treatment units for managing the wastes generated by research programs. The storage and treatment units are presently operated under interim status in accordance with federal (U.S. Environmental Protection Agency) and state (California Department of Health Services) requirements.

As required by the California Hazardous Waste Control Act and the Resource Conservation and Recovery Act (RCRA), LLNL has applied for a Part B permit to continue operating their storage and waste treatment facilities. As part of this permitting process, LLNL is required to conduct a health risk assessment to examine the potential health impacts to the surrounding community from continued storage and treatment of hazardous and mixed radioactive wastes in the future. This document presents the results of the second phase of the risk assessment. An accident analysis for the maximum credible chemical accident is also included in Section 12.0. The maximum credible radiological accident is evaluated in a separate report submitted by LLNL (Salazar, 1990).

The Phase II risk assessment was prepared in accordance with procedures set forth by the California Air Pollution Control Officers Association (CAPCOA) "Air Toxics Assessment Manual," and specific requirements of the California Department of Health Services (DHS). By following these procedures, this risk assessment presents a health-conservative analysis of a hypothetical Maximally Exposed Individual (MEI) using many worst-case assumptions that will not apply to an actual individual. As such, the risk estimates presented should be regarded as a worst-case estimate of any actual risk that may be present.

Many of the methods and assumptions employed in this risk assessment are intentionally health conservative. For example, certain methods used to quantify emissions from the various treatment units result in worst-case estimates that are likely to be significantly higher than what actually will occur. It is important to recognize, however, that

other technically valid and justifiable methods could be used to estimate emissions, exposures, or risks, many of which might result in lower values. The fact that health-conservative methods have been intentionally employed in this Phase II health risk assessment does not set precedent for methods that should be used in subsequent analyses prepared for or by LLNL.

Project Background

LLNL is operated by the University of California for the U.S. Department of Energy. Research programs conducted at LLNL (including biomedical, energy and resources, defense systems, laser isotope separation, and magnetic fusion energy) generate nonradioactive, radioactive, hazardous, and mixed (containing both radioactive and hazardous substances) wastes, which must be handled and disposed of according to applicable state and federal regulations and DOE orders. To manage these wastes, LLNL operates several hazardous waste storage and treatment units which include wastewater treatment, solidification, silver recovery, drum crushing, tank and container storage, and size reduction.

Because the wastes generated at LLNL consist of a variety of compounds and because several storage and treatment units are used to manage these wastes, it has been necessary to conduct this health risk assessment in three phases. The first phase, Phase I, examined the potential for adverse health effects from continued operation of the existing incinerator for the next 70 years. Although LLNL elected to discontinue operating the existing incinerator and has withdrawn the permit application, the Phase I risk assessment was completed in December and submitted to the agencies. The results of the Phase I risk assessment indicated a worst-case risk of nine in ten million (9×10^{-7}) and a plausible risk of approximately six in ten million (6×10^{-7}). The second phase of the risk assessment examines the potential for adverse health effects from all storage and treatment units included in the RCRA Part B permit application. The results of Phase II are presented in this document. Phase III of the risk assessment will evaluate the potential for adverse health effects from hazardous waste accumulation areas, generator bulking areas, and

retention tank transfer points in addition to the hazardous waste management units included in the RCRA Part B application. Phase III is scheduled for completion in 1991, and its results will be documented in a separate report.

Methodology

Prior to conducting Phases I and II of the health risk assessment, a protocol outlining the methods and evaluation procedures was prepared and submitted to the EPA (Region IX) and the California Department of Health Services (DHS) for review and comment. Preliminary comments were received and incorporated into the risk assessment as required. In general, the methods that were employed are consistent with EPA guidelines and the guidelines published in the California Air Pollution Control Officers Association "Air Toxics Assessment Manual."

The type of wastes generated at LLNL and the compounds present in those wastes were identified through a review of waste treatment records and the LLNL waste tracking database. The results of this evaluation were used to identify the types and quantities of compounds treated or stored in the LLNL storage and treatment units.

Once the wastes were characterized, potential emissions of toxic substances were determined. Waste types and treatment technologies at LLNL are such that not all units will result in significant emissions. The size reduction unit and the metal shredder were judged to have no significant potential for emissions or offsite impacts. At the size reduction facility, equipment is either dismantled or cut into smaller pieces with power tools. Particles emitted from the cutting operations will be large and are not likely to be picked up by the building ventilation system. The shredder, which is used to shred solid waste and containers into smaller pieces using a tearing mechanism, will not generate any significant particulate emissions since grinding or cutting does not occur. Given the operations and existing controls, emissions from these sources were considered negligible. Those treatment/storage operations with the potential for offsite impacts, and hence included in this risk assessment, were wastewater treatment, bulk solidification, waste

bulking, and drum crushing. The remaining treatment or storage limits were examined and consequently dismissed.

Based on the waste characterization and the emissions estimates, a hazard identification process was conducted to focus the risk assessment on those pollutants that could pose an off-site health risk and exclude those with no significant potential for adverse health effects. Relative toxicity and emission rates were the determining factors for excluding pollutants from further evaluation.

Once the hazards were identified, their concentration in the air and deposition rates on the ground could be estimated. Weather and wind patterns play an important role in how emitted substances move through the environment. Three years of hourly weather data obtained from a monitoring station at LLNL, supplemented by upper air data from the Oakland Airport, were used with two EPA- and California Air Resources Board (CARB)-approved computer models to simulate the movement of nonradioactive emissions and to estimate air concentrations and deposition rates at off-site locations. To simulate the movement of radionuclides, the EPA AIRDOS model was used with the LLNL weather data.

To estimate human exposure to nonradioactive emissions from LLNL, the risk assessment evaluated a hypothetical maximally-exposed individual (MEI) and approximately 200 additional receptors (i.e., locations) in the Livermore area. The MEI is assumed to be born, reside, and work for a 70-year lifetime at the point where the highest concentrations of emissions occur from the hazardous waste storage and treatment units. This approach does not account for differences resulting from periods spent at work, away from the residence, or time spent traveling out of the area. The MEI will consequently have an exposure greater than all individuals, and hence, is a health-conservative representation of public exposure.

In quantifying the amount of each nonradioactive compound that an individual could be exposed to, three primary exposure routes were considered: inhalation; ingestion (including soil, vegetation, wine and water); and dermal contact with soil. The methods for

quantifying exposure to nonradioactive emissions are documented in this report. Similar pathways of exposure were also considered for the radionuclides. The EPA AIRDOS-DARTAB model was used to carry out the calculations of radionuclide exposure.

Finally, the lifetime risk of developing cancer and the potential for acute and chronic noncarcinogenic effects were evaluated. A plausible case and a worst case are presented partially to account for the uncertainty in the health risk assessment process. The worst-case analysis includes two separate scenarios -- one based on DHS-prescribed cancer potency factors and the other based on EPA cancer potencies; in many cases, both potencies are equivalent. Several compounds which were identified in small quantities in the waste stream could not be evaluated in this risk assessment. The available toxicology data for these compounds were not sufficient to support a calculation of cancer risk. Therefore, their contribution to the estimated cancer risk from the LLNL storage and treatment operations could not be determined.

Noncarcinogenic effects were evaluated by comparing exposure rates to pollutant-specific, health-based criteria.

Results

The worst-case "theoretical" lifetime risk of developing cancer for the MEI was estimated to be 9.4 in one hundred million (9.4×10^{-8}). This means an individual residing for 70 years at the point of highest concentration of emissions, approximately 600 meters north of East Avenue along Greenville Road, has a 9 in one hundred million chance of developing some form of cancer. This risk estimate is based on cancer potency estimates recommended by DHS. When EPA-recommended potencies are used, the worst-case risk was estimated to be 8.7 in one hundred million (8.7×10^{-8}). The risk to the maximally-exposed individual in the plausible case was estimated to be 2 in one hundred million (2.0×10^{-8}). The risks for all scenarios are summarized in Table 1-1. The risk estimates shown in Table 1-1 represent a health-conservative estimate of potential risk for the compounds evaluated; the actual risk is likely to be lower and could be zero.

TABLE 1-1. SUMMARY OF CANCER RISK

Scenario	Nonradioactive	Radionuclides[d]	Total Risk[e]
Worst-Case			
DHS Potencies[a]	8.3×10^{-8}	1.1×10^{-8}	9.4×10^{-8}
EPA Potencies[b]	7.6×10^{-8}	1.1×10^{-8}	8.7×10^{-8}
Plausible Case[c]	8.9×10^{-9}	1.1×10^{-8}	2.0×10^{-8}

[a] Cancer risk values based on DHS-approved potency values and worst-case exposure assumptions.

[b] Cancer risk values based on EPA cancer potency values and worst-case exposure assumptions.

[c] Risk developed from plausible exposure assumptions and EPA cancer potency factors.

[d] Exposure and risk from radionuclide emissions were determined for only one scenario. A single risk estimate is, therefore, presented for all cases.

[e] Values represent the maximum exposed individual's lifetime risk of developing some form of cancer due to hazardous waste storage and treatment facility emissions.

The CAPCOA manual requires that cancer burden, the theoretical estimate of the increased number of cancer cases resulting from exposure to emissions, must be determined for all populations exposed to a risk of one in 10 million (1×10^{-7}) or greater. The maximum risk for the MEI was estimated to be 9.4 in one hundred million (9.4×10^{-8}). Therefore, a calculation of cancer burden is not required for this assessment.

The chronic noncarcinogenic effects assessment indicated that daily exposure estimates for each compound emitted from the hazardous waste treatment and storage units were below levels believed to be without any adverse effects, even for sensitive individuals such as the elderly or asthmatics. Similarly, the acute effects analysis indicated maximum one-hour concentrations of emissions below levels that would cause adverse effects.

In addition to potential adverse effects from routine operations of the hazardous waste management units, an accident analysis was performed for a hypothetical worst-case chemical accident. Science Applications International Corporation (SAIC) conducted an evaluation of the LLNL hazardous waste treatment and storage unit operations to identify potential chemical accidents. Using accident potential, the estimated emission rate, downwind concentration estimates, and an indication of toxicity as the basis for ranking accident scenarios, a formaldehyde spill was determined to represent the maximum credible accident to be evaluated in this health risk assessment (SAIC, 1990). The EPA- and ARB-approved INPUFF model was used to estimate the downwind impacts of the hypothetical accident at the nearest public road and residence. Formaldehyde exposure was examined assuming continuous exposure and for a reduced exposure period representing an individual passing through the plume. Based on the modeling results and the exposure scenarios, the potential for acute health effects as well as cancer risk was examined. Acute effects were evaluated by comparing the estimated concentration in air to health-based criteria. Cancer risk was estimated using the appropriate DHS and EPA cancer potency factor for formaldehyde.

The worst-case risk of developing cancer from continuous exposure to the formaldehyde release (approximately 1 hour) was found to be 3.6 in ten million (3.6×10^{-7}). For an individual passing through the plume at a normal pace (2 miles per hour), the risk

was estimated to be 2.1 in one billion (2.1×10^{-9}). At the nearest residence, a one-hour exposure would result in a cancer risk of 1.1 in ten million (1.1×10^{-7}). The maximum modeled concentration of formaldehyde at the nearest public road (14 ppm) was determined to be above the Emergency Response Planning Guideline-2 (ERPG-2), but appears to be below levels associated with significant and irreversible noncancer effects. The nearest residence, or individual passing through the plume in the road, would experience exposures below the ERPG-2 and levels associated with irreversible effects.

2.0 INTRODUCTION

2.1 Project Background

The Lawrence Livermore National Laboratory (LLNL) is a multiprogram facility operated by the University of California for the U.S. Department of Energy (DOE). Defense and nondefense-related research programs are conducted at LLNL, including defense systems, laser isotope separation, magnetic fusion energy, biomedical and environmental research, and energy and resources. The 821-acre LLNL site is in Alameda County adjacent to the eastern boundary of the City of Livermore.

A number of the research programs conducted at LLNL generate nonradioactive, radioactive, mixed (containing both hazardous constituents and radionuclides), and hazardous wastes that must be handled and disposed of according to current applicable regulations. To manage the wastes generated by these programs, LLNL operates several hazardous waste storage and treatment units at the facility. These units include liquid waste treatment, hazardous and mixed waste storage, size reduction, silver recovery, shredding, bulking, and solidification. LLNL operates these facilities under interim status. Pursuant to requirements of the Resource Conservation and Recovery Act (RCRA) and the California Hazardous Waste Control Act, a Part B permit is required to continue storage and treatment operations in the future. In conjunction with the Part B permit application, the California Department of Health Services (DHS) and the U.S Environmental Protection Agency (EPA) have requested LLNL to prepare a comprehensive health risk assessment to examine potential impacts on the surrounding community from continued operation of the storage and treatment units.

Due to the complexity of the LLNL hazardous waste storage and treatment operations and the variety of waste streams that are generated, the RCRA Part B health risk assessment is being prepared in three phases. The first phase, Phase I, was an evaluation of potential exposure and risk from the hazardous waste incinerator and associated waste feed systems. This risk assessment has been completed and submitted to

EPA and DHS in December 1989 even though LLNL does not intend to continue operating the incinerator and the application has been withdrawn. This document presents the methods and results of Phase II, which involves a comprehensive evaluation of all permitted waste management units included in the Part B application. Phase III, to be prepared in 1991, will involve an evaluation of all remaining waste management activities at LLNL in addition to the permitted units in the RCRA Part B application.

Waste management units evaluated in Phase II of this risk assessment include the following:

- Waste water treatment (Building 513);
- Size reduction (Building 419);
- Solidification (Buildings 419, 513);
- Bulking (Building 614);
- Shredding (Building 513);
- Silver recovery (Building 514);
- Drum crushing (Building 612); and
- Waste storage (Areas 514, 612; Building 693).

The waste water treatment tank farm treats waste water containing both hazardous constituents and radionuclides. Wastes received at this unit undergo a series of treatment methods including neutralization, flocculation, oxidation, reduction, precipitation, separation, and filtration. The size reduction facility is used to reduce the size of contaminated equipment for eventual disposal. Solidification involves solidifying liquid and semi-solid wastes for future disposal. Bulking, which is not a specific permitted activity or unit, involves combining compatible wastes from small containers into single large containers for disposal or future treatment. The shredding unit shreds solid wastes and/or containers for future treatment or disposal. The drum crushing facility is used to crush empty drums and containers. The silver recovery unit at Building 514 recovers silver from spent photographic solutions. Waste will be stored in a variety of containers and tanks in Areas 514 and 612, and in Building 693 (future). The LLNL Part B permit application presents a more detailed discussion of the various treatment units currently in operation.

2.2 Risk Assessment Methodology

A risk assessment protocol for the LLNL hazardous waste management facilities has been prepared and submitted to DHS and EPA for review (Radian, 1989). Among the reviewing agencies were the Toxic Substance Control Division and Epidemiological Studies Sections of DHS and EPA Region IX. The methods outlined in the protocol are the result of several discussions between LLNL, Radian Corporation, DHS, and EPA, and provide the basis for this risk assessment.

In general, the Phase II risk assessment followed the requirements of the California Air Pollution Control Officers Association (CAPCOA) manual (CAPCOA, 1987). In preparing a risk assessment that reflects LLNL operations, some deviation from the manual was necessary. Where such deviations occur, justification for the alternate approach is provided.

Many of the methods and assumptions employed in this risk assessment are intentionally health conservative. For example, certain methods used to quantify emissions from the various treatment units result in worst case estimates that are likely to be significantly higher than what actually will occur. It is important to recognize however, that other technically valid and justifiable methods could be used to estimate emissions, exposures, or risks, many of which might result in lower values. The fact that health conservative methods have been intentionally employed in this Phase II health risk assessment does not set precedent for methods that should be used in subsequent analyses prepared for or by LLNL.

Uncertainty in the potential risk was addressed by presenting two base cases: a plausible risk estimate, and a worst-case risk estimate. Differences in the two base cases include exposure parameters and toxicological factors. Within the worst-case assessment, two scenarios were evaluated: one utilizing EPA potency factors, and a second utilizing DHS potency factors listed in the CAPCOA manual. The purpose for including two slightly

different risk scenarios was to examine the effects on the worst-case cancer risk from using different cancer potency estimates.

Emissions from the various hazardous waste management processes were estimated from waste characterization data developed from LLNL treatment logs and the waste tracking database. Engineering principals and mass balance techniques were used in quantifying the release rate of each substance from the various treatment sources.

The EPA-approved SHORTZ and Industrial Source Complex Short-Term (ISCST) dispersion models were used to estimate the airborne concentration of emissions at a series of downwind receptors. The dispersion models selected for the risk assessment are in accordance with guidelines set forth by the California Air Resources Board (CARB) and incorporated in the CAPCOA manual.

The deposition rate of particulate-phase pollutants was estimated by applying a default deposition velocity to the estimated airborne concentration. Concentration estimates and deposition rates were obtained for approximately 200 locations surrounding the LLNL facility. The number of locations is dictated by the need to adequately identify the point of maximum off-site impact and the exposure at populated areas. The EPA AIRDOS model was used to simulate transport and dispersion of radionuclide emissions from the hazardous waste storage and treatment units.

Human exposure was evaluated for a hypothetical maximally-exposed individual (MEI) and a series of population receptors surrounding the LLNL site. The MEI was assumed to reside continuously for 70 years at the point of maximum off-site exposure to emissions. All potential pathways (including inhalation, ingestion, and dermal contact) appropriate for the land use and geology in the LLNL area were considered in estimating human exposure. The Radian Risk Assessment Model (R-RAM®) was used for calculating exposure from nonradioactive emissions, and AIRDOS-DARTAB was used for estimating exposure to radionuclides. R-RAM® is a proprietary computer model developed by Radian for calculating exposure and risk of nonradioactive substances. It has been used by Radian in several risk assessments reviewed and approved by DHS (Chevron Chemical Company

incinerator, Shell Oil Company incinerator, the Casmalia Resources hazardous waste disposal facility, and the Milliken waste-to-energy facility). AIRDOS-DARTAB is a computer model developed by the Oak Ridge National Laboratory for EPA.

Cancer risk for nonradioactive emissions was estimated for the MEI and each of the population receptors using potency estimates from the CAPCOA manual (CAPCOA, 1987) for the worst case (where available) and from EPA or other research organizations for the plausible case. Since DHS has not developed potencies for all carcinogens considered in the risk assessment, EPA potencies were also used.

The potential for noncarcinogenic effects was evaluated for both short-term exposures and chronic exposures to nonradioactive emissions from the hazardous waste units. Chronic effects were assessed by comparing estimated daily exposure rates to reference dose (RfDs) values developed by the EPA and informal RfDs developed from review of the toxicology literature. Acute noncarcinogenic effects were evaluated in a two-step process by comparing worst case, one-hour air concentrations from normal operations to toxicological standards.

In addition to routine emissions from the LLNL hazardous waste management facilities, two accident scenarios were evaluated: one involving the release of nonradioactive substances, and one involving the release of radionuclides. Both accidents are regarded as hypothetical events representing a maximum credible accident. The nonradioactive accident analysis is summarized in Section 12.0. Science Applications International Corporation (SAIC) conducted an evaluation of the LLNL hazardous waste treatment and storage unit operations to identify potential chemical accidents. Using accident potential, the estimated emission rate, downwind concentration estimates, and an indication of toxicity as the basis for ranking accident scenarios, a formaldehyde spill was determined to represent the maximum credible accident to be evaluated in this health risk assessment (SAIC, 1990). A source term was developed for the accident and a dispersion model was used to assess downwind impacts. The worst-case risk of cancer and the potential for acute noncancer effects were estimated or evaluated. The accident analysis

for the radioactive release is documented in a separate report prepared by LLNL (Salazar, 1990).

2.3 Document Organization

The balance of this document is organized in the following format:

- Section 3.0 presents waste characterization data;
- Section 4.0 outlines the methods used for estimating emissions;
- Section 5.0 presents the results of hazard identification;
- Section 6.0 discusses the dispersion modeling;
- Section 7.0 is environmental fate and human exposure;
- Section 8.0 is a characterization of the exposed population;
- Section 9.0 presents dose-response data;
- Section 10.0 presents the radioactive risk assessment;
- Section 11.0 presents risk characterization;
- Section 12.0 presents the chemical accident analysis; and
- Section 13.0 provides references.

Technical support data, dispersion model results, and AIRDOS-DARTAB and R-RAM® output can be found in the appendices.

3.0 WASTE CHARACTERIZATION

There are seven treatment units at LLNL that have been examined in this health risk assessment. In order to assess the possible health risks from each, the wastes treated must be characterized. The eight treatment areas include:

- Wastewater Treatment Tank Farm;
- Waste Solidification in Buildings 419 and 514;
- Bulking of Wastes;
- Silver Recovery from spent Photographic Chemicals;
- Empty Container Crushing;
- Size Reduction of Contaminated Materials;
- Shredding of Contaminated Materials; and
- Waste Storage.

Each of these areas is more thoroughly described in the following subsections. The maximum quantity of waste which could be treated in each of these treatment units is listed in Table 3-1.

The waste characterizations for each treatment unit or process are based on treatment records from Lawrence Livermore National Laboratory (LLNL). The data represent wastes treated from June 1988 to June 1989. In general, the available data include the amount and chemical composition of each individual batch of waste treated in this time frame. Wastes treated at LLNL during June 1988 to June 1989 are considered representative of typical wastes to be treated in the future.

The waste characterizations listed here represent the annual amount of each chemical that would pass through each treatment unit. To represent the large number of individual batches and chemicals treated, a health-conservative (i.e., protective of health) approach was used to calculate the chemical quantities in each waste stream. The amount of a chemical treated at a specific unit was assumed to be the product of the total quantity of waste and representative concentrations of each chemical. Depending upon chemical,

TABLE 3-1. MAXIMUM ANNUAL AMOUNT[a] OF TREATABLE WASTE AT EACH TREATMENT UNIT

Treatment Unit	Maximum Annual Amount[a]
Bulking	7,100 gallons
Container Crushing	3,700 cubic feet[b]
Shredding	2,100 cubic yards
Silver Recovery	5,500 gallons
Size Reduction	19,800 pounds
Solidification	170,000 pounds
Tank Farms	206,000 gallons

[a] Based on projections of the maximum quantity of waste that might be generated in any given year.

[b] This represents the volume of 500 empty 55-gallon drums.

this representative concentration is either a weighted average or the maximum single concentration of the chemical recorded in any waste batch.

When the range of chemical concentrations was small within the given waste batch, the maximum concentration was used to calculate the amount of each chemical. When the range varied significantly, then one of two values was used. If, for a specific chemical, there was a wide variation in chemical concentration, but the upper concentration range was represented by only a small fraction of the total quantity, then the representative concentration was assumed to be the maximum concentration representing the majority of the waste. In a few cases where a wide range of chemical concentrations existed throughout the waste stream, a weighted average concentration was used to calculate the annual chemical quantity.

3.1 Wastewater Treatment

The wastes treated in this unit are aqueous with trace amounts of organic, inorganic, and radioactive contaminants. Speciation of the wastes into individual chemicals followed the procedures described above. Any entries which were commercial products or chemical mixtures were further speciated using information from the products Material Safety Data Sheet (MSDS). Table 3-2 lists the quantity and types of chemicals potentially treated at the wastewater treatment tank farm.

A portion of the waste that is treated at the wastewater treatment tank farm is classified as mixed waste (i.e., containing both hazardous and radioactive substances). The chemical portion of these wastes has been included in Table 3-2. The activity of radionuclides present in the wastes is shown in Table 3-3.

3.2 Solidification

The wastes treated by solidification in building 419 and 513 are also primarily aqueous wastes with trace amounts of organic and inorganic contaminants. The filter cake

TABLE 3-2. CHARACTERIZATION OF TREATABLE WASTE AT THE WASTEWATER TREATMENT TANK FARM

Chemical	Maximum Annual Amount[a]	
	Grams	Pounds
Acetone	2300	5.0
Aluminum	0.78	0.0017
Ammonia	210000	460
Benzene	20.	0.43
Benz(a)anthracene	4.9	0.10
Benzo(a)pyrene	1.1	0.0024
Beryllium	1700	3.8
Borneol	440000	970
Boric Acid	2900	6.3
Butoxyethanol, 2-	55.	0.12
Cadmium	0.17	0.00037
Cesium	750	1.65
Chloroform	0.77	0.0017
Chromium	1000000	2200
Cobalt	1.7	0.0036
Copper	480000	1000
Copper Sulfate	11000	25.
Cyanide	3500	7.8
Dibromoethane, 1,2-	0.31	0.00068
Dichloroethane, 1,2-	37.	0.083
EDTA	17.	0.038
Erbium	220000	480
Ethyl Benzene	60.	0.13
Ethylene Glycol	30.	0.067
Hydrogen Chloride	9200	20
Hydrogen Fluoride	8500	19
Hydrogen Peroxide	4000	8.7
Iron	280000	610
Lead	130000	280
Mandellic Acid	0.20	0.00044
Manganese	0.00034	0.00000075
Mercury	110	0.23
Methyl Ethyl Ketone	140000	300
Methylene Chloride	130	0.28

(Continued)

TABLE 3-2. (Continued)

Chemical	Maximum Annual Amount[a] Grams	Pounds
Napthalene	92.	0.20
Nickel	230000	510
Nitric Acid	48000	100
Osmium	0.0038	0.000008
Oxalic Acid	1100	2.3
PCB	25.	0.056
Perchloroethane, 1,1,2,2-	380	0.83
Perchloroethene	52000	110
Phenanthrene	30.	0.067
Phosphoric Acid	2000	4.4
Potassium Hydroxide	29000	65.
Pyrene	61.	0.13
Silver	50.	0.11
Sodium Hydroxide	380000	840
Sulfuric Acid	3700	8.1
Tartaric Acid	2000	4.4
Toluene	98.	0.21
Trichloroethane, 1,1,1-	100000	230
Trichloroethane, 1,1,2-	2.7	0.0059
Trichloroethene	280	0.63
Trichlorotrifluoroethane	36000	80.
Terpineol, -α	4000000	8700
Xylene	290	0.64
Zinc	59000	130

[a] Based on projections of the maximum quantity of waste that might be treated in any given year.

TABLE 3-3. MAXIMUM RADIONUCLIDE LEVELS IN THE TREATABLE WASTEWATER

Nuclide	Symbol	Annual Activity[a] Curies
Barium-133	^{133}Ba	0.000003
Bismuth-214	^{214}Bi	0.000001
Carbon-14	^{14}C	0.001
Cerium-141	^{141}Ce	0.000001
Iodine-131	^{131}I	0.000003
Lead-214	^{214}Pb	0.000001
Niobium-95	^{95}Nb	0.000001
Plutonium-239	^{239}Pu	0.003
Potassium-40	^{40}K	0.000001
Rhenium-106	^{106}Rh	0.000001
Ruthenium-103	^{103}Ru	0.000005
Selenium-75	^{75}Se	0.001
Sulfur-35	^{35}S	0.001
Tritium	^{3}H	0.1
Uranium-238	^{238}U	0.03
Zirconium-95	^{95}Zr	0.000001
Mixed Fission Products (MFP)		0.003

[a] Based on projections of the maximum activity treated in any given year.

from the wastewater treatment vacuum filter could also be solidified at building 513. Speciation of wastes into individual chemicals was described above. Tables 3-4 and 3-5 characterize the wastes solidified in buildings 419 and 513 respectively.

As with the wastewater treatment tank farm, a portion of the waste treated by solidification is mixed waste. Radionuclide levels in the solidification wastes are presented in Tables 3-6 and 3-7.

3.3 Bulking

Bulking of small volume wastes occurs adjacent to building 614. Wastes of a similar nature which are stored in small containers are combined into larger containers to increase ease of handling. Only the type and quantity of waste were identified. Where accurate concentrations were not available, the waste quantity was assumed to be equally distributed among all identified chemicals. The waste characterization for bulking is listed in Table 3-8.

Water containing carcinogens are listed among the chemicals in Table 3-8. Based on a detailed review of the waste logs, the carcinogens in these entries are a dilute mixture of Texas Red, Hoesht, and Phycoerytchrin. No other carcinogens were identified in the two waste entries.

There are no radionuclides present in the bulking waste streams at Building 614.

3.4 Silver Recovery

Spent photographic solutions generated at LLNL contain recoverable quantities of silver as well as other trace level contaminants. Data on the specific spent photographic solutions treated in this unit were used to develop a waste characterization. Both the amounts and chemical concentrations were available for the solutions treated. Where commercial products were identified, a speciation of individual chemicals was made using

TABLE 3-4. CHARACTERIZATION OF TREATABLE WASTE SOLIDIFIED AT BUILDING 419

Chemical	Maximum Annual Amount[a] Grams	Pounds
Acetone	2.3	0.0052
Aluminum	1600	3.5
Benzene	0.12	0.00027
Benz(a)anthracine	0.053	0.00012
Benz(a)pyrene	0.012	0.000026
Beryllium	210	0.47
Borneol	4800	11.
Boric Acid	360	0.79
Cadmium	0.10	0.00023
Cesium	1.5	0.0033
Chloroform	0.0034	0.000007
Chromium	1200	2.6
Cobalt	0.2	0.00044
Copper	2700	5.8
Copper Sulfate	6.2	0.014
Cyanide	0.25	0.00056
Dibromoethane, 1,2-	0.0034	0.000007
Dichloroethane, 1,2-	0.41	0.0009
Ethylene Diamine Tetra Acetic Acid	2.2	0.0048
Erbium	27000	58.
Ethidium Bromide	0.083	0.00018
Ethyl Alcohol	5000	11.
Ethyl Benzene	0.65	0.0014
Ethylene Glycol	5000	11.
Flocculent Deflate	27000	59.
Hydrogen Chloride	1500	3.4
Hydrogen Fluoride	1100	2.3
Iron	35000	77.
Lead	96.	0.21
Mandellic Acid	130	0.28
Mercury	13.	0.029
Methyl Ethyl Ketone	17000	38.
Methylene Chloride	0.81	0.0018
Napthalene	1.2	0.0027
Nickel	650	1.4
Nitric Acid	6000	13

(Continued)

TABLE 3-4. (Continued)

Chemical	Maximum Annual Amount[a] Grams	Pounds
Osmium	0.00047	0.000001
Oxalic Acid	12.	0.026
Perchloroethene	6400	14.
Phenanthrene	0.33	0.00072
Phosphoric Acid	250	0.55
Potassium Hydroxide	191.	0.42
Pyrene	0.67	0.0015
Silver	0.14	0.00031
Sodium Hydroxide	47000	100
Sulfuric Acid	95000	210
Tartaric Acid	250	0.55
Toluene	1.1	0.0023
Trichloroethane, 1,1,1-	13000	29.
Trichloroethane, 1,1,2-	0.029	0.000063
Trichloroethene	33.	0.072
Trichlorotrifluoroethane	4300	9.6
Turpineol, -α	43000	95.
Xylenes	3.1	0.0069
Zinc	1500	3.2

[a] Based on projections of the maximum amount of waste that might be treated in any given year.

TABLE 3-5. CHARACTERIZATION OF TREATABLE WASTE SOLIDIFIED AT BUILDING 513

Chemical	Maximum Annual Amount[a]	
	Grams	Pounds
Acetone	2.3	0.0052
Aluminum	1600	3.5
Benzene	4300	9.5
Benz(a)anthracine	1900	4.2
Benz(a)pyrene	430	0.95
Beryllium	400	0.89
Borneol	10000	23
Boric Acid	360	0.79
Cadmium	0.10	0.00023
Cesium	1.5	0.0033
Chloroform	120	0.27
Chromium	1800	3.9
Cobalt	0.2	0.00044
Copper	2800	6.3
Copper Sulfate	6.2	0.014
Cyanide	0.25	0.00056
Dibromoethane, 1,2-	120	0.27
Dichloroethane, 1,2-	15000	33.
Ethylene Diamine Tetra Acetic Acid	2.2	0.0048
Erbium	27000	58.
Ethidium Bromide	0.083	0.00018
Ethyl Alcohol	5000	11.
Ethyl Benzene	23000	51.
Ethylene Glycol	5000	11.
Hydrogen Chloride	1500	3.4
Hydrogen Fluoride	1100	2.3
Iron	35000	77.
Lead	96.	0.21
Mandellic Acid	130	0.28
Mercury	13.	0.029
Methyl Ethyl Ketone	17000	38.
Methylene Chloride	30000	65.
Napthalene	43000	96.
Nickel	25000	56.
Nitric Acid	6000	13.

(Continued)

TABLE 3-5. (Continued)

Chemical	Maximum Annual Amount[a] Grams	Pounds
Osmium	0.00047	0.000001
Oxalic Acid	12.	0.026
Perchloroethene	5000000	11000
Phenanthrene	12000	26.
Phosphoric Acid	250	0.55
Potassium Hydroxide	190	0.42
Pyrene	24000	52.
Silver	0.14	0.00031
Sodium Hydroxide	47000	100
Sulfuric Acid	95000	210
Tartaric Acid	250	0.55
Toluene	38000	84.
Trichloroethane, 1,1,1-	10000000	23000
Trichloroethane, 1,1,2-	1100	2.3
Trichloroethene	34000	76.
Trichlorotrifluoroethane	3400000	7400
Turpineol, -α	94000	210
Xylenes	110000	250
Zinc	3800	8.2

[a] Based on projections of the maximum amount of waste that might be treated in any given year.

TABLE 3-6. RADIONUCLIDE ACTIVITY FROM TREATABLE WASTE SOLIDIFIED AT BUILDING 419

Isotope	Annual Activity[a] Curies
Tritium	1.4
Depleted Uranium	8.2×10^{-3}
Uranium-238	4.1×10^{-4}
Mixed Fission Products	1.0×10^{-1}
Thorium	9.0×10^{-6}

[a] Based on projections of the maximum activity treated at Building 419 in any given year.

TABLE 3-7. RADIONUCLIDE ACTIVITY FROM TREATABLE WASTE SOLIDIFIED AT BUILDING 513

Isotope	Annual Activity Curies
Tritium	1.5
Depleted Uranium	8.4×10^{-3}
Uranium-238	4.1×10^{-4}
Mixed Fission Products	1.0×10^{-1}
Thorium	9.0×10^{-6}

[a] Based on projections of the maximum activity treated at Building 513 in any given year.

TABLE 3-8. CHARACTERIZATION OF TREATABLE WASTES BULKED AT BUILDING 614

Chemical	Maximum Annual Amount[a] Grams	Pounds
Acetic Acid	28000	55.
Acetone	170000	320
Acetonitrile	1800	3.5
Benz(a)anthracene	0.97	0.0019
Benz(a)pyrene	0.22	0.00043
Benzene	17000	33.
Beryllium	170	0.38
Bisphenol A/Epichlorohydrin Resin	3700	8.2
Borneol	8800000	17000
Butanol, n-	77000	150
Butyl Glycidyl Ether, n-	660	1.3
Chloroform	38000	73.
Chromomycin A	760	1.7
Colloidol Silica	57000	130
Cupric Sulfate	410000	900
Cyclohexane	100000	190
Dichlorotrifluoroethane	34000	66.
Discetone Alcohol	7500	14.
Ethyl Alcohol	170000	330
Ethanol and Potassium Hydroxide Solution	50000	95.
Ethyl Benzene	12.	0.023
Dibromoethane, 1,2-	0.062	0.00012
Dichloroethane, 1,2-	7.4	0.014
Ethylene Glycol	160000	310
Heptane	100000	190
Isobutane	290000	550
Isopropyl Alcohol	15000	29.
Laser dye waste	770000	1500
Mercury	1300000	2800
Methyl Alcohol	440000	840
Methioine	22000	42.
Methyl Cyclohexane	100000	190
Methyl Ethyl Ketone	22000	42.
Methyl Isobutyl Ketone	62000	120
Methylene Chloride	230000	450

(Continued)

TABLE 3-8. (Continued)

Chemical	Maximum Annual Amount[a] Grams	Pounds
Naphthalene	6100	12.
PCB Oil	130000	250
Phenanthrene/anthracene	6.0	0.012
Pyrene	12.	0.024
Sodium Silicate Solution	38000	83.
Tetrachloroethylene	11000	21.
Tetraethylsilicate (Ethyl Silicate)	20000	39.
Thymidine	54000	100
Toluene	210000	410
Trichloroethane, 1,1,1-	59000	110
Trichloroethane, 1,1,2-	0.53	0.001
Trichloroethylene	87000	170
Trichlorotrifluoroethane, 1,1,2-	430000	830
Triethanolamine	21000	40.
Turpineol, -α	80000000	150000
Water (<0.1 % Carcinogen)	570	1.3
Water (<1.0 % Carcinogen)	2500	5.4
Xylene	42000	80.

[a] Based on projections of the maximum amount of waste that might be bulked in any given year.

the product MSDS. The total waste characterization for the silver recovery operation is presented in Table 3-9.

3.5 Container Crushing

Wastes which might be emitted from container crushing are a function of the contents of the container prior to being crushed. Since the drums are empty and usually dry, it is difficult to get precise data on residual chemicals which might remain in the empty containers.

Waste records were examined to determine the composition of chemicals previously stored in the containers. The amount of residual chemicals in the containers was assumed to equal 0.404 weight percent of the total capacity of containers crushed (EPA, 1987). The potential quantities of residual chemicals are listed in Table 3-10. These quantities are health conservative since they are based on the assumption that all containers contain the same quantity of residual waste.

3.6 Size Reduction

The possible chemicals involved with size reduction vary depending upon the equipment that is being dismantled or reduced in size. The majority of material reduced are pieces of lab equipment, ducting, or other materials which are decontaminated and appropriately disposed.

Therefore, the materials involved are primarily metallic and include steel, iron, chromium and aluminum items. Possible chemical contaminants include beryllium or mercury.

3.7 Shredding

Shredding operations are similar to the size reduction operations. The chemicals involved depend on the material that is being shredded. The shredder is primarily

TABLE 3-9. CHARACTERIZATION OF TREATABLE WASTES FOR SILVER RECOVERY

Chemical	Maximum Annual Amount[a] Grams	Pounds
Acetic Acid	290000	640
Alkali Metal Acetate	2600	5.8
Alkali Metal Sulfate	5300	12.
Aluminum Chloride	8300	18.
Aluminum Sulfate	750	1.7
Ammonium Hydroxide	22000	48.
Ammonium Sulfate	21000	46.
Ammonium Thiosulfate	3400000	7500
Ammonium [(ethylenedinitrilo)tetraacetol] ferrate	200000	430
Boric Acid	190	0.42
Chromium [b]	3.1	0.0068
Gluconic Acid	210000	460
Glutaraldehyde	1700	3.7
Glycerin	750	1.7
Hydroquinone	4100	9.1
Isopropyl Alcohol	150	0.33
Methylaminoethanol	750	1.7
Potassium Hydroxide	3300	7.3
Potassium Metabisulfite	1700	3.7
Pyrolidinone, 1-phenyl-3-	330	0.73
Pyrolidinone, N-methyl-2-	1700	3.7
Silver	1900	4.1
Sodium Acetate	5300	12.
Sodium Bisulfate	380000	850
Sodium Metaborate Octahydrate	210000	460
Sodium Sulfate	2600	5.8
Sodium/Potassium Sulfite	17000	37.
Sulfuric Acid	560	1.2
Trisodium Hydrogen Ethylene Diamine Tetraacetate	140000	300
Trisodium Phosphate	750	1.7

[a] Based on projections of the maximum amount of waste that might be treated in any given year.

[b] Evaluated as hexavalent chromium.

TABLE 3-10. CHARACTERIZATION OF POTENTIAL WASTE RESIDUES IN CRUSHED CONTAINERS

Chemical	Maximum Annual Amount[a] Grams	Pound
Acetic Acid	3,500	7.8
Acetone	11,000	23
Aluminum Chloride	910	2.0
Ammonium [(ethylenedinitro) tetraaceto] ferrate (III)	2400	5.2
Ammonium Hydroxide	260	0.58
Ammonium Thiosulfate	24,000	53
Benzene	77	0.17
Benz(a)anthracene	34	0.075
Benz(a)pyrene	7.7	0.017
Beryllium	0.42	0.00093
Borneol	170	0.37
Butanol, n-	500	1.1
Chloroform	85	0.19
Chromium	0.28	0.00063
Copper	0.55	0.0012
Cyanide	0.0000021	0.0000000046
Dichloromethane	190	0.41
Ethyl Alcohol	20,000	44
Ethyl Benzene	420	0.92
Dibromoethane, 1,2-	2.2	0.0048
Dichloroethane, 1,2-	260	0.57
Gluconic Acid	230	0.50
Glycerin	220	0.48
Isopropyl Alcohol	9,200	20
Methyl Alcohol	21,000	45
Methylaminoethanol	220	0.48
Methylene Chloride	5,400	11
Methyl Ethyl Ketone	1,700	3.8
Naphthalene	780	1.7
Nickel	0.0055	0.000012
Nitromethane	83	0.18
PCB	0.00042	0.000000
Perchloroethene	13,000	29
Phenanthrene	210	0.46
Pyrene	430	0.94

(Continued)

TABLE 3-10. (Continued)

Chemical	Maximum Annual Amount[a] Grams	Pound
Silver	260	0.58
Sodium Acetate	230	0.50
Sodium Bisulfite	4,800	11
Sodium Metaborate Octahydrate	230	0.50
Sodium Sulfate	310	0.69
Sodium Sulfite	280	0.61
Sodium Tetraborate	3,200	7.0
Terpinol, α-	1,900	3.4
Trichloroethane, 1,1,1-	50,000	110
Trichloroethane, 1,1,2-	19	0.040
Trichloroethene	1,500	3.3
Trichloro-1,2,2-trifluoroethane,1,1,2-	100,000	230
Trisodium Hydrogen Ethylene Diamine Tetraacetate	150	0.33
Trisodium Phosphate	220	0.48
Water	12,000	270
Xylene	83	0.18
Zinc	0.38	0.00083

[a] Based on projections of the maximum amount of waste that might be treated in any given year.

designed to shred solid classified and non-classified mixed waste. More specifically, these wastes may be contaminated with depleted uranium, thorium, and mixed fission products. As with the size reduction operations, much of the material is comprised of metals such as iron, aluminum, chromium, and steel. The wastes treated in this unit will not contain any liquids.

3.8 Waste Storage

Wastes are stored in a variety of container types and sizes in Areas 514 and 612, and Building 693 (future). The wastes stored on site could encompass all the waste previously characterized as well as wastes which are not treated on site.

4.0 EMISSIONS

A discussion of emissions from the hazardous waste treatment units at Lawrence Livermore National Laboratory (LLNL) includes the same units listed in Section 3.0. These units are:

- Wastewater Treatment Tank Farm;
- Waste Solidification in Buildings 419 and 514;
- Bulking of Wastes;
- Silver Recovery from Spent Photographic Wastes;
- Empty Container Crushing;
- Size Reduction of Contaminated Materials;
- Shredding of Contaminated Materials; and
- Storage

Emissions are based on the types and quantities of wastes as well as the physical characteristics of the operations. The methodologies used to quantify emissions are health conservative and will overstate any actual emissions. Emission calculations are presented in Appendix B. Quantities of wastes used in the calculations represent the maximum possible amount of waste that would be treated at each unit.

4.1 Wastewater Treatment Tank Farm

The Wastewater Treatment Tank Farm located at building 514 is designed to treat aqueous hazardous and mixed wastes. The operation consists of six 1,800 gallon treatment tanks, associated pumps and piping components, a diatomaceous earth vacuum filtration unit, and a bulking station.

Wastes are brought to the area and poured into the bulking station. This station is essentially a large funnel which feeds the waste via a pump to one or more of the treatment tanks. The bulking station is not covered. As liquid is poured into the bulking station, a portion of the volatile chemicals in the waste will be emitted.

The wastewater then undergoes a series of batch treatments. These treatments may include one or all of the following:

- Flocculation;
- Neutralization;
- Clarification;
- Oxidation;
- Reduction;
- Precipitation; and
- Separation.

Since these tanks are only partially covered, any volatiles that are in the waste may be emitted to the atmosphere.

In addition to the above treatments, batches of waste may also be passed through a vacuum filter. This filter is designed to remove particulates in the waste. Volatile chemicals will also be emitted from the filter.

Each batch of waste is likely to undergo a different series of treatments than another waste batch. This complexity, combined with the fact that volatile chemicals will be released during each phase of treatment, makes a rigorous determination of emissions difficult. To ensure a health-conservative estimate of emissions, it was assumed that 100 percent of all volatile chemicals in the waste treated at the tank farm are entirely emitted. Such an assumption will overstate actual emissions. For purposes of this assessment, "volatile chemical" is defined as any chemical whose saturation vapor pressure at 20°C was greater than 1 mm Hg. The emission of chemicals from the Wastewater Treatment Tank Farm is presented in Table 4-1. Based on an assumption of continuous treatment, these emissions represent both maximum annual emissions as well as peak hour emissions.

Most of the radionuclides in the waste are neither volatile nor would be present as part of a volatile compound. The only volatile compound containing a radionuclide is tritiated water. It is assumed to be 100 percent emitted. The remaining radionuclides are

TABLE 4-1. POTENTIAL EMISSIONS[a] FROM THE WASTEWATER TREATMENT TANK FARM

Chemical	Emission Rate		
	gram/sec	pounds/yr	pounds/hr
Acetone	7.3×10^{-5}	5.1×10^{0}	5.8×10^{-4}
Ammonia	6.6×10^{-3}	4.6×10^{2}	5.3×10^{-2}
Benzene	6.2×10^{-7}	4.3×10^{-2}	4.9×10^{-6}
Chloroform	2.4×10^{-8}	1.7×10^{-3}	1.9×10^{-7}
Dibromoethane, 1,2-	9.9×10^{-9}	6.9×10^{-4}	7.8×10^{-8}
Dichloroethane, 1,2-	1.2×10^{-6}	8.3×10^{-2}	9.5×10^{-6}
Ethyl Benzene	1.9×10^{-6}	1.3×10^{-1}	1.5×10^{-5}
Hydrogen Chloride	2.9×10^{-4}	2.0×10^{1}	2.3×10^{-3}
Hydrogen Fluoride	2.7×10^{-4}	1.9×10^{1}	2.1×10^{-3}
Hydrogen Peroxide	1.3×10^{-4}	8.8×10^{0}	1.0×10^{-3}
Methyl Ethyl Ketone	4.4×10^{-3}	3.1×10^{2}	3.5×10^{-2}
Methylene Chloride	4.1×10^{-6}	2.8×10^{-1}	3.2×10^{-5}
Nitric Acid	1.5×10^{-3}	1.0×10^{2}	1.2×10^{-2}
Perchloroethene	1.6×10^{-3}	1.1×10^{2}	1.3×10^{-2}
Perchloroethane, 1,1,2,2-	1.2×10^{-5}	8.3×10^{-1}	9.5×10^{-5}
Toluene	3.1×10^{-6}	2.2×10^{-1}	2.5×10^{-5}
Trichloroethane, 1,1,1-	3.3×10^{-3}	2.3×10^{2}	2.7×10^{-2}
Trichloroethane, 1,1,2-	8.5×10^{-8}	5.9×10^{-3}	6.7×10^{-7}
Trichloroethene	9.1×10^{-6}	6.3×10^{-1}	7.2×10^{-5}
Trichloro-1,2,2-trifluoroethane, 1,1,2-	1.1×10^{-3}	8.0×10^{1}	9.1×10^{-3}
Xylene	9.2×10^{-6}	6.4×10^{-1}	7.3×10^{-5}

[a]Emission rates are based on health-conservative assumptions, using the maximum possible quantity of wastewater that may be treated annually.

not expected to exist in the wastewater as part of volatile compounds. The emission rates of radionuclides are presented in Table 4-2.

4.2 Waste Solidification

Waste solidification treatment units are located in buildings 419 and 513. Liquid wastes and a solidification agent are transferred into containers. The contents of the drum are mixed or stirred until solid. The drums are then sealed and stored in the yard of Area 612.

Emission of volatile compounds occurs as the waste liquids and solidifying agents are poured into the containers. As they enter the container, volatile chemicals in the liquid waste enter the air space in the container. As the container is filled with liquid and a solidification agent, the vapors are pushed from the container. These vapors are then vented from the room and emitted to the atmosphere.

The emission rate of chemicals depends upon the chemical concentrations in the liquid, the chemical vapor pressures, and the amount of waste solidified. Emissions were estimated using the following assumptions:

- The concentration of chemicals in the air space is calculated using Raoult's Law assuming saturation for the liquid composition shown in Section 3.2;
- The volume of air displaced equals the maximum volume of liquid which can be solidified; and
- Only volatile chemicals are emitted from solidification. A volatile chemical is defined as any chemical with a saturation vapor pressure greater than 1 mm Hg at 20°C.

These assumptions are health-conservative and will overstate emissions.

TABLE 4-2. RADIONUCLIDE EMISSIONS FROM THE WASTEWATER TREATMENT TANK FARM

Nuclide	Symbol	Annual Activity Curies
Tritium[a]	^{3}H	0.1

[a] Tritium is emitted as tritiated water.

Maximum annual emissions from Building 419 and 513 are presented in Tables 4-3 and 4-4 respectively.

Maximum one hour emissions for Buildings 419 and 513 were calculated assuming that 125 and 110 gallons of waste, respectively, could be treated in any given hour. These emissions are listed in Table 4-5 and 4-6.

Only those radionuclides which might be part of a volatile chemical present in the waste would be emitted during solidification. The waste characterization lists only the actual radionuclide. The emission rate of radionuclides would be dependent upon the chemical's vapor pressure. The only volatile compound containing a radionuclide is tritiated water. The maximum possible emission rates of tritium from waste solidification at Building 419 and 513 are 7.7×10^{-13} and 8.2×10^{-13} curies per second, respectively.

4.3 Bulking

Waste bulking conducted outside building 614 involves the consolidation of similar wastes from small containers into larger containers. As these chemicals are transferred from one container to another, the air in the large container will be expelled. Any chemical vapors will be emitted into the air.

The concentration of volatile chemicals in the vapor space was assumed to equal the saturation concentration. This is a health-conservative assumption. The annual quantity of saturated air purged from the larger container was assumed to equal the maximum quantity of waste expected to be bulked during any given year. The annual average emission rates of chemicals from chemical bulking are presented in Table 4-7.

Maximum one hour emissions were calculated assuming that the maximum bulking rate is approximately 55 gallons per hour. These emission are presented in Table 4-8.

TABLE 4-3. POTENTIAL EMISSION RATES FROM WASTE SOLIDIFICATION AT BUILDING 419

Chemical	Emission Rate	
	grams/sec	pounds/yr
Acetone	3.1×10^{-8}	2.2×10^{-3}
Benzene	2.8×10^{-13}	2.0×10^{-8}
Chloroform	1.7×10^{-14}	1.2×10^{-9}
Dibromoethane, 1,2-	1.1×10^{-15}	7.9×10^{-11}
Dichloroethane, 1,2-	7.7×10^{-13}	5.4×10^{-8}
Ethyl Alcohol	1.8×10^{-5}	1.2×10^{0}
Ethyl Benzene	1.5×10^{-13}	1.0×10^{-8}
Hydrogen Chloride	2.4×10^{-10}	1.6×10^{-5}
Hydrogen Fluoride	1.2×10^{-10}	8.0×10^{-6}
Methyl Ethyl Ketone	4.2×10^{-8}	2.9×10^{-3}
Methylene Chloride	8.6×10^{-12}	6.0×10^{-7}
Nitric Acid	1.9×10^{-7}	1.3×10^{-2}
Perchloroethene	9.6×10^{-10}	6.7×10^{-5}
Toluene	7.2×10^{-13}	5.0×10^{-8}
Trichloroethane, 1,1,1-	4.1×10^{-8}	2.8×10^{-3}
Trichloroethane, 1,1,2-	1.5×10^{-14}	1.1×10^{-9}
Trichloroethene	6.1×10^{-11}	4.3×10^{-6}
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	3.6×10^{-8}	2.5×10^{-3}
Xylenes	6.5×10^{-13}	4.5×10^{-8}

TABLE 4-4. POTENTIAL EMISSION RATES FROM WASTE SOLIDIFICATION AT BUILDING 513

Chemical	Emission Rate grams/sec	Emission Rate pounds/yr
Acetone	3.1×10^{-8}	2.2×10^{-3}
Benzene	2.9×10^{-8}	2.0×10^{-3}
Chloroform	1.8×10^{-9}	1.2×10^{-4}
Dibromoethane, 1,2-	1.2×10^{-10}	8.3×10^{-6}
Dichloroethane, 1,2-	8.1×10^{-8}	5.7×10^{-3}
Ethyl Alcohol	1.8×10^{-5}	1.2×10^{0}
Ethyl Benzene	1.5×10^{-8}	1.1×10^{-3}
Hydrogen Chloride	2.4×10^{-10}	1.6×10^{-5}
Hydrogen Fluoride	1.2×10^{-10}	8.0×10^{-6}
Methyl Ethyl Ketone	4.2×10^{-8}	2.9×10^{-3}
Methylene Chloride	9.0×10^{-7}	6.3×10^{-2}
Nitric Acid	1.9×10^{-7}	1.3×10^{-2}
Perchloroethene	2.2×10^{-6}	1.5×10^{-1}
Toluene	7.5×10^{-8}	5.2×10^{-3}
Trichloroethane, 1,1,1-	9.2×10^{-5}	6.4×10^{0}
Trichloroethane, 1,1,2-	1.6×10^{-9}	1.1×10^{-4}
Trichloroethene	1.9×10^{-7}	1.3×10^{-2}
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	8.0×10^{-5}	5.6×10^{0}
Xylenes	6.7×10^{-8}	4.6×10^{-3}

TABLE 4-5. POTENTIAL 1-HOUR EMISSION RATES FROM WASTE SOLIDIFICATION AT BUILDING 419

Chemical	Emission Rate grams/sec	Emission Rate pounds/hr
Acetone	5.0×10^{-6}	4.0×10^{-5}
Benzene	4.6×10^{-11}	3.6×10^{-10}
Chloroform	2.7×10^{-12}	2.2×10^{-11}
Dibromoethane, 1,2-	1.8×10^{-13}	1.5×10^{-12}
Dichloroethane, 1,2-	1.3×10^{-10}	9.9×10^{-10}
Ethyl Alcohol	2.9×10^{-3}	2.3×10^{-2}
Ethyl Benzene	2.4×10^{-11}	1.9×10^{-10}
Hydrogen Chloride	2.8×10^{-2}	2.2×10^{-1}
Hydrogen Fluoride	6.5×10^{-3}	5.2×10^{-2}
Methyl Ethyl Ketone	6.8×10^{-6}	5.4×10^{-5}
Methylene Chloride	1.4×10^{-9}	1.1×10^{-8}
Nitric Acid	3.7×10^{-5}	2.9×10^{-4}
Perchloroethene	1.6×10^{-7}	1.2×10^{-6}
Toluene	1.2×10^{-10}	9.3×10^{-10}
Trichloroethane, 1,1,1-	6.6×10^{-6}	5.2×10^{-5}
Trichloroethane, 1,1,2-	2.5×10^{-12}	1.9×10^{-11}
Trichloroethene	9.9×10^{-9}	7.9×10^{-8}
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	5.8×10^{-6}	4.6×10^{-5}
Xylenes	1.0×10^{-10}	8.3×10^{-10}

TABLE 4-6. POTENTIAL 1-HOUR EMISSION RATES FROM WASTE SOLIDIFICATION AT BUILDING 513

Chemical	Emission Rate grams/sec	Emission Rate pounds/hr
Acetone	3.5×10^{-6}	2.8×10^{-5}
Benzene	3.3×10^{-6}	2.6×10^{-5}
Chloroform	2.0×10^{-7}	1.6×10^{-6}
Dibromoethane, 1,2-	1.4×10^{-8}	1.1×10^{-7}
Dichloroethane, 1,2-	9.2×10^{-6}	7.3×10^{-5}
Ethyl Alcohol	2.0×10^{-3}	1.6×10^{-2}
Ethyl Benzene	1.7×10^{-6}	1.4×10^{-5}
Hydrogen Chloride	2.0×10^{-2}	1.6×10^{-1}
Hydrogen Fluoride	4.6×10^{-3}	3.6×10^{-2}
Methyl Ethyl Ketone	4.7×10^{-6}	3.8×10^{-5}
Methylene Chloride	1.0×10^{-4}	8.1×10^{-4}
Nitric Acid	2.6×10^{-5}	2.0×10^{-4}
Perchloroethene	2.5×10^{-4}	1.9×10^{-3}
Toluene	8.4×10^{-6}	6.7×10^{-5}
Trichloroethane, 1,1,1-	1.0×10^{-2}	8.3×10^{-2}
Trichloroethane, 1,1,2-	1.8×10^{-7}	1.4×10^{-6}
Trichloroethene	2.1×10^{-5}	1.7×10^{-4}
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	9.1×10^{-3}	7.2×10^{-2}
Xylenes	7.6×10^{-6}	6.0×10^{-5}

TABLE 4-7. POTENTIAL BULKING EMISSION RATES

Chemical	Emission Rate grams/sec	Emission Rate pounds/yr
Acetic Acid	1.4×10^{-8}	9.8×10^{-4}
Acetone	8.1×10^{-8}	5.6×10^{-3}
Acetonitrile	6.1×10^{-10}	4.2×10^{-5}
Benzene	1.1×10^{-8}	7.8×10^{-4}
Butanol, n-	4.7×10^{-8}	3.3×10^{-3}
Butyl Glycidyl Ether, n-	6.4×10^{-10}	4.5×10^{-5}
Chloroform	3.8×10^{-8}	2.6×10^{-3}
Cyclohexane	6.9×10^{-8}	4.8×10^{-3}
Dichlorotrifluoroethane	5.3×10^{-8}	3.7×10^{-3}
Diacetone Alcohol	7.2×10^{-9}	5.0×10^{-4}
Ethyl Alcohol	6.6×10^{-8}	4.6×10^{-3}
Ethyl Benzene	1.0×10^{-11}	7.3×10^{-7}
Dibromoethane, 1,2-	9.6×10^{-14}	6.7×10^{-9}
Dichloroethane, 1,2-	6.1×10^{-12}	4.2×10^{-7}
Heptane	8.2×10^{-8}	5.7×10^{-3}
Isobutane	9.1×10^{-3}	$6.3 \times 10^{+2}$
Isopropyl Alcohol	7.5×10^{-9}	5.2×10^{-4}
Methyl Alcohol	1.2×10^{-7}	8.0×10^{-3}
Methyl Cyclohexane	8.1×10^{-8}	5.6×10^{-3}
Methyl Ethyl Ketone	1.3×10^{-8}	9.0×10^{-4}
Methyl Isobutyl Ketone	5.1×10^{-8}	3.6×10^{-3}
Methylene Chloride	1.6×10^{-7}	1.1×10^{-2}
Perchloroethene	1.5×10^{-8}	1.0×10^{-3}
Tetraethylsilicate (Ethyl Silicate)	3.5×10^{-8}	2.4×10^{-3}
Toluene	1.6×10^{-7}	1.1×10^{-2}
Trichloroethane, 1,1,1-	6.5×10^{-8}	4.5×10^{-3}
Trichloroethane, 1,1,2-	5.8×10^{-13}	4.1×10^{-8}
Trichloroethylene	9.4×10^{-8}	6.6×10^{-3}
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	6.7×10^{-7}	4.7×10^{-2}
Xylene	3.6×10^{-8}	2.5×10^{-3}

4.4 Silver Recovery

The silver recovery operation in building 514 is an electrochemical operation similar to electroplating. The spent photographic fixing solutions are placed in a tank with two electrodes. When a current is applied to the electrodes the silver is plated on to the cathode (the negative electrode).

The cause of emissions from silver recovery is the same as the cause of emissions from electroplating in general. As the current is applied to the solution, a small amount of the water is dissociated into hydrogen and oxygen. As these gases form they rise to the surface as bubbles. When the bubbles break the surface of the water they create a fine mist. Any chemical in the solution will also be in the mist. The concentration of chemicals in the mist is assumed to equal the concentration in the solution.

The mist is carried out of the room by the ventilation system and emitted into the atmosphere. The emission factors used to estimate emissions from this treatment unit were based on the emission factors developed for chrome plating by the California Air Resources Board (ARB, 1988). This emission factor assumes that emissions are directly proportional to the current applied and the hours of operation. The ARB chromium emission factor was adjusted to account for the difference in concentrations between chromium in plating and the individual chemical concentrations in the waste.

Annual average and maximum hourly emissions from silver recovery are presented in Table 4-9 and 4-10 respectively. The emission calculations are based on the conservative assumption that 100 percent of the mist generated is emitted from the room. The room vent, however, is not specifically designed to remove mist generated by plating. It is therefore unlikely that 100 percent of the mist is emitted from the silver recovery room.

The spent photographic chemicals treated at the silvery recovery treatment unit do not contain any radionuclides.

TABLE 4-8. POTENTIAL 1-HOUR EMISSION RATES FROM BULKING

Chemical	Emission Rate grams/sec	Emission Rate pounds/hr
Acetic Acid	9.6×10^{-7}	7.6×10^{-6}
Acetone	5.5×10^{-6}	4.3×10^{-5}
Acetonitrile	4.1×10^{-8}	3.3×10^{-7}
Benzene	7.6×10^{-7}	6.0×10^{-8}
Butanol, n-	3.2×10^{-6}	2.5×10^{-5}
Butyl Glycidyl Ether, n-	4.4×10^{-8}	3.5×10^{-7}
Chloroform	2.6×10^{-6}	2.0×10^{-5}
Cyclohexane	4.7×10^{-6}	3.7×10^{-5}
Dichlorotrifluoroethane	3.6×10^{-6}	2.9×10^{-5}
Discetone Alcohol	4.9×10^{-7}	3.9×10^{-6}
Ethyl Alcohol	4.5×10^{-6}	3.6×10^{-5}
Ethyl Benzene	7.1×10^{-10}	5.7×10^{-9}
Dibromoethane, 1,2-	6.5×10^{-12}	5.2×10^{-11}
Dichloroethane, 1,2-	4.1×10^{-10}	3.3×10^{-9}
Heptane	5.6×10^{-6}	4.5×10^{-5}
Isobutane	6.2×10^{-1}	$4.9 \times 10^{+0}$
Isopropyl Alcohol	5.1×10^{-7}	4.1×10^{-6}
Methyl Alcohol	7.8×10^{-6}	6.2×10^{-5}
Methyl Cyclohexane	5.5×10^{-6}	4.4×10^{-5}
Methyl Ethyl Ketone	8.8×10^{-7}	7.0×10^{-6}
Methyl Isobutyl Ketone	3.5×10^{-6}	2.8×10^{-5}
Methylene Chloride	1.1×10^{-5}	8.8×10^{-5}
Perchloroethene	1.0×10^{-6}	8.0×10^{-6}
Tetraethylsilicate (Ethyl Silicate)	2.4×10^{-6}	1.9×10^{-5}
Toluene	1.1×10^{-5}	8.8×10^{-5}
Trichloroethane, 1,1,1-	4.4×10^{-6}	3.5×10^{-5}
Trichloroethane, 1,1,2-	4.0×10^{-11}	3.2×10^{-10}
Trichloroethylene	6.4×10^{-6}	5.1×10^{-5}
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	4.6×10^{-5}	3.6×10^{-4}
Xylene	2.5×10^{-6}	2.0×10^{-5}

TABLE 4-9. POTENTIAL EMISSION RATES FROM SILVER RECOVERY

Chemical	Emission Rate grams/sec	Emission Rate pounds/yr
Acetic Acid	1.4×10^{-5}	9.6×10^{-1}
Alkali Metal Acetate	1.3×10^{-7}	8.7×10^{-3}
Alkali Metal Sulfate	2.5×10^{-7}	1.7×10^{-2}
Aluminum Chloride	3.9×10^{-7}	2.7×10^{-2}
Aluminum Sulfate	3.6×10^{-8}	2.5×10^{-3}
Ammonium Hydroxide	1.0×10^{-6}	7.2×10^{-2}
Ammonium Sulfate	1.0×10^{-6}	6.9×10^{-2}
Ammonium Thiosulfate	1.6×10^{-4}	1.1×10^{1}
Ammonium [(ethylenedinitrilo)tetraacetol] ferrate	9.3×10^{-6}	6.5×10^{-1}
Boric Acid	8.9×10^{-9}	6.2×10^{-4}
Chromium [a]	1.5×10^{-10}	1.0×10^{-5}
Gluconic Acid	9.9×10^{-6}	6.9×10^{-1}
Glutaraldehyde	7.9×10^{-8}	5.5×10^{-3}
Glycerin	3.6×10^{-8}	2.5×10^{-3}
Hydroquinone	2.0×10^{-7}	1.4×10^{-2}
Isopropyl Alcohol	7.1×10^{-9}	4.96×10^{-4}
Methylaminoethanol	3.6×10^{-8}	2.5×10^{-3}
Potassium Hydroxide	1.6×10^{-7}	1.1×10^{-2}
Potassium Metabisulfite	7.9×10^{-8}	5.5×10^{-3}
Pyrolidinone, 1-phenyl-3-	1.6×10^{-8}	1.1×10^{-3}
Pyrolidinone, n-methyl-2-	7.9×10^{-8}	5.5×10^{-3}
Silver	8.8×10^{-8}	6.1×10^{-3}
Sodium Acetate	2.5×10^{-7}	1.8×10^{-2}
Sodium Bisulfate	1.8×10^{-5}	1.3×10^{0}
Sodium Metaborate Octahydrate	9.8×10^{-6}	6.8×10^{-1}
Sodium Sulfate	1.3×10^{-7}	9.0×10^{-3}
Sodium/Potassium Sulfite	7.9×10^{-7}	5.5×10^{-2}
Sulfuric Acid	2.7×10^{-8}	1.9×10^{-3}
Trisodium Hydrogen Ethylene Diamine Tetraacetate	6.6×10^{-6}	4.6×10^{-1}
Trisodium Phosphate	3.6×10^{-8}	2.5×10^{-3}

[a] Evaluated as hexavalent chromium.

TABLE 4-10. POTENTIAL 1-HOUR MAXIMUM EMISSION RATES FROM SILVER RECOVERY

Chemical	Emission Rate grams/sec	Emission Rate pounds/hr
Acetic Acid	1.16×10^{-4}	9.22×10^{-4}
Alkali Metal Acetate	1.05×10^{-6}	8.32×10^{-6}
Alkali Metal Sulfate	2.10×10^{-6}	1.66×10^{-5}
Aluminum Chloride	3.30×10^{-6}	2.62×10^{-5}
Aluminum Sulfate	3.00×10^{-7}	2.38×10^{-6}
Ammonium Hydroxide	8.64×10^{-6}	6.86×10^{-5}
Ammonium Sulfate	8.39×10^{-6}	6.66×10^{-5}
Ammonium Thiosulfate	1.35×10^{-3}	1.07×10^{-2}
Ammonium [(ethylenedinitrilo)tetraacetol] ferrate	7.78×10^{-5}	6.17×10^{-4}
Boric Acid	7.50×10^{-8}	5.95×10^{-7}
Chromium [a]	1.23×10^{-9}	9.74×10^{-9}
Gluconic Acid	8.35×10^{-5}	6.63×10^{-4}
Glutaraldehyde	6.60×10^{-7}	5.24×10^{-6}
Glycerin	3.00×10^{-7}	2.38×10^{-8}
Hydroquinone	1.65×10^{-6}	1.31×10^{-5}
Isopropyl Alcohol	6.00×10^{-8}	4.76×10^{-7}
Methylaminoethanol	3.00×10^{-7}	2.38×10^{-6}
Potassium Hydroxide	1.32×10^{-6}	1.05×10^{-5}
Potassium Metabisulfite	6.60×10^{-7}	5.24×10^{-6}
Pyrolidinone, 1-phenyl-3-	1.32×10^{-7}	1.05×10^{-6}
Pyrolidinone, n-methyl-2-	6.60×10^{-7}	5.24×10^{-6}
Silver	7.38×10^{-7}	5.86×10^{-6}
Sodium Acetate	2.13×10^{-6}	1.69×10^{-5}
Sodium Bisulfate	1.53×10^{-4}	1.22×10^{-3}
Sodium Metaborate Octahydrate	8.25×10^{-5}	6.55×10^{-4}
Sodium Sulfate	1.05×10^{-6}	8.33×10^{-6}
Sodium/Potassium Sulfite	6.60×10^{-6}	5.24×10^{-5}
Sulfuric Acid	2.25×10^{-7}	1.79×10^{-6}
Trisodium Hydrogen Ethylene Diamine Tetraacetate	5.50×10^{-5}	4.37×10^{-4}
Trisodium Phosphate	3.00×10^{-7}	2.38×10^{-6}

[a] Evaluated as hexavalent chromium.

4.5 Empty Container Crushing

The crushing of empty drums and containers is a potential source of chemical emissions. Although the containers are always dry when crushed, it is possible that residual chemical vapors in the drum might be purged to the atmosphere as the container is crushed. The emission rate of chemical vapors would depend on how many containers were crushed, the internal volume of each container, and the amount of chemical vapor in the each container.

The amount of chemical vapor in each container is based on the quantity of residual chemicals. It was assumed that 100 percent of all volatile chemicals in the residue were emitted. The estimated annual emissions are presented in Table 4-11. Since not all containers contain volatile residual chemicals, this approach should overstate actual emissions.

The maximum emissions that might occur in any given hour depend on the amount of container volume crushed during any given hour. The drum crusher is capable of crushing 30 55-gallon drums per hour. Maximum hourly emission rates are listed in Table 4-12.

4.6 Size Reduction

Size reduction in building 419 is used to facilitate decontamination and disposal of large or complex pieces of equipment. Equipment is cut into smaller sizes by the use of a plasma arc torch or a cutoff saw. The cutting is done in a closed room. This room is vented to insure that dust created during the cutting does not exit the room by the doors. Also, the room vent is located away from the area where cutting is done, and the exhaust is vented through a HEPA filter. The fumes and particles created during cutting are heavy and will settle to the floor before they can be entrained in the ventilation air flow. Therefore, emissions of particulate matter from size reduction are assumed to be negligible.

TABLE 4-11. POTENTIAL CONTAINER CRUSHING ANNUAL EMISSION RATES

Chemical	Emissions grams/sec	Emissions Pounds/yr
Acetic Acid	1.1×10^{-4}	7.8
Acetone	3.4×10^{-4}	23.
Ammonium Hydroxide	8.4×10^{-6}	0.58
Benzene	2.5×10^{-6}	0.17
Butanol, n-	1.6×10^{-5}	1.1
Chloroform	2.7×10^{-6}	0.19
Dichloromethane	6.0×10^{-6}	0.41
Ethyl Alcohol	6.4×10^{-4}	44.
Ethyl Benzene	1.3×10^{-5}	0.92
Dibromoethane, 1,2-	6.9×10^{-8}	0.0048
Dichloroethane, 1,2-	8.2×10^{-6}	0.57
Isopropyl Alcohol	2.9×10^{-4}	20.
Methyl Alcohol	6.5×10^{-4}	45.
Methylene Chloride	1.7×10^{-4}	12.
Methyl Ethyl Ketone	5.4×10^{-5}	3.8
Nitromethane	2.6×10^{-6}	0.18
Perchloroethene	4.1×10^{-4}	29.
Trichloroethane, 1,1,1-	1.6×10^{-3}	110
Trichloroethane, 1,1,2-	5.9×10^{-7}	0.041
Trichloroethene	4.7×10^{-5}	3.3
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	3.3×10^{-3}	230
Water	3.8×10^{-3}	260
Xylene	2.6×10^{-6}	0.18

TABLE 4-12. MAXIMUM 1-HOUR EMISSION RATES CONTAINER CRUSHING

Chemical	Emission Rate grams/sec	Emission Rate pounds/hr
Acetic Acid	5.89×10^{-2}	4.68×10^{-1}
Acetone	1.77×10^{-1}	1.41×10^{0}
Ammonium Hydroxide	4.39×10^{-3}	3.49×10^{-2}
Benzene	1.29×10^{-3}	1.02×10^{-2}
Butanol, n-	8.31×10^{-3}	6.60×10^{-2}
Chloroform	1.42×10^{-3}	1.13×10^{-2}
Ethyl Alcohol	3.34×10^{-1}	2.65×10^{0}
Ethyl Benzene	6.94×10^{-3}	5.51×10^{-2}
Dibromoethane, 1,2-	3.60×10^{-5}	2.86×10^{-4}
Dichloroethane, 1,2-	4.32×10^{-3}	3.43×10^{-2}
Isopropyl Alcohol	1.53×10^{-1}	1.22×10^{0}
Methyl Alcohol	3.44×10^{-1}	2.73×10^{0}
Methylene Chloride	9.00×10^{-2}	7.14×10^{-1}
Methyl Ethyl Ketone	2.85×10^{-2}	2.26×10^{-1}
Nitromethane	1.39×10^{-3}	1.10×10^{-2}
Perchloroethene	2.17×10^{-1}	1.73×10^{0}
Trichloroethane, 1,1,1-	8.39×10^{-1}	6.66×10^{0}
Trichloroethane, 1,1,2-	3.09×10^{-4}	2.45×10^{-3}
Trichloroethene	2.46×10^{-2}	1.95×10^{-1}
Trichloro-1,2,2-trifluoroethane, 1,1,2-	1.73×10^{0}	1.37×10^{1}
Water	2.01×10^{0}	1.60×10^{1}
Xylene	1.39×10^{-3}	1.10×10^{-2}

An additional mitigating factor is the lack of volatile chemicals associated with the materials undergoing size reduction.

4.7 Shredding

The shredder in Building 513 uses blades to rip and shred drums and their contents into smaller and more manageable pieces. The cutting action of the blades is such that no fine metal particles are formed during the shredding. None of the shredded pieces is small enough to be emitted from the shredder. Since no volatile chemicals are expected to be in the drums that are shredded, and since the shredder will not create any particles which might be emitted, the emissions from this treatment unit are assumed to be negligible.

4.8 Waste Storage

Although a wide variety of container types are used to store the wastes at LLNL, there are essentially only two methods of storage: sealed containers or vented tanks.

It is assumed that sealed containers remain sealed during storage. Since the containers are sealed, no chemicals are emitted.

Emission from vented storage tanks is caused by expansion and contraction of the air in the unfilled portion of the tank. As the ambient temperature rises during the day, the air temperature in the tank rises and the air expands. During expansion, part of the air is vented from the tank. If this air contains any chemical vapors, these chemicals are also emitted.

Emissions from these tanks are therefore directly related to the amount of air in the tank and to the concentration and volatility of chemicals in the liquid. The tanks are generally maintained at a full level. This results in a lower volume of air and decreases the likelihood of emissions. The waste in these tanks is comprised of dilute aqueous wastes.

The combination of these factors makes significant emissions from chemical storage unlikely. Emissions are therefore assumed to be negligible.

5.0 HAZARD IDENTIFICATION

The Lawrence Livermore National Laboratory (LLNL) hazardous waste storage and treatment units emits organic and metal as well as radioactive constituents. The hazard identification process was intended to select the emissions that would potentially pose the most significant health risks to the surrounding public. This screening process focused the risk assessment on the compounds of greatest concern and deleted from further consideration those compounds with relatively low toxicity at given emission rates.

Two factors considered in the hazard identification process were the quantity emitted and the toxicity of the particular pollutant. Threshold Limit Values (TLVs®) were used as indicators of relative toxicity when screening the noncarcinogenic compounds. A TLV® is an eight-hour time-weighted-average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed, day after day, without adverse effect (American Conference of Governmental Industrial Hygienists, 1989). Compounds with TLVs® of 100 parts per million (ppm) or greater were omitted from the risk assessment. In this case, TLVs® did not serve as indicators of toxicity but suggest relative magnitudes of potential hazards from different emissions. All compounds evaluated as carcinogens by the U.S. Environmental Protection Agency's (EPA) Carcinogenic Assessment Group (CAG) or by the California Department of Health Services (DHS) were included, regardless of their emission rates. A minimum emission rate of one pound per year (lb/yr) was used as a screening criteria for the remainder of the nonradioactive, noncarcinogenic compounds for which TLVs® were below 100 ppm.

Table 5-1 presents the list of compounds in the wastes that have been excluded from the risk assessment and an indication of the basis for exclusion. Compounds appearing in the waste characterization results (Section 3.0) that are not deleted in Table 5-1 have been evaluated in the risk assessment and are shown in Table 5-2.

TABLE 5-1. SUMMARY OF RATIONALE FOR EXCLUSION OF COMPOUNDS

Compound	Reason for Exclusion[a]
Acetic Acid	Low Toxicity; Noncarcinogenic
Acetone	TLV = 750 ppm; Noncarcinogenic
Acetonitrile	<1 lb per year; Noncarcinogenic
Alkali Metal Acetate	<1 lb per year; Noncarcinogenic
Alkali Metal Sulfate	<1 lb per year; Noncarcinogenic
Ammonium Thiosulfate [d]	Low Toxicity;[b] Noncarcinogenic
Aluminum Chloride	<1 lb per year; Noncarcinogenic
Aluminum Sulfate	<1 lb per year; Noncarcinogenic
Ammonium [(ethylenedintrilo) tetraacetol]ferrate [d]	<1 lb per year; Noncarcinogenic
Boric Acid	<1 lb per year; Noncarcinogenic
Butyl Gycidyl Ether, n-	<1 lb per year; Noncarcinogenic
Cyclohexane	TLV = 300 ppm, Noncarcinogenic
Diacetone Alcohol	<1 lb per year; Noncarcinogenic
Dichlorotrifluoroethane	TLV = 1,000 ppm [c], Noncarcinogenic
Ethyl Alcohol	TLV = 1,000 ppm, Noncarcinogenic
Ethyl Benzene	TLV = 100 ppm; Noncarcinogenic
Gluconic Acid	<1 lb per year; Noncarcinogenic
Glutaraldehyde	<1 lb per year; Noncarcinogenic
Glycerin	<1 lb per year; Noncarcinogenic

(Continued)

TABLE 5-1. (Continued)

Compound	Reason for Exclusion[a]
Heptane, n-	TLV = 400 ppm; Noncarcinogenic
Hydroquinone	<1 lb per year; Noncarcinogenic
Isobutane	TLV = 600 ppm; Noncarcinogenic
Isopropyl Alcohol	TLV = 400 ppm; Noncarcinogenic
Methyl Alcohol	TLV = 200 ppm; Noncarcinogenic
Methyl Cyclohexane	TLV = 400 ppm; Noncarcinogenic
Methyl Ethyl Ketone	TLV = 200 ppm; Noncarcinogenic
Methyl Isobutyl Ketone	<1 lb per year; Noncarcinogenic
Methylaminoethanol	<1 lb per year; Noncarcinogenic
Nitro Methane	TLV = 100 ppm; Noncarcinogenic
Potassium Hydroxide	<1 lb per year; Noncarcinogenic
Potassium Metabisulfite	<1 lb per year; Noncarcinogenic
Pyrrolidinone, 1-phenyl-3-	<1 lb per year; Noncarcinogenic
Pyrrolidinone, n-methyl-2-	<1 lb per year; Noncarcinogenic
Silver	<1 lb per year; Noncarcinogenic
Sodium Acetate	<1 lb per year; Noncarcinogenic
Sodium Metaborate Octahydrate	<1 lb per year; Noncarcinogenic
Sodium/Potassium Sulfite	<1 lb per year; Noncarcinogenic

(Continued)

TABLE 5-1. (Continued)

Compound	Reason for Exclusion[a]
Sulfuric Acid Sodium Bisulfate Sodium Sulfate	<1 lb per year; Noncarcinogenic
Toluene	TLV = 100 ppm; Noncarcinogenic
Trichloroethane, 1,1,1-	TLV = 350 ppm; Noncarcinogenic
Trichloro-1,2,2-trifluoro-ethane, 1,1,2-	TLV = 1,000 ppm; Noncarcinogenic
Trisodium Hydrogen Ethylene Diamine Tetraacetate	<1 lb per year; Noncarcinogenic
Trisodium Phosphate	<1 lb per year; Noncarcinogenic
Xylenes (o-, p-, and m-isomers)	TLV = 100 ppm; Noncarcinogenic

[a] TLV's from ACGIH, 1989.

[b] Inert invivo, poorly absorbed in the intestine, up to 12.5 grams injected intraveneously without ill effects (Gasselin, 1984).

[c] TLV® from Aviado, Domingo et al., 1980.

[d] Hazard ID exclusion state refers only to the ion accompanying the ammonia. All ammonia emissions will be included in the Risk Assessment.

TABLE 5-2. COMPOUNDS INCLUDED IN THE RISK ASSESSMENT

Ammonia Compounds[a]	Hydrogen Fluoride
Benzene[b]	Hydrogen Peroxide
Butanol, n-	Methylene Chloride[b]
Chloroform[b]	Nitric Acid
Chromium VI[b]	Perchloroethene[b]
Dibromoethane, 1,2-[b]	Perchloroethane, 1,1,2,2-
Dichloroethane, 1,2-[b]	Tetraethylsilicate (Ethyl Silicate)
Hexane, n-	Trichloroethane, 1,1,2-[b]
Hydrogen Chloride	Trichloroethene[b]

[a] Only the ammonia fraction of the ammonia compounds will emitted.

[b] Carcinogenic Compound

6.0 DISPERSION AND DEPOSITION MODELING

Modeling is an important component of a health risk assessment. Following the identification of hazards and emissions calculations, the modeling process focuses on where pollutants end up in the environment and in what quantity. Using meteorological data, terrain data, and characteristics of release points, modeling provides an estimate of pollutant dispersion in the air and deposition rates on the ground.

For the Lawrence Livermore National Laboratory (LLNL) hazardous waste treatment and storage facility, concentration and deposition rates were calculated at a series of downwind locations using two U.S. Environmental Protection Agency (EPA)-approved dispersion models coupled with site-specific meteorological data. This section provides an overview of the modeling methodology employed in the risk assessment and a brief discussion of the models that have been used, the receptor grid (locations where concentration estimates were obtained), and the meteorological data incorporated into the models. Section 6.1 is specific to nonradioactive emissions modeling and Section 6.2 provides a discussion of modeling for radionuclide emissions.

6.1 Dispersion Modeling Methodology for Nonradioactive Emissions

The characteristics of release points are crucial variables in the modeling process. The movement of emissions differs depending on whether they are released from a point or area source. In the case of LLNL hazardous waste treatment and storage operations, most emissions will enter the atmosphere through dedicated vents and hence, were modeled as point sources. The wastewater treatment facility consists of several open top tanks that will act as an elevated area source. Bulking operations involve transferring liquid waste from small containers into drums. Volatile organic compounds will be released from the drums and are best modeled as point sources. Table 6-1 lists the individual sources modeled and their release configuration (i.e., point source or area source).

TABLE 6-1. LLNL WASTE TREATMENT AND STORAGE EMISSIONS SOURCES [a]

Source	Source No.	Location	Source Type
Solidification	101	419	Point[b]
Solidification	102	513	Point
Wastewater Treatment	103	513	Area (10m x 10m)
Silver Recovery	104	514	Point
Bulking	105	614	Point
Container Crushing	106	612	Point

[a] Only units for which emissions have been quantified are included in the modeling. Storage was not judged to be a significant emission source.

[b] Solidification emissions at building 419 may be released from 1 of 3 roof vents. The worst-case vent was used in the modeling.

The stack characteristics of Source 101 (located at the center of Building 419) represent the most conservative of the three individual sources which are located at various locations within (and vented from) this building. Sample model runs were made using each of the three individual sources to make this most conservative selection. The maximum ground-level concentrations (GLC) varied by approximately a factor of two among the three individual sources. Placing the selected source 101 at the center of the building rather than considering three separate source locations is reasonable given that the off-site areas of interest for risk analysis are located at least several hundred meters away.

Downwash effects from surrounding buildings were considered for all point sources. Sample sensitivity model runs were made, and it was determined that building downwash effects gave more conservative GLCs for sources 101 and 102 out to at least 300 meters, but resulted in lower concentrations for all other sources. Thus, building downwash effects in the production model runs were retained for sources 101 and 102 only.

For the area source representing the tank farm as well as an adjacent bulking area and open door venting from the east side of Building 514, an area ten meters on a side was chosen. The ISCST model requires square areas, and this 10m by 10m area is a reasonable representation of the total source area. The areas of interest for risk analysis are also located at least several hundred meters away, making this simple configurational representation reasonable.

The terrain in the immediate vicinity of LLNL is flat, but rolling hills higher than the roof vents or release points are present within 2 kilometers (km) east and south of the site. The presence of these hills requires the use of a dispersion model capable of simulating impacts on complex terrain. Of the three commonly used U.S. EPA dispersion models [Industrial Source Complex Short-Term (ISCST), Complex I, and SHORTZ], the only approved models with this capability are SHORTZ and Complex I. Complex I is typically the preferred model for regulatory application, but is not valid for area or volume sources. SHORTZ, on the other hand, is not restricted by release configurations and, therefore, was used for the complex terrain modeling. For areas of simple terrain (terrain

below vent height), the ISCST model is used as required by the California Air Resources Board (ARB).

To handle two dispersion models for a large receptor grid, a post-processor was developed to read the model results from ISCST and SHORTZ (binary output files) and create an input file for the Radian Risk Assessment Model (R-RAM®) to be used in estimating exposure. The processor reads model results at each receptor, and based on receptor height and release height, selects the appropriate model result for each source/receptor combination. In some cases, both the ISCST and SHORTZ results will be used at a given receptor because of the difference in the release height of the roof vents and other sources.

The SHORTZ and ISCST computer programs are designed to calculate the short- and long-term ground-level pollutant concentrations produced at selected receptors by emissions from multiple stack, building, and area sources. Each model uses sequential short-term (usually hourly) meteorological inputs to calculate concentrations for averaging times ranging from one hour to one year. The model options that have been used for this risk assessment follow the U.S. EPA Guidelines on Air Quality Models (Office of Air Quality Planning and Standards). Specific options that have been used are indicated in the model results in Appendix B.

The dispersion models were run using a unit emission rate of 1 gram per second (g/sec) for each of the sources. The actual concentration of a pollutant at a given receptor is calculated by multiplying the concentration in units of micrograms per cubic meter per gram per second ($\mu g/m^3/g/sec$) by the emission rate (grams per second) estimated for that pollutant and source. The total concentration ($\mu g/m^3$) of any pollutant at any receptor is the sum of the pollutant-specific contribution of each source.

The ISCST and SHORTZ dispersion models were run in the gaseous mode in accordance with ARB guidelines. This assumes that all pollutants exist in the gas phase with 100 percent reflection from ground surfaces (i.e., no deposition or plume depletion). This implies that the particulate pollutants, such as chromium, would disperse throughout

the study area and would not deposit on soil or above ground surfaces. In reality, the solid-phase particles are subject to deposition and a corresponding net decrease of mass contained in the plume. The need for assuming that all pollutants behave as a gas with 100 percent reflection stems from the absence of any agency-approved dispersion model suitable for simulating dispersion and transport of fine particulate matter. By neglecting deposition and plume depletion, the models produce an overestimate of concentrations at distant receptors.

Both the ISCST and SHORTZ models can be run in a deposition mode, but only when large particles (>20 micrometers) are involved. Methods for simulating deposition in the absence of an approved model are discussed in greater detail in Section 6.1.3.

6.1.1 Meteorological Data

The purpose of a dispersion model is to simulate the movement of a pollutant through the atmosphere and estimate the resulting downwind concentration. Atmospheric transport and the ground level impact of an emissions source are a function of source characteristics and prevailing weather conditions and wind patterns for the specific location being evaluated. Therefore, to ensure a realistic simulation that will provide reasonably accurate ambient concentration estimates, local meteorological data are an important component of any dispersion modeling exercise.

The models used for this risk assessment require hourly measurements of wind speed, wind direction, temperature, stability, and mixing height from a monitoring station representative of the Livermore Valley. LLNL operates an on-site meteorological station and has nine years of data available. The Bay Area Air Quality Management District (BAAQMD) has suggested the use of data collected at the nearby Livermore Airport. If sufficiently complete, on-site measurements are preferred over a more distant station, especially since available wind direction data from Livermore Airport were recorded only

to 8 points of the compass (45° sectors) instead of to the nearest degree as for the on-site data.

A review of the LLNL on-site data indicates 97 percent data capture for 1988, 88 percent capture from 1987, and 85 percent data capture from 1986. Most of the missing data for the latter two years resulted from individual month-long outages. Although 1987 and 1986 data are not complete, there is sufficient data from the previous year to fill in data gaps. Therefore, the LLNL on-site data were judged to be superior to the Livermore Airport data and were consequently used in the risk assessment.

Table 6-2 shows ranked periods with continuous hours of missing on-site data for LLNL from 1985 through 1988. Since it is desirable to process three years of data through the models, 1986, 1987, and 1988 data were selected for modeling. The missing months of data in both 1986 and 1987 were filled in with 1985 calendar-equivalent data. With the added 1985 data, the effective data capture for 1986 and 1987 increases to over 96 percent. Periods of a few hours with missing surface data were filled with wind speeds of 1.0 meters per second (m/sec) and wind direction the same as the previous good hour. Selection of the calms processing option for ISC then considered these periods as calm and did not use the data in the modeling. Days with eight hours or more of missing data were completely omitted from modeling.

The dispersion models require both surface meteorological data and mixing height information [where mixing height is defined as the height above the surface through which relatively vigorous vertical mixing occurs (Holzworth, 1972)]. Vertical profiles of temperature (obtained from rawinsonde measurements), and surface temperature are required to compute mixing height by the EPA-approved Holzworth method. Precipitation occurrence data are required to flag periods when the assumptions regarding mixing height computation do not apply, and thus mixing heights cannot be specifically computed using the Holzworth methodology. Ideally, surface and upper air temperatures and hourly observations of precipitation would be obtained from the same location; however, such is frequently not the case. For instance, in this case, the nearest upper air (rawinsonde)

TABLE 6-2. RANKED LISTING OF NUMBER OF CONTINUOUS HOURS MISSING FOR LLNL ON-SITE METEOROLOGICAL DATA

Year	Number of Continuous Hours Missing	Number of Occurrences	Remarks
1985	744	1	August missing
	160	1	
	96	1	
	26	1	
	25	1	
	5	2	
	2	1	
	1	1	
Yearly Total	1,064 (12.2%)		
1986	1,028	1	Jun 19 - Jul 30
	240	1	missing
	51	1	
	24	1	
	8	1	
Yearly Total	1,351 (15.4%)		
1987	744	1	May missing
	160	1	
	46	1	
	27	1	
	24	1	
	5	2	
	1	1	
Yearly Total	1,012 (11.6%)		
1988	78	1	
	63	1	
	39	1	
	37	1	
	24	1	
	8	1	
	6	1	
	4	1	
	3	1	
	2	1	
	1	12	
Yearly Total	276 (3.1%)		

station is Oakland. Mixing height data for Oakland itself would be obtainable from the National Climatic Data Center (NCDC), but adjustments would still be required for the LLNL site based on the on-site surface temperature. The BAAQMD has a set of nomographs to perform this adjustment for various Bay Area locations.

Rather than relying on the BAAQMD nomographs and the Oakland upper air data, an alternative method for generating mixing heights for the LLNL site was used. This method involves the use of a Radian program that directly computes mixing heights for the LLNL site by the Holzworth method. The Radian program is equivalent to that used by the National Climatic Data Center. Needed inputs are sounding data from the nearest upper air station (Oakland), local surface temperature data (the LLNL on-site data), and hourly precipitation information from the San Francisco Airport. On-site LLNL hourly precipitation data have not been previously recorded. Analysis of the climatological record of precipitation frequencies at San Francisco and Oakland indicates that use of either data set would produce comparable mixing height estimates. The San Francisco precipitation data was used because of its ready availability.

For the periods in 1986 and 1987 where surface temperature data were filled in with calendar-equivalent 1985 data, mean seasonal mixing heights from Holzworth (1972) were used to prevent using 1985 surface data with 1986 or 1987 upper air data in the computation routine. These seasonal values were also used for filling in precipitation and cold advection periods when mixing height could not be computed using the Holzworth methodology.

6.1.2 Receptor Grid Selection

The receptor grid used in the risk assessment is composed of a course grid with 250 meter spacing, numerous fine grids with 100 meter receptor spacing, and several discreet receptors representing census tracts and areas of interest. The grid system, described above, is illustrated in Figure 6-1. The two rows of receptors placed along and west of the west boundary of the site were used to demonstrate that the highest point of

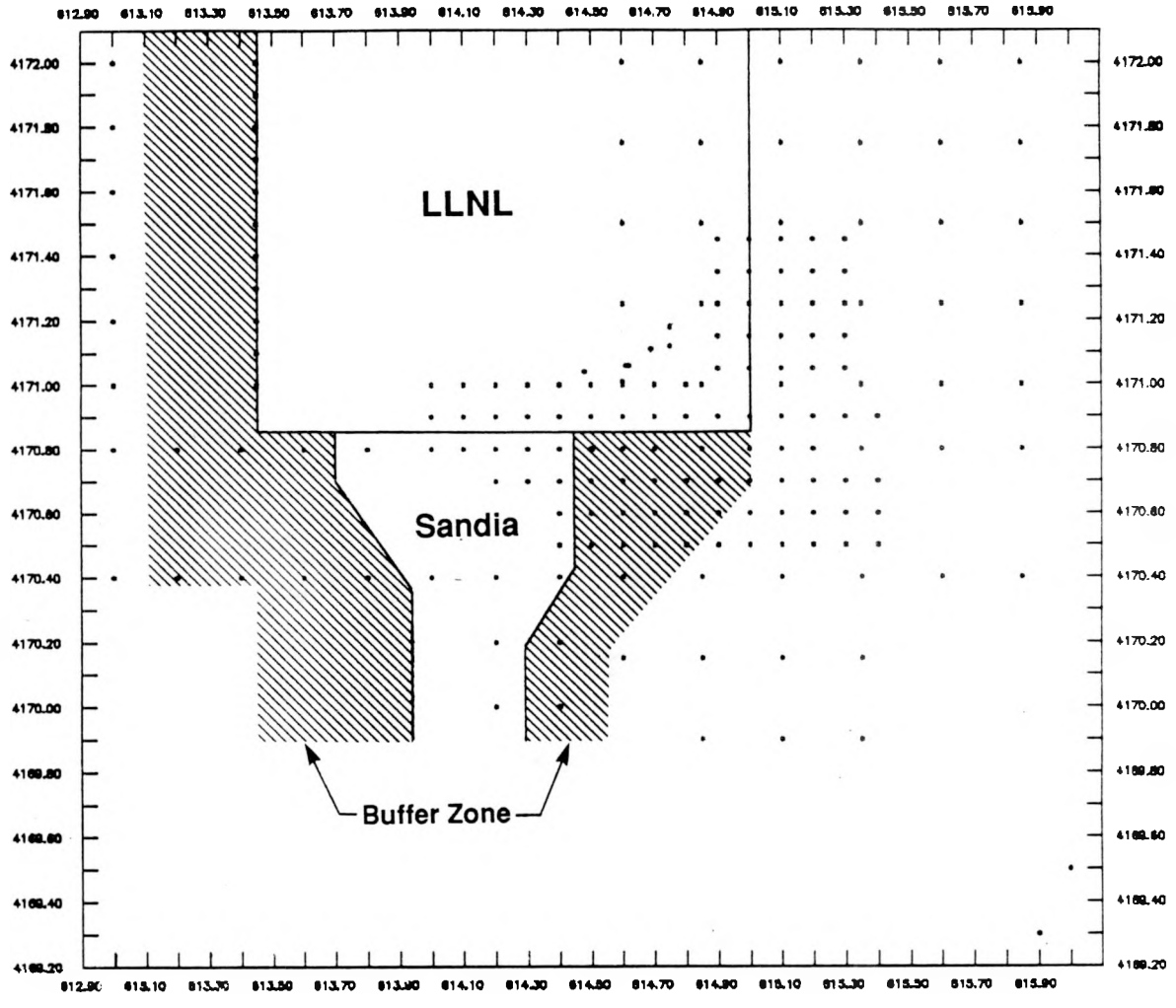

Figure 6-1. Receptor Grid.

impact does not occur to the west of the site. Table 6-3 identifies those receptors that are located in an area that would permit residential dwellings (i.e., areas not within LLNL property, the DOE buffer zone, or Sandia Laboratory).

For large census tracts located outside the uniform grids, several discreet receptors were used, spaced evenly throughout the tract. If the tract was relatively small, only one receptor was used. The Universal Transverse Mercator (UTM) coordinates and receptor numbers can be found in Appendix B in the post-processed model results and in Figure 6-1.

6.1.3 Deposition Modeling

The deposition rate, or rate at which a particle falls from the atmosphere, is a function of particle size and atmospheric conditions. Currently, there are no computer models approved by the EPA or ARB that will calculate deposition with any level of accuracy. The ISCST and SHORTZ models include deposition algorithms that calculate deposition by applying a settling velocity to the modeled ground-level concentration (GLC), but this is only representative for large particles [20 micrometers (μm) and larger] where settling velocity is the dominant mechanism for deposition. For small particles (less than 10 μm), deposition is governed predominantly by diffusion and meteorological conditions.

The deposition flux of solid particulates used in this risk assessment was calculated using a default methodology that tends to overestimate the deposition. The ambient concentrations, calculated by the ISCST and SHORTZ models, are multiplied by a default deposition velocity of two centimeters per second (cm/sec), recommended by ARB, which yields a flux term in units of $\mu g/m^2$-sec. A basic assumption in calculating deposition using the ARB approach is that deposition has no effect on the ground-level concentrations. In reality, as particulates are removed by deposition, the plume becomes depleted, and the GLCs decrease. Therefore, the ARB methodology is inherently conservative, overestimating both the rate of deposition and the airborne concentration of solid phase pollutants.

TABLE 6-3. RECEPTORS IN RESIDENTIAL LAND USE[a]

3	23	111	172	190	217	245
4	24	112	173	191	218	246
5	30	113	174	194	219	247
6	31	114	175	195	220	248
12	32	124	177	198	224	254
13	33	125	178	199	234	255
14	39	126	184	200	235	256
15	40	127	185	203	238	257
21	41	128	186	204	237	258
22	42	171	189	205	244	259

[a]Receptors fall in a location that would permit a residential dwelling. Land use zoning could be residential or rural/agricultural. Refer to R-RAM® results for receptor numbers and locations.

The ISCST and SHORTZ dispersion model results can be found in Appendix D.

6.2 Dispersion Modeling Methods for Radionuclide Emission Sources

The EPA AIRDOS model was used to simulate transport of radionuclide emissions from LLNL waste treatment and storage operations. Appendix E presents the AIRDOS model outputs.

AIRDOS consists of two components: a Gaussian air dispersion model and a terrestrial fate/human exposure assessment model. This section describes the air dispersion model and Section 10.0 discusses the exposure assessment model. The dispersion model estimates ground-level concentrations and deposition fluxes at user-specified locations. The assumptions, algorithms, and data used in the dispersion model are similar to other EPA guideline air quality models. Air concentrations and deposition fluxes are sector-averaged on a radial grid. Deposition modeling accounts for both dry deposition (does not apply to tritium) and scavenging (wet deposition). The model also accounts for plume depletion using a source depletion algorithm.

AIRDOS uses a modified Gaussian plume dispersion model to calculate annual average radionuclide concentrations for each grid sector. Building wake effects and downwash are not included in the AIRDOS-EPA models. The same type of rise calculation (buoyant, momentum, or fixed) is used for all sources. As many as six sources may be assessed, but for calculational purposes, they are all considered to be co-located at the origin of the assessment grid.

Input to AIRDOS includes a source term, which describes facility-specific parameters, and options that control the dispersion model and exposure calculations; meteorological data are also required as input. AIRDOS assigns default values for all other parameters. AIRDOS is formatted to read a stability array rather than the detailed hourly data used in ISCST and SHORTZ. For consistency, the 1986, 1987, and 1988 on-site data

prepared for nonradioactive emissions modeling (ISCST and SHORTZ) were processed and converted into stability array format for use with the AIRDOS model.

Options selected for the AIRDOS dispersion model were:

- Point and area sources;
- A circular grid with a 20 km radius;
- Sector-averaged radionuclide concentrations; and
- Buoyant plume rise calculations.

The source term includes annual radionuclide release rates, stack height, diameter, heat release rate, and stack gas exit velocity. The radionuclide release rates are listed in Section 4.0 and Appendix B of this document. Table 6-4 lists the release parameters used for the modeling effort.

TABLE 6-4. SOURCE PARAMETER FOR AIRDOS

Source	Release Height (m)	Source Type	Exit Velocity (m/s)	Diameter (m^2)	Temper-ature (k)
101	9.5	Point	4.56	0.56	290
102	6.4	Point	3.10	0.20	290
103	3.0	Area	NA[a]	NA	290

[a] Area source with dimensions of 10m x 10m.

7.0 ENVIRONMENTAL FATE AND HUMAN EXPOSURE ASSESSMENT

The objectives of the exposure assessment are two-fold. The first step is to identify all relevant exposure pathways for the Livermore area. An exposure pathway is defined as any activity through which an individual receives some level of exposure to a pollutant. For example, one exposure pathway would be through consumption of vegetables subject to pollutant deposition. Secondly, the exposures contributed by each pathway are estimated and then summed to estimate the total daily exposure of each pollutant emitted from the hazardous waste treatment and storage units. A quantitative evaluation of the potential for adverse effects from these exposures is presented in Section 11.0.

Based on the land use and geographic characteristics within the Livermore area, the following pathways were considered to be potentially significant contributors to total daily exposure for the Lawrence Livermore National Laboratory (LLNL) hazardous waste treatment and storage facility:

- Inhalation;
- Ingestion of soil, locally grown fruits and vegetables, potable water, locally produced wine; and
- Direct dermal contact.

There were no significant sources of fresh water fish or poultry operations identified in the study area (greater Livermore area); therefore, these were not considered potential exposure routes in this risk assessment.

As previously discussed, this risk assessment has evaluated worst-case and plausible-case health risks. Differences in these cases include the assumptions and values

used in the exposure assessment. Parameters that vary depending on case are presented in Table 7-1.

As required in the California Air Pollution Control Officers Association (CAPCOA) manual (CAPCOA, 1987), exposure was estimated for a hypothetical maximally-exposed individual (MEI). This hypothetical individual is assumed to reside for 70 years at the location of greatest off-site exposure. This approach provides a highly conservative estimate of the actual exposure. It assumes that the individual is always in the same location and exposed to the same ambient concentration, which would seldom, if ever, occur. Periods spent away from the residence during work or vacation would result in lower exposure. The above factors, combined with the assumption of 100 percent absorption for the inhalation pathway and the inherent conservatism of the Industrial Source Complex Short-Term (ISCST) and SHORTZ models, tend to overestimate exposure.

The following subsections describe the methods used to estimate environmental fate and human exposure from the pathways listed above. The methodology presented here has been submitted to and approved by the California Department of Health Services (DHS) in past risk assessments[1] and is presented here again for complete documentation.

7.1 Inhalation Pathway

7.1.1 Inhalation Exposure

Exposure to pollutants suspended in ambient air occurs through inhalation of both gases and particulates. For the purpose of this assessment, pollutants adsorbed to inhaled particulates were considered to be entirely absorbed in the lungs, yielding a conservative estimate of exposure. In reality, only a fraction of the inhaled particulates would deposit in the alveolar region of the lung and be absorbed by the body fluids in the

[1] Examples of these include the Casmalia Resources Health Risk Assessment (HRA), Milliken Waste-to-Energy HRA, and Chevron Chemical Company HRA.

TABLE 7-1. EXPOSURE ASSESSMENT VARIABLES FOR THE WORST-CASE AND PLAUSIBLE SCENARIOS

Variable	Worst Case	Plausible
Exposure periods	70 years	30 years
Soil ingestion	1.0×10^{-4} kg/day	7×10^{-5} kg/day
Plant interception fraction	0.054	0.06
Cleansing efficiency	0.08	0.25
Plant consumption rate	0.15 kg/day	0.03 kg/day
Root uptake factors	See Table 7-10	See Table 7-10
Soil-half lives	See Table 7-6	See Table 7-6

Note: References for the parameters selected in the worst and plausible cases are provided in the following subsections.

lung. Generally, particulates ranging in diameter from 0.1 to 10 microns would become deposited in the lung (these constitute the respirable fraction of an aerosol). Smaller particulates would be exhaled while larger particulates would become deposited in the upper respiratory tract. Particulates deposited in the upper respiratory tract would be cleared by ciliary action and then swallowed. Exposure to non-respirable particulates would then occur through the gastrointestinal (GI) tract. Some particulate clearance would also occur from the alveolar regions.

Absorption of volatile organic compounds (VOCs) in the lung is dependent upon the solubility of the vapor in lung tissue and the concentration of the vapor in the gas phase (Menzel and Amdur, 1986). Generally, absorption of VOCs is not complete. For example, retention of inhaled trichloroethylene has been reported to range from 33 to 74 percent (Monster et al., 1976); hence, the assumption that inhaled pollutants are absorbed completely probably results in an overestimate of exposure.

7.1.2 Inhalation Calculations

Exposure to inhaled pollutants is determined by multiplying the estimated concentration in air by an average daily inhalation volume and dividing that quantity by the body weight. Table 7-2 shows the equation and variables used in the calculation.

This approach provides a conservative estimate of the exposure due to inhalation. It assumes that the 20 cubic meter (m^3) per day respiration rate is constant, and that the individual remains in the same location exposed to the same ambient concentration of pollutant for an entire lifetime. Periods spent away from the residence for vacation or work would result in an exposure less than the maximum ground-level concentration (GLC). These factors, combined with the assumption of 100 percent absorption and the conservative assumptions inherent in the dispersion models, overpredict inhalation exposure, though the magnitude of this overprediction is not known.

TABLE 7-2. INHALATION EXPOSURE

$$X_{I,i} = \frac{GLC_i * RR * C_1}{BM}$$

Where: $X_{I,i}$ = Inhalation exposure to chemical i, mg/kg/day

GLC_i = Ground-level concentration of chemical i, $\mu g/m^3$

RR = Respiration rate, 20 m^3/day

C_1 = 1 mg/1000 μg

BM = Body mass, 70 kg

7.2 Ingestion Pathway

Several potential exposure routes via ingestion are discussed in the subsections that follow. These include ingestion of soil, plants, wine, and drinking water. Of the nonradioactive pollutants potentially emitted from LLNL, only hexavalent chromium is subject to deposition and accumulation in the food chain. Therefore, the following subsections describing the ingestion exposure methodology apply only to chromium. These discussions are preceded by a brief discussion of gastrointestinal absorption factors.

7.2.1 Gastrointestinal Absorption Factors

In calculating risk from a pollutant for which ingestion is the route of exposure, it is necessary to estimate the total dose received. The total dose received is in turn dependent upon the absorption of the chemical across the lining of the gastrointestinal (GI) tract. The amount of a chemical available for absorption may differ from one matrix to another (e.g., fly ash versus water or food). A GI factor is often used to adjust for this difference in availability, particularly when a potency has been developed using a matrix with greater absorption efficiency than is expected for the human exposure route. In the context of this risk assessment, GI factors were not necessary to adjust the absorbed dose.

This risk assessment conservatively assumes hexavalent chromium presents a cancer risk by the ingestion route. However, no cancer potency factor specific to ingestion is currently available. In the absence of an ingestion potency, the CAPCOA manual mandates the use of the inhalation potency factor for calculating ingestion risk (CAPCOA, 1987). Because of the differences in the exposure routes (inhalation versus ingestion), and the low rate of hexavalent chromium absorption in the GI tract, it is necessary to correct the hexavalent chromium ingestion exposure for systemic absorption and subsequent transfer to the lung. For this risk assessment, a GI factor of 10 percent has been used (EPA, 1985l).

7.2.2 Soil Pathway

7.2.2.1 Soil Exposure

Pollutants emitted from the LLNL hazardous waste storage and treatment facility in the particulate phase are subject to deposition onto surface soil. Deposition onto soil serves as a pathway for several ingestion routes of exposure. Pollutants in the soil (adsorbed to particulates in soil) may accumulate in food crops consumed by humans, and soil can be ingested directly by animals, young children, and, to a lesser degree, adults.

The concentration of pollutants in the soil depends on several competing processes. Removal processes include degradation, volatilization, leaching, resuspension, and soil runoff. These processes, in most instances, will operate simultaneously with deposition to produce a steady-state soil concentration. The methods for estimating pollutant soil concentrations in this assessment use several simplifying and conservative assumptions including:

- Compound concentrations in the soil are not assumed to be affected by any removal processes, except in the case of semivolatiles such as naphthalene and phthalates, which are subject to degradation;

- Semivolatile concentrations in the soil are estimated using a first-order rate equation. Different decay constants for each chemical were used for the worst-case plausible scenarios;

- Pollutants are assumed to be equally distributed in a layer of soil, defined by a mixing depth of 1 centimeter (cm) (i.e., all pollutants accumulate in the top 1 cm of the soil).

7.2.2.2 Soil Ingestion Exposure Calculations

The quantity of pollutants ingested with soil is assumed to be the product of the soil concentration and the soil consumption rate. A daily inadvertent soil ingestion value of 70 milligrams per day (mg/day) was used in the plausible scenario based on the work of Hawley (1985). This value is age-weighted to reflect the much higher quantities of soil inadvertently ingested by young children. A daily soil ingestion rate of 100 mg/day was used in the worst-case scenarios, based on an evaluation by Sedman (1989). Equations for calculating exposure from soil ingestion are presented in Table 7-3.

The concentration of a chemical in soil depends upon whether or not it is affected by any physical or chemical removal processes. Metals, unlike semivolatiles, are not assumed to undergo any removal process (although removal does actually occur). The soil concentrations are calculated by different equations depending upon chemical type.

As pollutants deposit on the soil, they will mix into the upper surface layers. The depth to which mixing occurs is due to several factors: water solubility, water filtration rate, organic content, soil porosity, etc. As a default value, a mixing depth of 1 cm was assumed for the worst case and 15 cm for the plausible case (U.S. Nuclear Regulatory Commission, 1975). Soil density was assumed to be 1500 kilograms per cubic meter (kg/m^2) (Perry, 1963).

7.2.3 Plant Pathway

Locally grown produce, either from commercial agriculture or family gardens, is subject to pollutant accumulation through deposition and root absorption. In the absence of any significant commercial produce (with the exception of grapes) near the LLNL site, backyard gardens were assumed to present a secondary exposure pathway. Although exposure due to wine consumption was estimated via the ingestion pathway, but not the plant pathway directly, the consumption of locally grown table grapes was included in the plant pathway calculations. Specifically, table grape consumption was included in the

TABLE 7-3. SOIL INGESTION EXPOSURE AND SOIL CONCENTRATION

Soil concentration neglecting removal processes:

$$C_{s,i} = \frac{DF_i * C_2 * t * C_1}{P * MD}$$

Soil concentration including decay:

$$C_{s,i} = \frac{DF_i * C_2 * C_1}{k_i * P * MD}(1 - e^{-k_i t})$$

$$k_i = 0.693/t_{1/2,i}$$

Exposure:

$$X_{s,i} = \frac{C_{s,i} * S * GI_{s,i}}{BM}$$

Where: $X_{s,i}$ = exposure of pollutant i from soil ingestion, mg/kg-day

$C_{s,i}$ = soil concentration of chemical i, mg/kg

DF_i = deposition flux of chemical i, $\mu g/m^2/sec$

S = soil consumption, kg/day, see text

worst-case = 1.0×10^{-4}

plausible = 7.0×10^{-5}

$GI_{s,i}$ = fractional absorption factor (see Section 7.3.1)

BM = body mass, 70 kg

C_1 = 1 mg/1000 μg

C_2 = 31,536,000 sec/yr

t = time, 70 years

P = soil density, 1,500 kg/m^3 (Perry, 1963)

MD = mixing depth, 0.01

k_i = soil elimination constant of chemical i, 1/year

$t_{1/2,i}$ = half-life of chemical i, year

estimation of exposures via ingestion of backyard garden produce. This approach is conservative because table grapes grown at the location of the MEI will have higher levels of contaminants from the LLNL waste treatment and storage facility than will table grapes grown elsewhere in the Livermore Valley.

7.2.3.1 Plant Ingestion Exposure

The concentration of pollutants in crops results from atmospheric deposition and root uptake from the soil. To determine the quantity of particulates that falls on the edible portion of above-ground plants, a weighted interception fraction was determined (Baes et al., 1984). Root vegetables, which are not subject to deposition, accumulate pollutants through root uptake and soil adhering to root surfaces. Soil ingestion estimates include a significant degree of conservatism to account for soil adhering to root vegetables. The weighted interception fraction represents the interception of deposited particulates on the edible portions of different types of crops. Total vegetative yield (i.e., the entire crop surface area receiving the particulate deposition) is several times the edible portion yield of a crop, suggesting that for certain crops only a fraction of the deposited particulates will fall onto edible portions.

Following deposition, the particulates are susceptible to removal by weathering. The half-life for particulate weathering on foliage surfaces has been calculated to be approximately 18 days, based upon the equation describing the fraction of a deposited pollutant remaining after a set period of time following deposition (U.S. Nuclear Regulatory Commission, 1975).

Accumulation of organic and inorganic species into plant tissue from the soil was considered in the assessment of exposure from crop consumption. Crop/soil uptake factors for non-organic substances were derived from Baes et al. (1984) for both the vegetative edible crops and for reproductive edible crops. According to the authors, the vegetative uptake factors are appropriate only for leafy vegetables while the reproductive uptake factor is appropriate for all other crops. They derive a weighted uptake factor

based on the relative quantity of vegetative versus reproductive plant consumption. To account for translocation of chemical species into the non-root portion of the plant, uptake factors were multiplied by the appropriate root to non-root vegetable ratio. Metals are assumed to translocate into the non-root portion of the plant. Detailed calculations of uptake factors are presented in Appendix C.

The crop/soil uptake factors represent a conservative estimate of potential uptake. The uptake factors, published in Baes et al. (1984), are derived mostly from nutrient studies, metals uptake from sewage sludge applications, or concentrations naturally in soil.

The soil concentration of emitted pollutants is calculated differently for the plant ingestion pathway than for the soil ingestion pathway. This difference is attributed to the effects of tilling or otherwise cultivating soil. The mixing depth is fixed at 15 centimeters (cm) for all scenarios examined; however, because of tilling or cultivating, pollutants will be mixed into this layer each year. At the onset of facility operation, the concentration of pollutants in soil from facility emissions is zero. The concentration then increases over time until a maximum is reached after 70 years. For metals and certain organics, loss mechanisms have not been considered in calculating soil concentrations. Therefore, an average soil concentration that would yield an equivalent lifetime exposure is the concentration determined after 35 years of deposition; 35 years represents the midpoint of the 70 year exposure period

7.2.3.2 Plant Ingestion Calculations

Exposure to contaminated plants occurs from ingestion of edible crops grown in backyard gardens. The total exposure from plant consumption is the combined exposure from:

- Consumption of pollutants deposited directly onto the plant;

- Consumption of pollutants absorbed into the above ground portion of the plant from chemicals in the soil; and

- Consumption of pollutants absorbed into the below ground portion of the plant.

The equations used for calculating the exposure from plant ingestion are presented in Table 7-4.

Fractional absorption of pollutants by the body is accounted for by applying a GI factor as shown in the equation. The rationale for the application of GI factors in the assessment of potential health risks is discussed in Section 7.2.1, Gastrointestinal Absorption Factors.

The calculation of exposure from crop consumption includes a correction for cleansing particulates from plants before ingestion. Activities in food preparation that may remove surface particulates from foliage include washing, blanching or cooking, and peeling and canning. Foliar retention of particulates may also be influenced by particle size and solubility of the deposited particulate. The available data on surface particulate removal by food preparation is obtained largely from studies of pesticide residues and radionuclide contamination.

Smaller particulates may be more resistant to removal by washing than larger particles. Foliar retention of submicrometer particles ranged from 20 to 92 percent on bean plants following leaching by simulated rainfall. Removal of particulates depended on particle size, solubility, residue time prior to leaching, and acidity of the leachate (Cataldo et al., 1981). The authors concluded that submicron particulates persisted longer on foliage than particles greater than 1 micrometer (μm).

These data suggest that the amount of surface particulates removed during food preparation may be quite variable. This variability is assessed by using a removal efficiency of eight percent, the lowest value from Cataldo et al. (1981) in the worst-case scenario, and

TABLE 7-4. PLANT CONSUMPTION EXPOSURE

Exposure

$$X_{p,i} = \frac{[(C_{D,i} * 1\text{-}CE * GI_{P,i}) + (C_{R,i} * GI_{T,i})]\ AC + (C_{R,i} * GI_{T,i} * BC)}{BM}$$

Deposition concentration:

$$C_{D,i} = \frac{DF_i * C_3 * I * C_1}{Y * k_w} * (1 - e^{-k_w t_w})$$

Root uptake concentration:

$$C_{R,i} = C_{s\text{-}ave,i} * RUF_i$$

Average soil concentration (without removal):

$$C_{s\text{-}ave,i} = \frac{DF_i * C_1 * C_2 * t}{D * MX}$$

Average soil concentration (with removal):

$$C_{s\text{-}ave,i} = \frac{DF_i * C_1 * C_2 * C_4}{k_i * D * MX} (1 - e^{-k_i t_{ave,i}})$$

$$k_i = 0.693/t_{1/2,i}$$

$$t_{ave,i} = \ell n \left(\frac{1 - e^{-k_i t}}{k_i t}\right) \left(\frac{1}{-k_i}\right)$$

Where: $X_{p,i}$ = plant consumption exposure of chemical i, mg/kg/day
$C_{D,i}$ = deposition concentration of chemical i, mg/kg
$C_{R,i}$ = root uptake concentration of chemical i, mg/kg
$C_{s\text{-}ave,i}$ = average soil concentration of chemical i, mg/kg

(Continued)

TABLE 7-4. (Continued)

CE = cleaning efficiency, fraction

AC = consumption of above ground portion of crops, kg/day

BC = consumption of below ground portion of crops, kg/day

$GI_{p,i}$ = GI absorption factor of chemical i on plant surfaces

$GI_{T,i}$ = GI absorption factor of chemical i in plant tissue

BM = body mass, 70 kg

DF_i = deposition flux of chemical i, $\mu g/m^2/sec$

C_1 = 1 mg/1000 μg

C_2 = 31,536,000 sec/yr

C_3 = 86,400 sec/day

C_4 = 365 days/yr

I = interception fraction

k_w = weathering elimination constant, $days^{-1}$

t_w = days plant is exposed to weathering, 60 days

Y = plant yield, 2 kg/m^2

RUF_i = root uptake factor for chemical i

t = time, 35 years

D = soil density, 1,500 kg/m^3

MX = mixing depth, 0.15m

k_i = soil elimination constant of chemical i, 1/years

$t_{ave,i}$ = time at which average soil concentration of chemical i is reached, years

$t_{½,i}$ = half-life of chemical i in soil, years

a removal efficiency of 50 percent from Larrson and Sahlberg (1982) in the plausible scenario.

The above studies consider removal efficiencies based on two minutes of washing, which may not be appropriate for typical home food preparation. However, no studies based on short duration washing (30 seconds or less) were identified.

With the exception of locally produced grapes, land use patterns surrounding the LLNL facility include very little commercial agriculture. Produce raised in household gardens potentially represents a source of ingestion exposure. Home-grown produce appears to provide a larger contribution to the diets of rural farming and nonfarming families across the country than to the diets of urban families (U.S. EPA, 1980). Estimation of the contribution of home-grown produce for different levels of urbanization has not been performed specifically for families in California.

The U.S. EPA has reported that median daily consumption of six food groups, (cereals/grains, potatoes, leafy vegetables, legumes, root vegetables, and fruits) is 738 grams per person per day. For purposes of estimating exposure from crop ingestion, the median value for each food group was multiplied by an average percentage of annual consumption that is home grown. These values are developed by the U.S. Department of Agriculture (USDA), and are summarized by the U.S. EPA (1980a). They are presented in Table 7-5. The average consumption rate for urban households was used in the plausible scenario and the average rate for rural households was used in the upper-bound scenario.

7.2.4 Wine Pathway

Land use within the Livermore area includes both active vineyards and production wineries. The grapes grown in the area and the wine produced by these vintners have the potential to accumulate particulates and certain volatile pollutants emitted from existing mobile sources (e.g., automobiles, trucks) and stationary industrial sources found

TABLE 7-5. HOME-GROWN PRODUCE CONSUMPTION

Food Group	Median Consumption (g/day)[a]	Urban Household Percent Consumption from Home-Grown Sources	Urban Consumption (g/day)	Rural Household Percent Consumption from Home-Grown Sources	Rural Consumption (g/day)
Cereals	157.2	0.13	0.20	0.82	1.29
Potatoes	141.7	1.21	1.70	14.62	20.72
Leafy Vegetables	99.7	7.55	7.53	35.65	35.54
Legumes	98.0	7.55	7.40	35.65	34.94
Root Vegetables	109.7	7.55	8.28	35.65	39.11
Fruits	131.8	3.24	4.27	14.20	18.72
	738.1		29.4		150.32
Above Ground Consumption			20		90
Root Vegetable			10		60

Source: U.S. EPA, 1980a

[a] Average daily consumption rate of fruits and vegetables from all sources (garden, store, etc.).

at LLNL and other industrial facilities. Locally produced wine, therefore, presents a secondary exposure pathway for consumers. To examine the potential for adverse effects from LLNL operations via wine consumption, a screening-level analysis was conducted. The methodology employed in that analysis and the results obtained are discussed in the following subsection.

7.2.4.1 Wine Pathway Calculations for Nonradioactive Compounds

Accumulation of environmental pollutants in wine occurs in a manner similar to accumulation in agricultural crops or backyard garden produce (deposition of particulates onto the exposed grapes, and for certain pollutants, absorption into the root system and translocation to the fruit). Therefore, the potential concentration in wine can be estimated using methods developed for backyard garden produce by adjusting for differences in the environmental fate parameters such as interception, yield (kg/m^2), cleansing or filtration, growing period, and deposition rate.

Grapes grown in California for making table wines are produced at a rate of two to five tons of fruit per acre of vines (Cook, 1989). The lower end of this range is for Chardonnay grapes (a premium white varietal) and the upper end of the range is more representative of heavy red wines such as Cabernet. For this analysis, a value of three tons per acre was assumed to be a reasonable worst case. A larger yield, four or five tons per acre, would result in lower pollutant concentrations in the wine. In a normal year, 165 gallons of premium wine can be produced from a ton of grapes. Assuming 3 tons per acre and 165 gallons per ton of grapes, the wine yield would be 482 grams per square meter. As for deposition onto backyard gardens (refer to Section 7.2.3), not all particulates depositing on a vineyard will intercept the grapes. Using the data from Baes et al. (1984), a weighted interception fraction of 5.4 percent was calculated for the mix of crops grown in a backyard garden. The data presented by Baes et al., (1984) does not specifically include grapes. Backyard gardens and agricultural fields are typically planted with a high density and minimal spacing between plants. Vines, on the other hand, are planted in a precise manner and include much more spacing between the rows. The vegetative canopy

over grapes is also different than would be found for most other produce. The leaves are thicker and more dense, and are likely to protect the fruit to a greater degree than on other crops. In the absence of any specific data, and considering the differences noted above, an interception fraction for grapes was conservatively assumed to be approximately five percent. This means that five percent of the particulates falling on a square meter of surface area remain on the grape clusters.

The growing season for grapes is typically from April 1 through October 1 (Cook, 1989). This will allow sufficient time for steady-state levels of particulates to be reached assuming a weathering half-life equivalent to agricultural crops (18 days). The plant pathway assumes a growing period of 60 days which results in 90 percent of the equilibrium level. Therefore, in the screening-level analysis, the extended growing period must be factored into the analysis.

Wine-making involves several steps that may remove solid-phase pollutants. Most wine, and particularly today's premium varietals, follow a two step filtering process for removing impurities. The first step is a rough filtering stage composed of diatomaceous earth or a rough pad filter (Cook, 1989). Ion exchange filtration is also used at times to reduce metal content. The second stage of filtration is primarily for polishing and is designed to remove the very fine impurities and microorganisms prior to bottling (Cook, 1989). This fine filtration stage uses a tight pad filter and membrane system capable of removing particles down to 0.45 micrometers. Separating the skins from the grapes (white wines especially) and washing grapes before crushing will also remove particulates. Given today's micro-fine filtering practices, washing, and separating skins, it was assumed for this analysis that a total of 95 percent of the particulate-phase pollutants will be removed.

7.2.4.2 Wine Exposure Calculations

The average daily consumption rate for wine will vary tremendously from individual to individual. Some persons may drink little or no wine, while others might consume several bottles a week. At the time of this printing, data on the upper-bound

consumption rate could not be found. Therefore, the following assumptions were made to quantify intake under a worst-case scenario:

- Age 0 to 15 -- none consumed;
- Age 16 to 20 -- one bottle per week; and
- Age 21 to 70 -- one bottle per day.

This results in a lifetime average of nearly five bottles per week or 0.54 kilograms per day. It was assumed that all of the wine consumed originated from local wineries and vineyards. As a plausible estimate, it was assumed that individuals between the ages of 21 and 70 consumed 2 bottles a week of Livermore area wine, or 0.08 kg per day averaged over a lifetime.

The concentration of any pollutant in the wine will be a function of the above parameters and the ground-level concentration modeled at the vineyard location. A majority of the grapes are grown west and south of the LLNL facility. This is opposite the direction of predominant winds and the location of the point of maximum impact. Therefore, in evaluating the potential exposure from wine consumption, the difference in ground-level concentration of emissions at the nearest vineyard location relative to the maximum impact location must be addressed.

Exposure to nonradioactive emissions from wine ingestion was evaluated for the MEI by configuring a specific Radian Risk Assessment Model (R-RAM®) run solely for wine; for simplicity, the other pathways were not incorporated into the run. This R-RAM® run can be found in Appendix F.

LLNL conducts annual surveys of tritium levels in wines produced in the Livermore area. Past surveys have not provided data on other radionuclides. From data collected in 1987 and reported in 1988, the average level of tritium in wine was found to be 3.8×10^{-7} microcuries per milliliter (Holland et al., 1988; Holland et al., 1989). It should be noted that all measured tritium levels represent accumulation from all sources, both natural and man made and not just the tritium released from the LLNL hazardous waste

treatment and storage facilities. Therefore, in estimating exposure, the tritium levels in wine were adjusted by the ratio of hazardous waste treatment and storage emissions to total LLNL emissions as reported in Holland et al. (1988 and 1989). The data presented for tritium (adjusted) can be used with the above assumption of wine consumption to provide an estimate of exposure.

7.2.5 Drinking Water Pathway

7.2.5.1 Drinking Water Exposure

The study area for this risk assessment contains two surface water bodies that are used for drinking water, the South Bay Aqueduct (SBA) and the Patterson Reservoir. The portion of the SBA that feeds the reservoir and treatment plant is outside of the study area and not impacted by emissions from the hazardous waste treatment and storage facilities. However, the water present in the reservoir is subject to deposition and must be evaluated. A three-mile section of the SBA south of the reservoir falls within the study area and is used as a source of drinking water further downstream. Both water bodies contribute to the Livermore area drinking water supply. To evaluate any potential impacts to area residents, a screening level calculation was conducted for the most sensitive source.

7.2.5.2 Drinking Water Exposure Calculations

The concentration of compounds emitted from the LLNL facility in the SBA or reservoir water is a function of the deposition rate, the surface area of the SBA or Patterson Reservoir, and the volumetric flow rates through the two bodies of water. An engineer's report produced for Zone 7 (Alameda County, 1986b) states that the SBA has an average flow rate of 55 million gallons per day (mgd). The average flow rate through the Patterson Reservoir is 7 mgd. The surface area for the Patterson reservoir is estimated to be 23,000 square meters. The surface area of the SBA was calculated for the three-mile portion of the aqueduct within the study area assuming the aqueduct was 15 meters wide. The total surface area of the SBA was determined to be 22,000 square meters. With

the surface areas nearly equivalent and the flow rate in the reservoir being a factor of eight lower than the SBA flow, the highest concentration of emissions will occur in the reservoir water. Therefore, this screening level analysis focused on the reservoir water.

The following assumptions were used to estimate the concentration of particulate emissions in the reservoir water:

- The Patterson Reservoir surface area is 23,000 square meters;
- The deposition rate of emissions is based on the dispersion model results for the reservoir location (receptor 64) assuming a deposition velocity of 2 centimeters per second (cm/sec); and
- The reservoir is such that soil run-off will not enter the water as a result of storm drainage.

Once deposition into the reservoir has occurred, a number of mechanisms may commence that would remove constituents of concern from the water. These mechanisms include photolysis, volatilization, adsorption, biodegradation, and settling. In addition to these naturally occurring mechanisms, treatment processes at the water treatment plants would also act to remove constituents. For this analysis, loss mechanisms were neglected.

Exposure from drinking water was estimated assuming a 2 kilogram per day water ingestion rate and all water consumed originated from the aqueduct or reservoir. The equations used in R-RAM® for calculating exposure are shown in Table 7-6.

TABLE 7-6. DRINKING WATER EXPOSURE

Concentration:

$$C_{w,i} = \frac{DF_i * C_1 * AR * C_2}{FR}$$

Exposure:

$$X_{w,i} = \frac{W * C_{w,i} * GI_{w,i}}{BM}$$

Where:
$X_{w,i}$ = exposure to chemical i from consumption of drinking water, mg/kg/day
W = consumption of drinking water, 2 kg/day
$C_{w,i}$ = concentration of chemical i in drinking water, mg/kg
$GI_{w,i}$ = fractional adsorption of chemical i from drinking water
BM = body mass, 70 kg
DF_i = deposition flux of chemical i, $\mu g/m^2$-s
C^1 = mg/1000 μg
AR = surface area of reservoir, m^2
FR = net outflow of reservoir, kg/day
C_2 = 86,400 seconds per day

7.3 Dermal Pathway

7.3.1 Dermal Exposure

Dermal exposure results from deposition of airborne emissions on the skin and direct contact with surface dust. Comparisons of dermal exposure from airborne emissions and surface dust indicate that airborne emissions are insignificant compared to surface dust as a route of exposure (refer to Appendix C). Therefore, the airborne emissions have been omitted in the calculation of exposure.

7.3.2 Dermal Calculations

Dermal exposure from surface dust is calculated using the equation presented in Table 7-7.

Contact with surface dust on skin has been estimated to result in a maximum skin loading of 0.5 milligrams per square centimeter per day ($mg/cm^2/day$) (Lepow et al., 1974, and Lepow et al., 1975). Given the climate of the study area, the exposed skin surface area is conservatively assumed to be 4,170 square centimeters (cm^2) (ICRP, 1984).

TABLE 7-7. EXPOSURE FROM DERMAL CONTACT WITH SOIL

$$X_{D,i} = \frac{SD * SA * C_{s,i} * GI_{D,i}}{BM * C_1}$$

Where:
- $X_{D,i}$ = exposure to chemical i from dermal contact with soil
- SD = surface dust on skin, 0.5 mg/cm^2/day
- SA = exposed skin surface area, 4170 cm^2
- $C_{s,i}$ = soil concentration of chemical i, see Table 7-3
- $GI_{D,i}$ = fractional absorption factor for chemical
- BM = body mass, 70 kg
- C_1 = 1,000,000 mg/kg

8.0 CHARACTERIZATION OF POPULATIONS AT RISK

Potential human exposure to emissions from the Lawrence Livermore National Laboratory (LLNL) hazardous waste storage and treatment operations would be influenced by several factors. These factors include the following:

- Locations of populations relative to the emission sources;
- Types of land use surrounding the facility (i.e., residential, rural, or commercial);
- Types of water supplies; and
- Extent of local agricultural production.

Exposure to hazardous waste storage and treatment emissions from LLNL was determined for the point of highest impact and at a series of receptors corresponding to populated areas. Census data were used to identify the populated areas and the number of people in each location (census tract).

The study area for the health risk assessment is described in Section 8.1. Section 8.2 provides a description of the population at risk. Section 8.3 provides a discussion of land use characteristics of the area surrounding LLNL, as well as types of water supplies and local agricultural production.

8.1 Definition of the Study Area

The study area for this risk assessment has been arbitrarily defined as all land within a five kilometer radius of the LLNL facility. Within this boundary are seven census tracts that were used in estimating cancer burden. (Cancer burden is the increased number of cancer cases in the exposed population that could result from a 70-year exposure to

emissions from the treatment and storage operations at LLNL.) Two of these tracts (4511 and 4512) are geographically large due to low population densities.

8.2 Demographic Characterization

Based on local dispersion of pollutants from the treatment and storage operations, the potentially-exposed population would be confined to the Livermore area. The size of this population was estimated from population data for this area from the 1980 Census. Census tract data for the Livermore area are presented in Table 8-1. Population projections for each census tract in the Livermore area for the year 2025 were obtained by applying population growth rates projected by the Alameda County Planning Department and the Association of Bay Area Governments (ABAG).

Population projections for the period 1980 to 2005 for the Livermore area are presented in Table 8-2. Population in the Livermore area is expected to increase by 57 percent from 49,612 in 1980 to 78,000 in 2005 (ABAG, 1985; Alameda County Planning Department, 1986a). The 1980 residential population for the Livermore area obtained from the 1980 census data differs slightly from the population used by ABAG for projections. The 1980 census data includes only areas currently within the city limits, while ABAG included a small area outside the current city limits that may be annexed in the near future.

To estimate cancer burden for the study area population, exposure and risk were determined for each individual census tract. Because many of the census tracts cover large areas, it was necessary to use several discrete receptors spaced evenly throughout a tract to account for the spatial differences in modeled air concentrations. The 2025 population of each of these receptors was estimated to be the total tract population divided by the total number of discrete receptors. For example, tract 4511 contains nine discrete receptors and has a total population of 3,921 persons. Each individual receptor in tract 4511 would then have 436 persons. This method assumes that the population within a tract is evenly distributed over the total area.

TABLE 8-1. CENSUS TRACTS AND POPULATION SURROUNDING THE LLNL FACILITY

Tract Number	Total Population 1980[a]	Total Population 2025[b]
4511	1,875	3,921
4512	4,170	8,720
4513	5,134	10,735
4514	9,231	19,302
4515	10,287	21,511
4516	6,715	14,041
4517	10,937	22,870
Total	48,349	101,100

[a] Source: U.S. Bureau of the Census, 1980.

[b] The projected population is based on projection estimates (to the year 2005) presented in Table 5-2. Population estimates for the year 2025 are based on the assumption that the population increase after 2005 is linear.

TABLE 8-2. LIVERMORE AREA POPULATION PROJECTIONS, 1980 TO 2005[a]

Year	Residential Population
1980	49,612
1985	53,900
1990	57,400
1995	62,800
2000	68,200
2005	78,000

[a] Sources: Alameda County Planning Department, 1986c; Association of Bay Area Governments, 1985.

8.3 Land Use Characteristics

The area surrounding the LLNL site includes residential developments, as well as commercial, light industry, or rural farm land (Alameda County Planning Department, 1986b). Some acreage southwest, west, south, and east of the facility is devoted to growing grapes. The major agricultural products in Alameda County (excluding nursery products) are cattle and poultry. The surrounding land use is largely rangeland, with minor areas devoted to hay and wheat. Vegetable crops and wine grapes provide a small, but significant portion of the agricultural production in the county. The Alameda County Agricultural Crop Report makes no mention of a dairy industry in the county (County of Alameda, 1985). However, several small dairies do exist in the study area and were included in this risk assessment. Table 8-3 summarizes the crops grown and acreage harvested for the entire county.

8.4 Risk Isopleth

The California Air Pollution Control Officers Association (CAPCOA) manual requires a risk assessment to identify a one in ten million (1×10^{-7}) risk isopleth, and to consider exposure, risk, and cancer burden for all populations residing within that area. In general terms, the 1×10^{-7} risk isopleth defines the geographical area that, under worst-case exposure conditions, would be subject to a risk of 1×10^{-7} or greater. In the case of the LLNL hazardous waste storage and treatment facility, the risk level falls below the one in ten million level. Therefore, a risk isopleth has not been identified.

TABLE 8-3. CROPS AND HARVESTED ACREAGE IN ALAMEDA COUNTY IN 1985[a]

Crop	Harvested Acreage
Field Crops	
Barley	1,550
Hay, Alfalfa	1,145
Hay, Grain	9,725
Irrigated Pasture	390
Range (Pasture)	203,000
Wheat	2,525
Miscellaneous Field Crops[b]	2,390
Vegetable Crops	
Miscellaneous Vegetables	2,391[c]
Fruits and Nut Crops	
Grapes	1,661[d]
Berries	197
Walnuts	9[e]
Miscellaneous Fruit	——
Total	224,983

[a] Source: County of Alameda, 1985.

[b] Includes oats, corn, safflower, sugar beets, and stubble.

[c] Includes cabbage, cauliflower, greens, corn, cucumber, lettuce, and tomatoes.

[d] Based on Livermore Valley Wine Growers Association Survey.

[e] Includes apricots, kiwis, and persimmons.

9.0 DOSE-RESPONSE FOR NON-RADIONUCLIDES

This section presents the information necessary to convert exposures estimated in Section 7.0 to the potential risk values presented in Section 11.0. Subsection 9.1 discusses the role of dose-response in risk assessment and gives background information about the development of cancer potency factors (CPFs) and reference doses (RfDs); Section 9.2 discusses the CPFs and their derivation; and Section 9.3 presents the RfDs (values used to evaluate exposures to noncarcinogens) and their derivation.

9.1 Introduction to Dose-Response

Dose-response assessment has been defined as "an attempt to describe the expected human response to any given level of an exposure" (Hart and Turturro, 1986). Multiple governmental agencies and scientific organizations, such as the U.S. Environmental Protection Agency (EPA), the National Academy of Science, the World Health Organization, and the California Department of Health Services (DHS), have developed dose-response relationships for numerous chemicals. Dose-response assessment generates two factors for use in evaluating health effects: a CPF for carcinogens, and an RfD for substances not currently considered to be carcinogenic.

Cancer potency factors relate the risk of developing cancer to a lifetime exposure to a unit concentration or dose of a given chemical. Potency values are derived by extrapolating responses from relatively high exposures used in long-term animal bioassays or from occupational epidemiology studies to a response from low doses observed in the general population. For a noncarcinogenic chemical, the RfD defines a dose at which no adverse effects would be expected if an individual were continuously subjected to that dose for a lifetime. These values are also generally derived from the results of animal studies and human experience.

The agencies involved in quantifying dose-response relationships for various chemicals may weigh the factors which affect the dose-response relationship differently,

resulting in a range of values for the same chemical. Therefore, to assess the risk to an exposed population, it is necessary to follow these steps in a risk assessment:

1) Examine the factors that were considered in deriving the numerical values defining the dose-response;

2) Choose the dose-response scenario with the factors that most closely approximate the risk assessment scenario; and

3) Apply the dose-response values from that scenario for a particular risk assessment.

The purpose of the next section is to examine some of the factors involved in developing CPFs and RfDs, and to discuss their application for specific situations to accomplish steps 2 and 3 in the process.

9.1.1 Dose

All toxic effects due to chemical exposure, with the possible exception of carcinogenesis and mutagenicity by certain mechanisms, exhibit a threshold of response that is dose-dependent. That is, at low doses a response is not observed, but as the dose is increased and crosses a "threshold," a response is seen. Several different mechanisms may be responsible for this effect. One such mechanism is the body's ability to maintain homeostasis or to adjust to pollutant effects. Another involves the metabolic pathway(s) used to metabolize toxic substances. For example, two different pathways may be available for metabolizing a particular chemical. The normal or predominant pathway may deactivate the chemical and, therefore, no toxic response would be seen. However, the secondary pathway may activate the chemical and induce the toxic response. In this scenario, a toxic response would be seen only when the primary metabolic pathway had been overloaded by an increased dose.

The policy of the regulatory agencies is that chemicals which induce a carcinogenic response have no threshold of response. That is, any exposure to the chemical constitutes a finite risk of developing cancer.

9.1.2 Exposure Route

The exposure routes of interest in this risk assessment are discussed in Section 7.0, Environmental Fate and Human Exposure Assessment. The exposure routes for which CPFs and RfDs are commonly developed are ingestion and inhalation, primarily because these two routes typically contribute most significantly to total dose. Numerical estimates of dose-response relationships are often only available for the ingestion pathway, in which case the ingestion values can be adapted for use in other pathways. Another alternative is to adjust for the differences in absorption between the two exposure pathways and then apply the adjusted value.

The exposure route for a chemical is of critical importance in assessing the potential toxic response. In some cases, there are good data indicating that a chemical is carcinogenic by one route but not by another. This occurs most commonly with agents that show only local activity (i.e., agents that produce carcinogenic changes at the site of initial contact but not systemically). The best example of this are the large number of materials, including inert solids, that produce sarcomas at the point of injection - usually epidermal. This type of data, since it does not involve a route of exposure plausible for population exposure, is not used to develop CPFs and is interpreted as not indicating a true carcinogenic response. Other agents, most notably metal compounds, produce pulmonary epithelial tumors after inhalation exposures, but are not carcinogenic by other routes. Assuming that they are carcinogenic when ingested unrealistically increases the risk estimate.

The route of exposure may play an important role in determining bioavailability, the ability of the body to absorb the chemical from the media in which it is transported. This absorption of a chemical by a tissue is dependent on the solubility of the chemical in both aqueous and lipid solutions and on the media in which the chemical is transported.

Chemicals administered in lipid solutions are much more available for absorption by the GI tract than are chemicals bound in fly ash or in plant tissue. For this reason the CPFs and the RfDs developed from animal studies, where chemicals are often administered to test animals in corn oil, do not always directly reflect the absorption scenarios experienced by humans. Due to this difference in absorption, the CPFs and RfDs are, for the most part, very health conservative. The risk assessment can deal with this difference by considering bioavailability factors in calculating dose.

9.1.3. Worst Case and Plausible Case

Two cases were developed for this risk assessment. The worst-case scenario was developed using health-conservative values, and the plausible scenario was developed using less conservative values to provide an indication of the uncertainty in the risk assessment process. The plausible scenario is health conservative and more realistic than the worst-case scenario.

9.2 Cancer Potency Factors

Numerical estimates of cancer dose-relationships are presented as CPFs. Assuming dose-response linearity at low doses, the potency factor defines the cancer risk due to continuous lifetime exposure to one unit of carcinogen concentration. In this risk assessment, CPFs are in inverse units of milligrams per kilogram of body weight per day $(mg/kg\text{-}day)^{-1}$.

Since the risk resulting from low levels of exposure cannot be measured directly either by using animal studies or epidemiological studies, mathematical models are used to extrapolate health effects from high to low dose. The linearized multistage low-dose extrapolation model is currently used by the U.S. EPA's Carcinogen Assessment Group (CAG) and the DHS (U.S. EPA, 1986a and DHS, 1985a). Use of the multi-stage model leads to an upper limit to the risk that is consistent with some mechanisms of carcinogenesis

(U.S. EPA, 1986b); however, it should be noted that actual potencies may be considerably less than the theoretical values derived from animal studies.

The CAG has ranked the relative theoretical carcinogenic potencies of over 50 substances suspected of being human carcinogens. DHS has evaluated the carcinogenicity of 11 of these substances. Although the methods and data used by DHS and EPA are equivalent, different assumptions and interpretations have resulted in different potency estimates for the same substance. To examine the effects of this variability on estimated risk, several sets of potencies were used. The plausible-case risk estimates are based entirely on EPA potencies developed by CAG, which are generally lower than DHS values for the same substance. In the worst-case scenario, risks were estimated using DHS potencies from the 1987 California Air Pollution Control Officers Association (CAPCOA) manual (incorporating updated information not yet included in the CAPCOA manual), and EPA potencies where DHS values do not exist. An additional worst-case scenario based on all EPA potencies is also presented. Tables 9-1 and 9-2 show the potencies used in the worst-case risk calculations. Table 9-1 is based on current DHS-approved values, while Table 9-2 consists entirely of EPA values. Table 9-3 indicates the potency factors used in the plausible case. Tables 9-2 and 9-3 are both based on EPA potencies, but the substances with a weight-of-evidence classification of C (as determined by EPA) have been deleted from the risk calculations in the plausible case. These substances show only weak evidence of carcinogenicity in animals and, as a plausible assumption, present no appreciable risk of cancer to humans at low environmental exposures.

The CAPCOA manual assumes hexavalent chromium presents a cancer risk from oral exposure and has potency equivalent to that of inhalation; however, the EPA has taken an opposite stance. The EPA drinking water health advisory for chromium lists the elements with a weight-of-evidence classification of "D", which means it is not yet classified. The basis for the designation is an absence of adequate evidence to demonstrate oral carcinogenicity in laboratory animals. In the absence of conclusive evidence, EPA has elected not to consider this element as having carcinogenic potential by the oral route. EPA's position is also reflected in the regulations implementing Proposition 65. Drawing

TABLE 9-1. CANCER POTENCY FACTORS FOR THE WORST CASE - CAPCOA RECOMMENDED VALUES[a]

Compound	Inhalation $(mg/kg/day)^{-1}$	Oral $(mg/kg/day)^{-1}$	Weight of Evidence[d]
Benzene	1.70×10^{-1}	NA	A
Chloroform	8.10×10^{-2} [b]	NA	B2
Chromium VI	5.10×10^{2}	5.1×10^{2} [c]	A
Dibromoethane, 1,2-	2.40×10^{-1}	NA	B2
Dichloroethane, 1,2-	7.33×10^{-2}	NA	B2
Methylene Chloride	3.33×10^{-3}	NA	B2
Perchloroethene	3.30×10^{-3} [b]	NA	B2
Perchloroethane, 1,1,2,2-	2.00×10^{-1} [b]	NA	C
Trichloroethane, 1,1,2-	5.70×10^{-2} [b]	NA	C
Trichloroethene	1.70×10^{-2} [b]	NA	B2

[a] DHS, 1989, unless otherwise noted.

[b] U.S. EPA, 1989.

[c] DHS considers Hexavalent Chromium an ingestion carcinogen.

[d] U.S. EPA weight-of-evidence classifications:
A - Human Carcinogen (Sufficient Human Evidence)
B - Probable Human Carcinogen
B1 (Limited Human Evidence)
B2 (Sufficient Animal Evidence and Inadequate Human Evidence)
C - Possible Human Carcinogen (Limited Animal Evidence and Inadequate Human Evidence)

TABLE 9-2. CANCER POTENCY FACTORS FOR THE WORST CASE - EPA RECOMMENDED VALUES[a]

Compound	Inhalation $(mg/kg/day)^{-1}$	Weight of Evidence
Benzene	2.90×10^{-2}	A
Chloroform	8.10×10^{-2}	B2
Chromium VI	4.10×10^{1}	A
Methylene Chloride	1.65×10^{-3}	B2
Dibromoethane, 1,2-	7.60×10^{-1} [b]	B2
Dichloroethane, 1,2-	9.10×10^{-2}	B2
Perchloroethene	3.30×10^{-3}	B2
Perchloroethane, 1,1,2,2-	2.00×10^{-1}	C
Trichloroethane, 1,1,2-	5.70×10^{-2}	C
Trichloroethene	1.70×10^{-2}	B2

[a] U.S. EPA, 1989.

[b] Integrated Risk Information System, 1988.

[c] CAPCOA, 1989.

[d] U.S. EPA weight-of-evidence classifications:
A - Human Carcinogen (Sufficient Human Evidence)
B - Probable Human Carcinogen
B1 (Limited Human Evidence)
B2 (Sufficient Animal Evidence and Inadequate Human Evidence)
C - Possible Human Carcinogen (Limited Animal Evidence and Inadequate Human Evidence)

TABLE 9-3. CANCER POTENCY FOR THE PLAUSIBLE CASE[a]

Compound	Inhalation $(mg/kg/day)^{-1}$	Weight of Evidence
Benzene	2.90×10^{-2}	A
Chloroform	8.10×10^{-2}	B2
Chromium VI	4.10×10^{1}	A
Dibromoethane, 1,2-	2.40×10^{-1} [c]	B2
Dichloroethane, 1,2-	9.10×10^{-2}	B2
Methylene Chloride	1.65×10^{-3}	B2
Perchloroethene	3.30×10^{-3}	B2
Trichloroethene	1.70×10^{-2}	B2

[a] U.S. EPA, 1989

[b] U.S. EPA, 1988

[c] CAPCOA, 1989.

[d] U.S. EPA weight-of-evidence classifications:
A - Human Carcinogen (Sufficient Human Evidence)
B - Probable Human Carcinogen
B1 (Limited Human Evidence)
B2 (Sufficient Animal Evidence and Inadequate Human Evidence)
C - Possible Human Carcinogen (Limited Animal Evidence and Inadequate Human Evidence)

on the conclusions of EPA, this risk assessment has assumed that chromium presents no appreciable risk by ingestion.

For additional information on CPFs, refer to Appendix A.

9.3 Reference Doses

The RfDs used in this assessment were obtained primarily from the EPA's Integrated Risk Information System (IRIS) and from the EPA Health Effects Assessment Summary Tables, Third Quarter Update, and are shown in Table 9-4.

Some of the compounds included in the risk assessment have formally established CPFs but have no formal RfDs. For these compounds, an informal RfD was not developed in the risk assessment because carcinogenicity is a much more sensitive toxic endpoint than are noncarcinogenic effects from chronic exposures.

For those compounds for which informal RfDs were developed, one of several methods was used depending on the toxicological data available. If adequate toxicology data was available in the literature, an informal RfD was developed from the most appropriate data. If a threshold limit value (TLV®) time-weighted average was available for a compound, an informal RfD was developed from that value if the basis for the TLV® was appropriate. A TLV® is defined as the "concentration for a normal eight-hour workweek, to which nearly all workers may be repeatedly exposed, day after day, without adverse effects" (ACGIH, 1989).

For purposes of evaluating chronic noncancer effects from acid gases, hydrogen fluoride and nitric acid will be combined with emissions of hydrogen chloride and evaluated on the basis of hydrogen chloride toxicity.

For additional information on RfDs, refer to Appendix A.

TABLE 9-4. REFERENCE DOSES[a]

Chemical	Inhalation mg/kg/day
Ammonia	1.03×10^{-1}
Butanol, n-	1.02×10^{-1} [b]
Hydrogen Peroxide	1.02×10^{-3} [b]
Methylene Chloride	8.60×10^{-1}
Hydrochloric Acid	4.29×10^{-3} [c]
Tetraethylsilicate (Ethyl Silicate)	5.78×10^{-2} [b]

[a] Reference dose from U.S. EPA, 1989, unless otherwise noted.

[b] Developed from a TLV®.

[c] U.S. EPA, 1988.

10.0 RADIONUCLIDE RISK ASSESSMENT - METHODOLOGY

Cancer risks associated with routine emissions of radionuclides have been estimated using the AIRDOS and DARTAB computer models. The AIRDOS computer model is a methodology that estimates radionuclide concentrations in air; rates of deposition on ground surfaces; ground surface concentrations; intake rates via inhalation of air and ingestion of meat, milk, and fresh vegetables; and radiation doses to humans from airborne releases of radionuclides. The DARTAB computer model combines radionuclide environmental exposure data with dosimetric and health effects data to generate tabulations of the predicted impact of airborne radioactive pollutants.

The DARTAB computer code provides tabulations of predicted impacts of radioactive airborne effluents by combining information on environmental concentrations (obtained from AIRDOS) with dosimetric and health effects data obtained from a database called RADRISK (developed by the EPA Office of Radiation Programs). These data are used to estimate cancer risks and risks of genetic effects. The interaction of these computer codes is illustrated in Figure 10-1.

Both models were developed at the Oak Ridge National Laboratory (ORNL) to be used by the U.S. Environmental Protection Agency (EPA) as a methodology to evaluate health risks to humans from atmospheric radionuclide releases. This section provides brief descriptions of these models, the input parameters to AIRDOS for estimating radionuclide exposure, and a summary of the results from these models. Detailed descriptions of these models are provided in the user's manuals. The AIRDOS/DARTAB outputs are included in Appendix E.

10.1 AIRDOS Exposure Model Description

AIRDOS consists of two components: an air dispersion model and a terrestrial fate/human exposure assessment model. This section discusses the exposure assessment

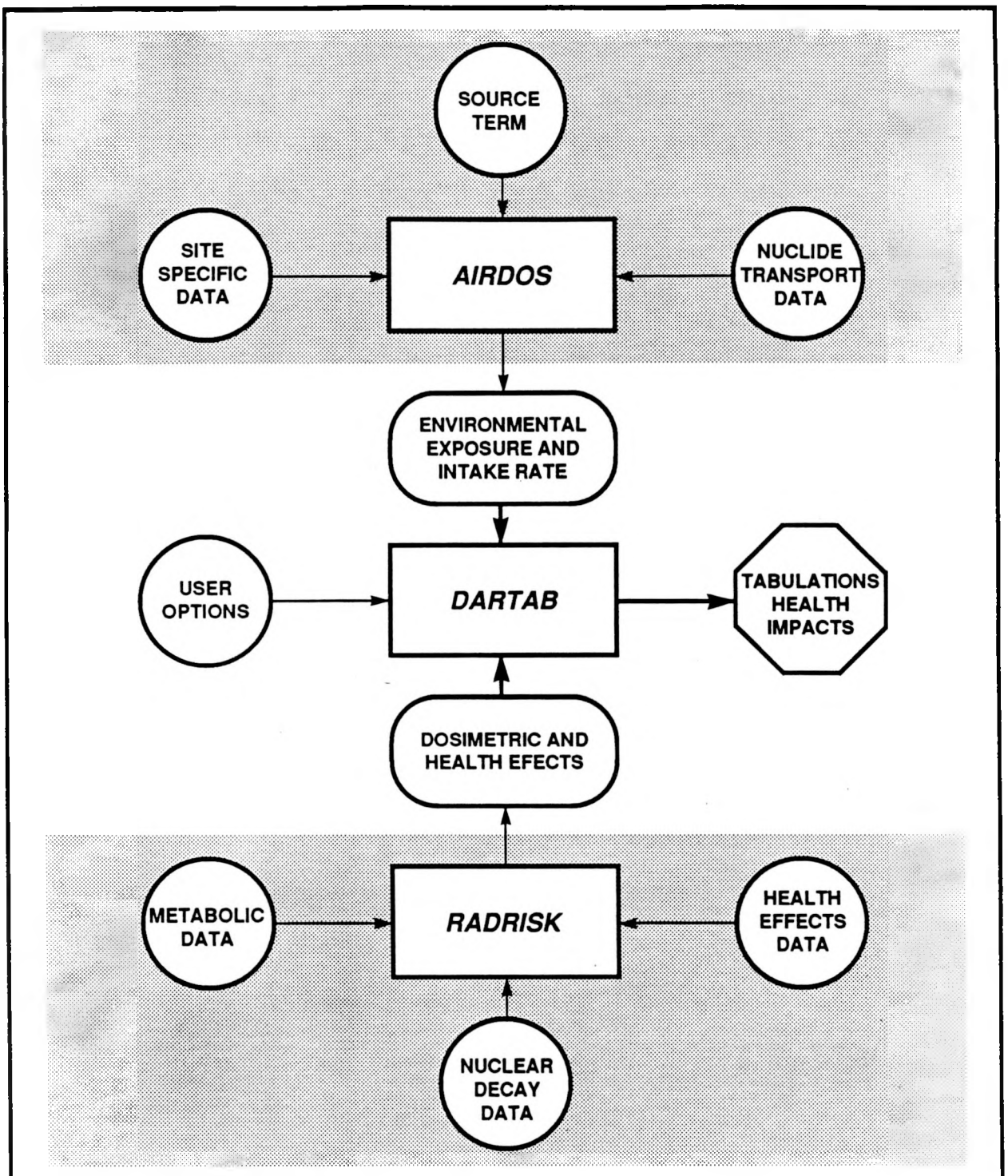

Figure 10-1. Interaction of Computer Codes for Radiological Health Impact Assessment.

part of AIRDOS. The dispersion model was discussed in Section 6.2, Dispersion Modeling Methods for Radionuclide Emission Sources.

Ingestion and inhalation exposure pathways represent internal doses (i.e., taken into the body), while air immersion and ground-surface exposure pathways represent external doses (doses to the skin). External doses do not contribute significantly to estimated health risks (U.S. EPA, 1984). Inhalation doses are calculated using concentrations in air and inhalation rates for adults; this method is similar to the inhalation exposure method used for the nonradioactive chemicals. For terrestrial environmental fate and ingestion exposure assessment, general methods specified by the U.S. Nuclear Regulatory Commission's (NRC) Regulatory Guide 1.109 were used. This general method also was adopted for assessing exposure to nonradioactive chemicals. The methods for estimating ingestion exposures for both radionuclides and nonradioactive chemicals are essentially the same.

The environmental fate pathways considered in AIRDOS are presented in Figure 10-2. Initially, the radionuclide source releases the materials as particulates or gases, forming a plume that disperses downwind. Concentrations of these radionuclides can potentially affect humans in two ways, either through external doses caused by photons emitted from airborne radionuclides, or internal doses through the inhalation of radionuclides. As described previously, risks from external doses are an insignificant proportion of the risks from all radionuclide exposure (U.S. EPA, 1984). Finally, small fractions of radionuclide particulates become deposited onto plant surfaces and the soil; these enter the foodchain through crops, meat, and milk. Consumption of these food items contributes to internal doses.

Radionuclide concentrations in meat, milk, and fresh produce are estimated by coupling the deposition rate output of the atmospheric dispersion models with the Regulatory Guide 1.109 (U.S. NRC, 1977) terrestrial food chain models. Radionuclide concentrations for specified distances and directions are calculated for the following exposure pathways:

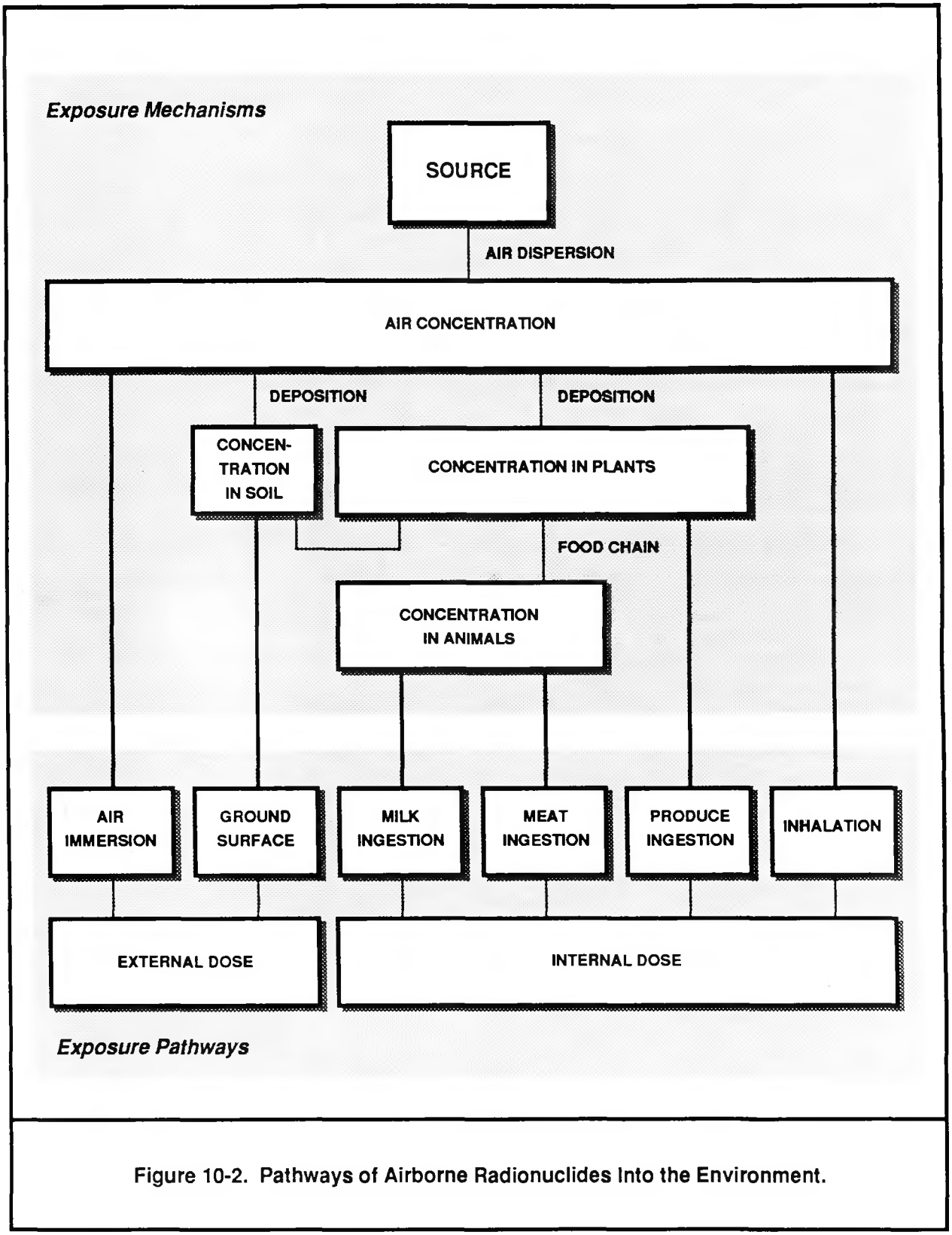

Figure 10-2. Pathways of Airborne Radionuclides Into the Environment.

- Immersion in air containing radionuclides;
- Exposure to ground surfaces contaminated by deposited radionuclides;
- Inhalation of radionuclides in air; and
- Ingestion of food grown or produced in the area.

The code may be used to calculate either annual individual exposures or annual population exposures at each grid location. For either option, AIRDOS-EPA output tables summarize air concentrations and surface deposition rates as well as the intakes and exposures for each location.

An AIRDOS-EPA assessment is based on a snap-shot view of environmental concentrations after the proposed facility has been operating for some period of time. The code uses release rates expressed in units of curies per year (Ci/year). It estimates the annual average concentration in picocuries per cubic centimeter (pCi/cm^3) of each radionuclide released as a function of distance and direction from the source. Annual-average frequencies of wind direction, wind speed and atmospheric stability categories are employed as input data (Moore et al., 1979). The operating lifetime of a facility generally is not considered in AIRDOS.

The only place where the operating lifetime of the facility is considered is the accumulation time used in estimating concentrations in soil and crops. The length of an environmental accumulation time affects only those pathways dependent on terrestrial concentrations, (i.e., ground surface exposure and food intakes). Usually, the accumulation time for an individual assessment is chosen to be consistent with the expected life of the facility (or 100 years when a similar facility might be expected to replace the present one at the end of its useful life). For the risk assessment, the facility life was assumed to be 70 years.

Generally, the procedures for estimating radionuclide concentrations in food are similar to the procedures described in the assessment for nonradioactive emissions (see Section 6.0). (The AIRDOS food chain modeling procedures were the original basis for food chain modeling in R-RAM®, the Radian Risk Assessment Model). Radionuclide

intake through the food chain depends on both the concentration in food and human use. The concentration in food depends on the food source. Use of foods grown in proximity to the source, the fraction of an individual's food that is home produced, and other factors can strongly influence the significance of exposure through the food pathway. When the EPA used AIRDOS to estimate health effects from radionuclide exposure, it concluded that risks from the ingestion pathway were one percent of the total risks (U.S. EPA, 1984).

Input values for the food chain model were obtained from the EPA Radionuclides Background Information Document for Final Rules (U.S. EPA, 1984). Since the Livermore area has some agricultural production, rural values for the fraction of food produced within the model grid and imported from outside the area were used. California values for cattle population density and vegetable crop fraction were selected. All other parameters were assigned default values. These site-specific and default parameters are presented in Table 10-1.

Special consideration was given to tritium or ^{3}H. The activity of ^{3}H is assumed to be proportional to the humidity in air. Concentration of this nuclide in vegetation was calculated assuming that the water content of the vegetation has the same activity as in the atmosphere.

10.2 DARTAB Model Description

DARTAB integrates the results from AIRDOS with dosimetric and health effects data obtained from the RADRISK database to provide estimates of health risks to humans (Begovich et al., 1981). The dosimetric data includes consideration of internal exposure resulting from ingestion and inhalation of radionuclides, as well as external exposure from photons emitted by radionuclides (i.e., air immersion and emissions from surface-deposited radionuclides). Dose values provided by DARTAB are in units of radiation-absorbed-dose (RAD), specific for each organ. The RAD is a measurement of

TABLE 10-1. SITE-SPECIFIC DEFAULT PARAMETERS USED FOR AIRDOS-EPA

Symbolic Variable	Description	Value
BRTHRT	Breathing rate (cm^3/hr)	9.17×10^5
T	Surface buildup time (days)	3.65×10^4
DDI	Activity fraction after washing	0.5
TSUBH1	Time delay - pasture grass (hr)	0
TSUBH2	Time delay - stored food (hr)	2.16×10^3
TSUBH3	Time delay - leafy vegetables (hr)	336
TSUBH4	Time delay - produce (hr)	336
LAMW	Weathering removal rate factor (hr^{-1})	2.10×10^{-3}
TSUBE1	Exposure period - pasture (hr)	720
TSUBE2	Exposure period - crops or leafy vegetables (hr)	1.44×10^3
YSUBV1	Productivity - pasture (dry weight) kg/m^2	.280
YSUBV2	Productivity - crops and leafy vegetables kg/m^2	.716
FSUBP	Time fraction - pasture grazing	0.40
FSUBS	Pasture feed fraction - while pasture grazing	0.43
QSUBF	Feed or forage consumption rate (kg-dry/day)	15.6
TSUBF	Consumption delay time - milk (day)	2.0
UV	Vegetable utilization rate (kg/yr)	176
UM	Milk utilization rate (kg/yr)	112
UF	Meat utilization rate (kg/yr)	85
UL	Leafy vegetable utilization rate (kg/yr)	18
TSUBS	Consumption time delay - meat (days)	20

(Continued)

TABLE 10-1. (Continued)

Symbolic Variable	Description	Value
FSUBG	Produce fraction (garden of interest)	1.0
FSUBL	Leafy vegetable fraction (garden of interest)	1.0
TSUBB	Soil buildup time (yr)	100
P	Effective surface density of soil ($kg/^2$)	215
TAUBEF	Meat herd - slaughter rate factor (d^{-1})	3.81×10^{-3}
MSUBB	Mass of meat of slaughter (kg)	200
VSUBM	Milk production rate of cow (L/day)	11.0
R1	Deposition interception fraction - pasture	0.57
R2	Deposition interception fraction - leafy vegetables	0.20

Source: U.S. EPA, 1984a.

energy deposition in any organ by all types of ionizing radiation. One RAD is equal to 0.01 joule per kilogram (J/kg) (Hobbs and McClellan, 1986). Estimated cancer risks and genetic effects are quantified on the basis of RADS.

DARTAB also presents absorbed dose values separately for both low- and high-linear energy transfer (LET) radiation associated with the decay of each nuclide. The LET is the rate at which charged particles transfer their energies to the atoms in an organ, and is a function of the energy and velocity of the charged particles (Hobbs and McClellan, 1986). The LET classification in DARTAB considers alpha particles and recoil nuclei from alpha particles to be high-LET, while beta and gamma particles are considered to be low-LET.

The dosimetric database also provides information for estimating organ doses using the environmental data provided by AIRDOS. For example, dosimetric data for inhaled aerosols considers activity median aerodynamic diameter (AMAD) and lung clearance classes; these data have been developed by the International Commission on Radiological Protection. Absorbed doses from ingestion exposures are characterized in terms of GI-tract absorption factors specific for the physicochemical forms for the nuclide. The dose rate units per unit intake are expressed in units of millirad per year per picocurie per year (mrad/year per pCi/year). This dose rate is combined with the health effects database to provide estimates of cancer risks.

DARTAB assesses health risks associated with low-level chronic exposure (i.e., lifetime exposure) to radionuclides (Begovich et al., 1981). The DARTAB methodology is also used to estimate fatal cancer risk associated with chronic, routine releases of radionuclides. Risk estimates are not associated with annual emissions; rather, DARTAB assumes that the annual concentrations obtained from AIRDOS represent a lifetime (70 year) event (Sjoreen, 1988; Parks, 1988).

10.3 RADRISK Model Description

Dosimetric and risk factors in DARTAB are generated by the RADRISK code (Begovich et al., 1981). RADRISK is described as a methodology designed to yield estimates of health effects assuming constant, lifetime exposure to a given radionuclide. The model selects a theoretical group or cohort of 100,000 persons all simultaneously live-born and assumes that each member continuously inhales or ingests, over an entire lifetime, 1 pCi/year of a given parent radionuclide. The health effects factors are evaluated as the number of incremental deaths within the cohort (Dunning et al., 1980). The environmental concentrations developed from AIRDOS are converted to exposures by DARTAB, and are compared with the health effects estimates per unit exposure to estimate fatal cancer risk. The risk factors are expressed as effects/10^5 per pCi/year ingested or inhaled.

RADRISK uses a life table methodology for estimating the incremental risk of cancer from exposure to radiation. A life table is essentially a table of data describing age-specific mortality rates from all causes of death for a given population. The life table in RADRISK is based on mortality rates for the U.S. Population from 1969-1971 as reported by the National Center for Health Statistics. The life table can be used to estimate the number of individuals who will die from radiation-induced cancer, excluding those who might have died from radiation-induced cancer, but actually die from other causes. The risk factors for radiation-induced cancer are obtained from the Biological Effects of Ionizing Radiation (BEIR) 1980 report developed by the National Academy of Sciences (Dunning et al., 1980). The life table methodology assumes that no individual lives longer than 110 years, however, the mean lifetime of the cohort is 70.7 years, a result obtained from the age-specific mortality data (U.S. EPA, 1984). Hence, a typical lifetime exposure for the AIRDOS/DARTAB methodology is about 70 years.

The radiological risk assessment method is then comparable to the cancer risk assessment method for chemical carcinogens. The cancer potency slopes (CPFs) used in the health risk assessment represent the increased individual lifetime risk for a 70-kilogram individual exposed for a 70-year lifespan (Anderson et al., 1983).

10.4 Conversion of Fatal Cancer Risk to Cancer Incidence

Fatal cancer risk from radionuclide exposure is typically estimated from wide experience with human exposure to radioactive compounds, including survivors of nuclear warfare. These data do not correspond to the estimated CPFs developed for nonradioactive compounds. A procedure for correcting these data to correspond with the nonradioactive risk estimate (i.e., cancer incidence) is based on a survey comparing the probability at birth of developing cancer with the probability of eventually dying from cancer (Seidman et al., 1985).

A summary of results from this survey is presented in Tables 10-2 and 10-3. Fatal cancer risks were corrected to reflect risks of developing cancer by multiplying the risks obtained from AIRDOS/DARTAB by a ratio as follows:

$$\frac{\text{Probability of developing cancer}}{\text{Probability of dying from cancer}}$$

These ratios are based on an average of the probability for all cancers for males and females for the year 1985. The ratio for males is 1.59, while the ratio for females is 1.81, yielding an average of 1.70.

TABLE 10-2. PROBABILITY AT BIRTH OF EVENTUALLY DEVELOPING CANCER IN THE U.S.

Site	White Males			White Females		
	1975	1980	1985	1975	1980	1985
All cancer	.303	.336	.369	.339	.350	.361
Buccal cavity and pharynx	.015	.016	.016	.007	.008	.009
Esophagus	.005	.005	.005	.002	.002	.003
Stomach	.012	.012	.012	.009	.008	.008
Colon/rectum	.053	.059	.065	.058	.064	.069
Pancreas	.012	.012	.012	.011	.012	.013
Larynx	.088	.008	.009	.001	.002	.002
Lung	.069	.078	.087	.025	.033	.042
Breast				.096	.099	.102
Uterus				.070	.060	.050
Cervix				.037	.032	.027
Ovary				.015	.015	.015
Prostate	.061	.074	.087			
Testis	.002	.003	.087			
Kidney	.008	.011	.013	.005	.006	.007
Bladder	.025	.029	.032	.010	.011	.012
Melanoma	.006	.009	.013	.006	.009	.011
Thyroid	.002	.002	.002	.005	.005	.005
Leukemia	.012	.012	.012	.010	.009	.009
Lymphoma and multiple myeloma	.016	.018	.002	.016	.018	.020

Source: Seidman et al., 1985.

TABLE 10-3. PROBABILITY AT BIRTH OF EVENTUALLY DYING OF CANCER IN THE U.S.

Site	White Males			White Females		
	1975	1980	1985	1975	1980	1985
All cancer	.189	.210	.232	.171	.186	.200
Buccal cavity and pharynx	.005	.005	.005	.002	.003	.003
Esophagus	.004	.004	.005	.002	.002	.002
Stomach	.008	.008	.007	.006	.006	.006
Colon/rectum	.024	.027	.029	.029	.030	.031
Pancreas	.010	.010	.011	.009	.011	.012
Larynx	.003	.003	.003	.000	.001	.001
Lung	.058	.068	.078	.018	.026	.034
Breast				.031	.034	.036
Uterus				.010	.009	.009
Ovary				.009	.010	.012
Prostate	.020	.023	.026			
Testis	.001	.000	.000			
Kidney	.004	.005	.005	.003	.003	.003
Bladder	.007	.007	.008	.003	.004	.004
Melanoma	.002	.003	.003	.002	.002	.002
Thyroid	.000	.000	.000	.001	.001	.001
Leukemia	.008	.009	.010	.007	.007	.008
Lymphoma and multiple myeloma	.010	.010	.010	.010	.010	.010

Source: Seidman et al., 1985.

11.0 RISK CHARACTERIZATION

The purpose of risk characterization is to present quantitative estimates of potential health risk using the results of the exposure and dose-response assessments. Two health effects were considered: the risk of developing cancer and the potential for systemic toxicity from exposure to noncarcinogenic substances.

A quantitative estimate of health risk is necessarily based on numerous assumptions and data inputs. For each assumption, there may be several plausible alternative values that represent environmental conditions (e.g., the amounts of vegetables consumed by an individual, the rate of degradation of a compound in the soil, carcinogenic potency of a compound, etc.). The variability in possible values for each assumption leads to some uncertainty in the numerical estimates of risk, and the general use of conservative worst-case assumptions throughout the risk assessment results in a compounding of that uncertainty.

In this health risk assessment, uncertainties in the numerical estimates of risk for nonradioactive exposures were addressed by developing two primary exposure scenarios that provide a worst case and a plausible estimate of potential risk. The risk estimate for the plausible case is still believed to be a conservative estimate of actual risk. Due to the complexity of evaluating exposure to radionuclides, risk estimates are presented for a single case based on U.S. Environmental Protection Agency (EPA) default values for exposure assumptions.

11.1 Health Risks for Nonradioactive Emissions

Numerical evaluations of health risks were performed separately for carcinogenic and noncarcinogenic effects. Carcinogenic risk evaluation involved estimating individual cancer risks and cancer burden. Evaluation of noncarcinogenic effects involved direct comparison of pollutant exposure levels with reference doses (RfDs) or other health effects data.

Individual lifetime cancer risk is the probability of an individual developing cancer under specified conditions of lifetime exposure. It is estimated by summing the cancer risks for all carcinogenic pollutants through all potential exposure pathways. U.S. EPA guidelines for carcinogen risk assessment use the assumption that cancer risks are additive from exposure to multiple carcinogens (U.S. EPA, 1986b). In this health risk assessment, individual cancer risks were estimated using carcinogenic potency factors (CPFs) in inverse units of milligrams per kilogram per day $(mg/kg\text{-}day)^{-1}$ and chemical intake rates in units of mg/kg-day. This may be expressed as follows:

$$Risk_i = Exposure_i \text{ (mg/kg-day)} \times CPF_i \text{ (kg-day/mg)}$$

Separate CPFs for ingestion and inhalation exposures were not available for all pollutants primarily because of a lack of data or the absence of a positive carcinogenic response for a particular exposure route. In the worst case, and per California Department of Health Services (DHS) requirements, the available CPF was used regardless of exposure route (CAPCOA, 1987). In the plausible case, the CPF was assumed to be zero if no carcinogenic response has been noted for a given pathway.

Cancer risk was estimated for a hypothetical maximally-exposed individual (MEI) and a series of population receptors. The MEI is defined as that receptor with the highest risk giving appropriate consideration to its geographical location. For example, for receptors located in areas zoned for commercial or industrial business, the exposure period would be limited to nine hours per day, five days per week, for a total period of 40 years. Over a lifetime, this is equivalent to a continuous exposure of 10.7 years.

The point of maximum off-site exposure to LLNL waste treatment and storage emissions occurs at the eastern boundary of the Lawrence Livermore National Laboratory (LLNL) facility approximately 600 meters north of East Avenue along Greenville Road. Land use in this area is rural and current zoning does not restrict residential dwellings. Therefore, this receptor does not require any adjustment for reduced exposure duration. The worst-case risk to this hypothetical MEI, assuming continuous exposure for a 70-year lifetime, was calculated to be 8.3×10^{-8} (8.3 in one hundred million) based on DHS-

approved cancer potencies, and 7.6 x 10^{-8} (7.6 in one hundred million) assuming all EPA potency values. The risk to the MEI under the plausible scenario was estimated to be 8.9 x 10^{-9} (8.9 in one billion). A breakdown of risk by pollutant and by pathway is shown in Tables 11-1 and 11-2 for the two worst-case scenarios, and Table 11-3 for the plausible case.

When interpreting these risk results, it is important to remember that these are theoretical risk estimates based on a conservative framework. For those chemicals evaluated, the actual risks are expected to be lower than presented here, and may be as low as zero (U.S. EPA, 1986b).

The California Air Pollution Control Officers (CAPCOA) guidelines require cancer burden to be calculated within a 10^{-7} cancer risk isopleth. Since the risk to the MEI is less than 1 x 10^{-7} for all off-site areas, a cancer burden calculation is not required.

11.2 Noncarcinogenic Effects for Nonradioactive Emissions

The potential for noncarcinogenic effects from exposure to nonradioactive emissions were evaluated for both chronic and acute exposures using appropriate exposure and health effects criteria discussed and presented in Section 9.0, Dose-Response for Nonradionuclides.

Chronic effects were evaluated by comparing the estimated inhalation or total ingestion exposure for each pollutant to formal or informal RfDs (see Section 9.3). This analysis involves dividing the RfD by the estimated exposure to calculate a hazard index HI). The presence of a HI less than 1.0 suggests that a noncancer effect from that pollutant is unlikely. A HI value was estimated only for those compounds that are not considered to be human carcinogens. Cancer risk, as demonstrated by numerous risk assessments, is a more sensitive indicator of adverse health effects for chronic exposure.

TABLE 11-1. 70-YEAR LIFETIME CANCER RISK FOR THE MEI WORST-CASE SCENARIO WITH CAPCOA POTENCIES

Pollutant	Pathway						
	Inhalation	Soil	Plant	Dermal	Water	Wine	Total
Benzene	4.3×10^{-9}						4.3×10^{-9}
Methylene Chloride	5.3×10^{-9}						5.3×10^{-9}
Chloroform	2.0×10^{-9}						2.0×10^{-9}
Hexavalent Chromium	3.7×10^{-10}	5.4×10^{-10}	1.6×10^{-10}	1.1×10^{-9}	3.0×10^{-12}	1.4×10^{-11}	2.2×10^{-9}
Dibromoethane, 1,2-	1.6×10^{-10}						1.6×10^{-10}
Dichloroethane, 1,2-	5.9×10^{-9}						5.9×10^{-9}
Perchloroethane, 1,1,2,2-	1.3×10^{-8}						1.3×10^{-8}
Perchloroethene	4.1×10^{-8}						4.1×10^{-8}
Trichloroethane, 1,1,2-	3.3×10^{-10}						3.3×10^{-10}
Trichloroethene	8.0×10^{-9}						8.0×10^{-9}
Total	8.1×10^{-8}	5.4×10^{-10}	1.6×10^{-10}	1.1×10^{-9}	3.0×10^{-12}	1.4×10^{-11}	8.3×10^{-8}

TABLE 11-2. 70-YEAR LIFETIME CANCER RISK FOR THE MEI WORST-CASE SCENARIO WITH EPA POTENCIES

Pollutant	Pathway						
	Inhalation	Soil	Plant	Dermal	Water	Wine	Total
Benzene	7.4×10^{-10}						7.4×10^{-10}
Methylene Chloride	2.6×10^{-9}						2.6×10^{-9}
Chloroform	2.0×10^{-9}						2.0×10^{-9}
Hexavalent Chromium	2.9×10^{-11}	4.3×10^{-11}	1.6×10^{-11}	9.0×10^{-11}	2.4×10^{-13}	1.4×10^{-11}	1.9×10^{-10}
Dibromoethane, 1,2-	5.1×10^{-10}						5.1×10^{-10}
Dichloroethane, 1,2-	7.3×10^{-9}						7.3×10^{-9}
Perchloroethane, 1,1,2,2-	1.3×10^{-8}						1.3×10^{-8}
Perchloroethene	4.1×10^{-8}						4.1×10^{-8}
Trichloroethane, 1,1,2-	3.3×10^{-10}						3.3×10^{-10}
Trichloroethene	8.0×10^{-9}						8.0×10^{-9}
Total	7.7×10^{-8}	4.3×10^{-11}	1.6×10^{-11}	9.0×10^{-11}	2.4×10^{-13}	1.4×10^{-11}	7.6×10^{-8}

TABLE 11-3. 10-YEAR LIFETIME CANCER RISK FOR THE MEI PLAUSIBLE SCENARIO

Pollutant	Inhalation
Benzene	1.0×10^{-10}
Methylene Chloride	3.8×10^{-10}
Chloroform	2.8×10^{-10}
Hexavalent Chromium	4.2×10^{-12}
Dibromoethane, 1,2-	2.3×10^{-11}
Dichloroethane, 1,2-	1.0×10^{-9}
Perchloroethene	5.9×10^{-9}
Trichloroethane, 1,1,2-	4.6×10^{-11}
Trichloroethene	1.1×10^{-9}
Total	8.9×10^{-9}

The absence of a significant risk for a carcinogen will also signify the absence of a significant noncancer risk. It should be noted that if an RfD was available for a carcinogen, that compound was considered in evaluating the potential for noncancer effects as well as the cancer endpoint.

Many of the pollutants considered in this risk assessment have the same or similar toxic end points. Therefore, the potential for additive effects on an organ (e.g., liver, kidney, lung) or system [e.g., central nervous system (CNS), skeletal] may exist. While the exposure for any given pollutant may be less than its respective RfD, the summation of those pollutants acting at similar sites in the body may be sufficient to exceed a threshold. To examine this potential, a cumulative HI was calculated for similar toxic end points.

An organ-specific HI is simply the sum of the exposure-to-RfD ratio for each pollutant having the same or similar health effect. It is expressed mathematically as:

$$HI = \sum_{i=1}^{N} \text{Exposure}_i/\text{RfD}_i$$

Where: N is the total number of pollutants within a health effect category, and i refers to a specific pollutant.

If the HI is less than 1.0, then adverse effects are not likely for that organ or system. If the HI is greater than 1.0, this does not imply that an adverse effect will occur, but that a more detailed analysis may be needed. Table 11-4 presents the worst-case organ-specific hazard index values for chronic health effects. As seen from the results, all organs or systems have a HI less than one. This indicates that adverse effects are not likely under the worst-case set of assumptions. The plausible case was not specifically evaluated, but would have HI values less than shown in Table 11-4.

TABLE 11-4. CHRONIC HAZARD INDEX FOR TARGET ORGANS*

	Inhalation			Ingestion		
Target Organ	Chemical	Chemical Specific Hazard Index	Organ Specific Hazard Index	Chemical	Chemical Specific Hazard Index	Organ Specific Hazard Index
Respiratory Tract	Ammonia	3.6×10^{-4}				
	Hydrochloric Acid	2.7×10^{-3}				
	Hydrogen Peroxide	7.0×10^{-4}				
	Tetraethyl Silicate	5.4×10^{-9}	3.8×10^{-3}			
Liver	Methylene Chloride	1.9×10^{-6}				
	Tetraethyl Silicate	5.4×10^{-9}	1.9×10^{-6}			
Eye	Butanol, n-	1.4×10^{-6}	1.4×10^{-6}			
Kidney	Tetraethyl Silicate	5.4×10^{-9}	5.4×10^{-9}			
Ear	Butanol, n-	1.4×10^{-6}	1.4×10^{-6}			
None Specified				Chromium VI	3.2×10^{-9}	3.2×10^{-9}

* Hazard Index calculated for noncarcinogens and carcinogens with established RfDs for relevant pathways. Hazard Index based on DHS worst case.

11.3 Acute Effects

The potential for acute health effects from normal hazardous waste treatment and storage operations was evaluated by comparing estimated maximum one-hour concentrations in air to acute health effects criteria. Using a two-step process, the one-hour concentration was first compared to the Threshold Limit Value/10 (TLV®/10) as an initial screening. This was done for all chemicals except hydrogen choride. For hydrogen chloride, an EPA-recommended value of 150 was used as the basis for evaluating acute effects potential (U.S. EPA, 1988). The purpose of this first step is to screen the one-hour exposures and eliminate from any further analysis pollutants with no potential for adverse effects. TLV®s have been developed by the American Conference of Governmental Industrial Hygienists (ACGIH) for purposes of regulating occupational exposures in the work place. They represent ambient work place concentrations to which an employee can be exposed for up to 8 hours (time weighted) per day for a 40-year working life without significant risk of adverse health effects. The ACGIH does not recommend the use of TLV®s for protecting the health of the general public. As applied in this risk assessment, the TLV®s serve only as an initial indicator of the potential for adverse effects. Further, a safety factor of 10 has been applied to the TLV® to account for lower thresholds of sensitive individuals.

To evaluate the potential for acute effects, a modeling post processor was used to identify the worst one-hour hazard index for each substance over the three-year meteorological data set. The acute post processor calculates a hazard index for each substance based on a ratio of the modeled concentration and the TLV®/10. This calculation is performed at each receptor for every hour of the three-year meteorological data set. It then scans these results and identifies the single hour with the highest hazard index for all substances combined. Based on the processor results, the highest hazard index value was 0.17. The acute modeling post processor output can be found in Appendix G.

11.4 Evaluation of Radionuclide Emissions

The cancer risks associated with radionuclide emissions from the LLNL waste treatment and storage facilities were assessed for normal operations. Section 10.0 of this report provides a detailed discussion of the methods. From the AIRDOS-DARTAB model, the worst-case risk to the MEI was estimated to be 1.1×10^{-8} (1.1 in one hundred million). This estimate includes the 1.7 factor for converting fatal risk to the risk of developing, but not necessarily dying from, cancer. The AIRDOS/DARTAB model assumes a lifetime exposure; therefore, no plausible case could be evaluated.

Cancer burden estimates were not required for radionuclide emissions. As in the case of nonradionuclide emissions, the cancer risk from radionuclide emissions was determined to be less than 1×10^{-7}. Determination of the cancer burden, therefore, is not required.

In addition to the radionuclide exposure pathways discussed in Section 10.0, the potential risk of cancer from ingestion of locally produced wine containing tritium emitted from the LLNL waste treatment and storage facilities was assessed. Based on monitored tritium levels in Livermore Valley wines, an exposure rate in microcuries per day was estimated. Using a conversion factor of 63 rems per curie (ICRP, 1979), the lifetime committed dose of tritium from ingestion of locally produced wines was estimated to be 1.04×10^{-5} millirems under the worst-case and 1.5×10^{-6} millirems under the plausible case. These dose rates result in an estimated lifetime risk of developing cancer of approximately three in one trillion (3×10^{-12}) and 4 in ten trillion (4×10^{-13}), respectively. A conversion factor of 1.6×10^{-4} deaths per rem was used to convert from dose to risk (Federal Register, 1986). This analysis has conservatively assumed that all tritium measured in the Livermore Valley wines originated from LLNL. In actuality, a portion of the measured tritium may have resulted from natural or other man-made sources. Supporting calculations can be found in Appendix C.

11.5 Conclusions

In conjunction with Resource Conservation and Recovery Act (RCRA) and the California Hazardous Waste Control Act, permitting activities for the LLNL hazardous waste treatment and storage facilities, emissions were evaluated for potential adverse health effects on the exposed human population. Risk to the population from exposure to potentially carcinogenic or toxic emissions was evaluated by employing a series of conservative exposure assumptions that tend to overestimate the total dose received.

The cancer risk estimates and population cancer burden values were based on two exposure scenarios that defined plausible and worst-case risks. The worst-case risk of developing cancer based on DHS potencies was 8.3 in one hundred million (8.3×10^{-8}). When all EPA potencies were used, the worst-case risk was estimated to be 7.6 in one hundred million (7.6×10^{-8}). The plausible risk (also based on EPA potencies) was estimated to be 8.9 in one billion (8.9×10^{-9}). The risk from exposure to radionuclides was determined to be 1.1 in one hundred million. Table 11-5 summarizes the total cancer risk and burden estimates combining impacts from nonradioactive and radioactive emission sources. Under the DHS worst-case, the total risk (combining radionuclide and nonradionuclide risk) was estimated to be 9.4 in one hundred million (9.4×10^{-8}).

Cancer risk and burden estimates presented in this document are based on total dose received from all possible exposure routes. These cancer risks do not reflect, in any manner, actual risks to the exposed population in the vicinity of the facility. Rather, they are estimates of the maximum risk using conservative assumptions within the framework of accepted health risk assessment methods. For these substances, the risk estimates calculated for the LLNL emissions are not likely to be higher and could be zero (U.S. EPA, 1986a).

TABLE 11-5. SUMMARY OF CANCER RISK

Scenario	Nonradioactive	Radionuclides[d]	Total Risk[e]
Worst-Case			
DHS Potencies[a]	8.3×10^{-8}	1.1×10^{-8}	9.4×10^{-8}
EPA Potencies[b]	7.6×10^{-8}	1.1×10^{-8}	8.7×10^{-8}
Plausible Case[c]	8.9×10^{-9}	1.1×10^{-8}	2.0×10^{-8}

[a] Cancer risk values based on DHS-approved potency values and worst-case exposure assumptions.

[b] Cancer risk values based on EPA cancer potency values and worst-case exposure assumptions.

[c] Risk developed from plausible exposure assumptions and EPA cancer potency factors.

[d] Exposure and risk from radionuclide emissions were determined for only one scenario. A single risk estimate is, therefore, presented for all cases.

[e] Values represent the maximum exposed individual's lifetime risk of developing some form of cancer due to hazardous waste storage and treatment facility emissions.

The risk of adverse health effects other than cancer were evaluated by comparing concentrations of contaminants with levels considered to be without adverse health effects. Even at the point of maximum concentration, estimated levels were below levels that would pose a risk of adverse effects.

There are numerous methods that can be used to quantify human exposure and evaluate the potential for adverse health effects. The methodology that has been employed in this Phase II risk assessment follows the general regulatory requirements outlined in the CAPCOA manual (CAPCOA, 1987) and intentionally results in a conservative estimate of risk. The methods should not, however, be considered a precedent for any subsequent analyses nor dictate the manner in which other studies or risk assessments are performed.

12.0 CHEMICAL ACCIDENT ANALYSIS

12.1 Scenario Development

The accident scenario is developed based on both the probability of the accident occurring and on the severity of the potential off-site acute effects resulting from such an accident. The accident that will be analyzed reflects a worst-case scenario accident using conservative assumptions, but which is still credible given the facility design, operation, and location. The accident scenario is limited to the equipment or processes directly related to the hazardous waste storage and treatment activities.

An analysis of the Lawrence Livermore National Laboratory (LLNL) hazardous waste treatment and storage operations was performed by Science Applications International Corporation (SAIC). Based on the SAIC preliminary analysis of the LLNL system, several potential accident scenarios were identified. The potential accident scenarios were then ranked by SAIC based on accident potential, emission rate, downwind impacts, and toxicity. The scenario consists of spilling a drum containing an aqueous solution of formaldehyde (37 percent formaldehyde), and the subsequent release of the formaldehyde to the atmosphere. This scenario was selected as the maximum credible accident to be examined in the risk assessment (SAIC, 1990).

The solvent drum rupture selected as the hypothetical catastrophic event consists of the sudden loss of six gallons of the stored liquid (approximately 37 percent formaldehyde in water). The spill is uncontained and occurs on an asphalt pad with a slight downward slope in Area 612. It is assumed the accident occurs during worst-case meteorological conditions with an ambient temperature of 100°F.

Typically when evaluating an accident scenario, and primarily when estimating cancer risk, it is necessary to incorporate into the calculations the probability of the maximum credible accident occurring. The formaldehyde spill identified by SAIC was

conservatively assumed to have a 100 percent probability of occurring over a 70 year lifetime. Therefore, probability will not be incorporated into the calculation of cancer risk.

12.2 Emission Estimates

The formaldehyde emissions estimates for the hypothetical accident were obtained from standard mass transfer equations. The mass transfer equations involve quantifying the liquid phase mass transfer, the liquid to gas phase transfer, and gas phase mass transfer. Details of these calculations are provided in Appendix H.

A fairly thin pool depth of 0.2 centimeters was assumed for this spill due to a slight grade at the potential spill site; therefore, a pool area of approximately 11 square meters would result from the six-gallon spill. Given the pool size and estimated depth, the mass emission rate of formaldehyde vapors was estimated to be 1.6 grams per second. It was assumed that this peak emission rate was achieved instantaneously and persisted throughout the spill duration. About two gallons of pure formaldehyde are contained in the liquid mixture. Depletion of the formaldehyde would occur within one hour based on the estimated emission rate.

By assuming a continuous emission rate over the spill duration, the effects of a shrinking emission source are conservatively neglected. When the spill occurs, the pool will grow in size allowing for a gradual increase in the exposed surface area for formaldehyde release. Similarly, as clean-up efforts commence or evaporation begins, the pool will gradually decrease in exposed surface area until all liquid has been contained or evaporated. The gradual increase and then decrease in pool size will result in emissions that are lower than the peak rate assumed for the spill duration.

12.3 Modeling Analysis

INPUFF, an episodic puff-type dispersion model developed by EPA, was used to simulate the transport and downwind concentration of formaldehyde. Estimated ambient

concentrations were obtained for two off-site receptors: the nearest public road and the nearest residence. In order to obtain worst-case results, the wind was assumed to blow directly toward each receptor. A constant wind direction was input into INPUFF and the concentrations were obtained directly downwind of the source at distances corresponding to each receptor. A one meter per second (m/s) wind speed was used along with "F" stability to simulate worst-case meteorological conditions. With a temperature of 100°F, and the presence of buildings and hot asphalt, it is unlikely that "F" stability and a one meter per second wind will occur. Building wake effects were considered in estimating the initial horizontal and vertical dispersion coefficients.

The modeling was performed to identify the maximum concentrations at each receptor resulting from the formaldehyde spill scenario. The maximum concentrations will occur from the emission rate during the first few minutes since the distance to each of the receptors is relatively short and travel time is minimal. Therefore, the modeling was performed using the maximum emission rate until steady state concentrations were obtained at each receptor. The steady state values were assumed to persist for the 60 minute duration. Once the spill has been cleaned up, the ambient concentrations will rapidly drop off.

Formaldehyde has a low odor threshold limit and would be easily detected by most individuals. To address a short duration exposure that might be associated with moving away from the plume or traveling through the plume, INPUFF was used to estimate the cross-wind concentration at the East Avenue receptor during peak concentration. A 10 meter grid spacing was used for estimating cross wind concentration values. From these data, it is also possible to define the plume width at the nearest receptor.

The pertinent parameters and options used with INPUFF are summarized in Table 12-1. Table 12-2 presents the modeling results obtained from INPUFF. The model output is included as Appendix H.

TABLE 12-1. DISPERSION MODEL INPUT FOR FORMALDEHYDE

Emissions

Continuous emissions with a constant emission rate
Emission rate = 1.6 grams/second

Release Parameters

Release height = 0.0 feet
Release temperature = 100°F
Spill area = 11 meters
Pressure = 1 atmosphere (ambient)
Nearest building = Building 612
Building height = 7 meters
Building width = 24 meters

Meteorological Conditions

Wind speed = 1 meter per second
Wind direction = not applicable (wind assumed directly at receptors)
Stability = "F"

Receptors

Nearest public road, 150 meters from source
Nearest residence, 350 meters from source

TABLE 12-2. DISPERSION MODEL RESULTS

Receptor	Formaldehyde Concentrations (ppm)			
	Center Line	10m[a]	20m[a]	30m[a]
Road (150 m)	14	5.9	0.46	0.0069
Residence (350 m)	4.1			

[a] Concentration at specified distance from the plume center line.

12.4 Exposure Assessment

Human exposure to accident emissions will be a function of downwind formaldehyde concentration, the duration of exposure, and the absorption of formaldehyde vapors in the lung. Based on the worst-case analysis of constant wind direction and speed, the nearest public road (East Avenue) will have the highest concentration, followed by the nearest residential location.

As a worst case, the East Avenue receptor will be evaluated assuming continuous exposure, even though it appears to be an unlikely scenario. This road serves both occupational traffic going to and from work and residential traffic. A prolonged exposure to accident emissions (up to one hour) is plausible but not likely. Formaldehyde has a low odor threshold. If an individual were exposed to even low ppm levels in the air, the odor would alert the individual to the presence of the chemical and prompt them to move away from the area. This may not be true for the nearest residence where mobility could potentially be restricted, and modeled levels are much lower than at East Avenue. Therefore, exposure at the East Avenue fenceline will be evaluated under a variety of conditions corresponding to the traffic profile for that street.

Formaldehyde exposure at East Avenue was evaluated under the following assumed scenarios:

- Scenario 1: An individual walks through the width of the plume on East Avenue, 150 meters from the source; and

- Scenario 2: As a worst case, an individual is exposed at the plume center line at the fenceline, 150 meters from the source, for the duration of the accident emissions.

Exposure at the nearest residence, 350 meters from the source, was evaluated assuming continuous exposure throughout the duration of the accident.

The INPUFF modeling provided estimates of the plume center line concentration and also cross-wind concentration estimates during the peak emission period at the East Avenue receptor. Only peak plume centerline concentrations were determined at the more distant residential location. Time-weighted exposure estimates were determined at each receptor for purposes of calculating cancer risk.

Exposure under Scenario 1 is calculated assuming an individual walks through the plume at a speed of 2 miles per hour. The INPUFF model predicts a plume width of 60 meters. Given the assumed speed of 2 mph, the pedestrian will require 67 seconds to move completely through the INPUFF plume. By dividing the gaussian-shaped concentration distribution into equal segments, a time-weighted concentration of 4.4 ppm was estimated from the INPUFF results. Exposure to this concentration occurs for only 67 seconds. To calculate cancer risk, it is necessary to estimate a prorated exposure over a lifetime that would result in the same intake rate for the 67 second period. This calculation is needed to be consistent with the assumptions used in developing the cancer potency factor. Prorated over a lifetime and converting to mass units, the weighted average concentration was found to be 1.6×10^{-4} $\mu g/m^3$.

Scenario 2 addresses potential exposure to a maximally exposed individual (MEI) who remains at each of the two receptors (East Avenue and nearest residence) throughout the spill and clean-up efforts (approximately 60 minutes). The prorated ambient concentrations at each of the receptors are summarized in Table 12-3.

12.5 Dose Response Assessment for Formaldehyde

Pharmacokinetics

Approximately 100 percent of formaldehyde (CH_20) is readily absorbed when inhaled or ingested. The skin is not a major route of absorption unless it comes in direct contact with liquid. Formaldehyde is water soluble enabling its distribution uniformly throughout the body within minutes of exposure to the compound. After distribution, levels

TABLE 12-3. PRORATED LIFETIME FORMALDEHYDE CONCENTRATIONS AT EAST AVENUE

Receptor	Modeled Lifetime Concentration [a] ($\mu g/m^3$)
East Avenue	
Pedestrian	1.6×10^{-4}
MEI [b]	2.8×10^{-2}
Nearest Residence [b]	8.3×10^{-3}

[a] Assumes continuous exposure for one hour.

[b] Concentration estimates represent prorated lifetime values, i.e., a lifetime exposure to the above values would yield an exposure equivalent to the 67-second or one-hour exposure during the accident.

decrease quickly with time, due to its rapid biotransformation and excretion or irreversible binding to molecules in the body. The major route of elimination for formaldehyde is exhalation of its biotransformation product, CO_2. A secondary route of elimination of formaldehyde from the body is in the urine as a transformation product either as formic acid or a conjugate of urea (US EPA, 1985).

Biotransformation

Formaldehyde is a natural product of the body. It is produced during the breakdown of the amino acids histidine, serine, glycine, and tryptophan, natural building blocks in the body. It is then utilized in the formation of new molecules. Therefore, a certain amount of formaldehyde is always present in the body. Formaldehyde, introduced to the body from an external source, is metabolized by oxidation to formic acid (CH_2O_2) and then to CO_2. Metabolism has been observed to occur in various tissues of the body. Metabolism of formaldehyde requires reduced glutathione and NAD, two molecules which allow the oxidation to take place.

Health Effects

Acute Toxicity (Short term exposure to high concentrations)

The lethal dose of formaldehyde for 50 percent of animal subjects ranges from 500-800 mg/kg of body weight. The lethal inhalation concentration for the rat is approximately 450-500 ppm (Van Otten, 1983). The lethal dose for humans is considered to be 523 mg/kg (U.S. EPA, 1985). This is the dose that would be received if 100 percent absorption occurred during exposure to 62 ppm for 1 hour. Tissue damage is likely to occur at levels between 25 and 50 ppm. Upon termination of exposure, however, recovery is rapid. While tissue damage and death are possible at higher concentrations, exposure to lower concentrations produce primarily irritation effects. Formaldehyde irritates the mucous membrane surfaces of the upper respiratory tract, the eyes, and exposed surfaces of the skin (Brabec, 1981).

The average detectable odor level of formaldehyde is approximately 0.1 ppm. Human exposure data suggests that exposures to 10 ppm formaldehyde for one hour are unpleasant but tolerable to most individuals (Brabec, 1981; Kulle, 1987). Levels of up to but not in excess of 25 ppm can be tolerated for a period of one hour without the development of life threatening effects (Brabec, 1981).

Emergency response planning guidelines (ERPGs) have been established by the American Industrial Hygiene Association for formaldehyde. ERPG's are defined as follows:

> *"The Emergency Response Planning Guideline (ERPG) values are intended to provide estimates of concentration ranges where one might reasonably anticipate observing adverse effects as described in the definitions for ERPG-1, ERPG-2, and ERPG-3, as a consequence of exposure to the specific substance.*
>
> *The ERPG-1 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor.*
>
> *The ERPG-2 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action.*
>
> *The ERPG-3 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing or developing life-threatening health effects.*
>
> *It is recognized by the committee (and should be remembered by all who make use of these values that human responses do not occur at*

precise exposure levels, but can extend over a wide range of concentrations. The values derived for ERPGs should not be expected to protect everyone but should be applicable to most individuals in the general population. In all populations there are hypersensitive individuals who will show adverse responses at exposure concentrations far below levels where most individuals would normally respond." (American Industrial Hygiene Association, 1988.)

Table 12-4 lists the occupational and emergency response guidelines for formaldehyde. These values ranged from 1 ppm (ACGIH TLV®) to 25 ppm (ERPG-3) and are useful as indicators of potential effects. The lower value is an eight hour time-weighted value; the higher value is an emergency response planning guideline recommended by the American Industrial Hygiene Association.

Tissue damage from formaldehyde appears to be in the form of cell damage. Cytotoxicity, toxicity to the cells, is evident in cells which are depleted of glutathione. One suggested mechanism of toxicity involves depletion of glutathione, which is necessary to detoxify free radicals which also cause damage. While glutathione is being utilized to oxidize formaldehyde, an increase in the level of free radicals which are not able to be detoxified due to the lack of availability of glutathione causes lipid peroxidation. Lipid molecules, which are constituents of the cell membrane and other pieces of the cell, lose their structure resulting in cell damage (Ku and Billings, 1984). Due to its ability to bind to molecules in the cell, formaldehyde itself is also toxic (Bernstein, et al., 1984).

Chronic (Longer term exposure)

Studies on rats show that at ranges between 1 and 3 ppm, nasal, lung and liver toxicity develop, and at exposure levels greater than 5.6 ppm a decreases in body weight occurs. In humans, chronic exposure to formaldehyde has been associated with irritant effects including respiratory symptoms and mucosal irritation, decreased breathing efficiency with forced exhalation, as well as eye and throat irritation (U.S. EPA, 1985). Based on the

TABLE 12-4. OCCUPATIONAL EXPOSURE LEVELS AND EMERGENCY RESPONSE GUIDELINES FOR FORMALDEHYDE

Existing Federal OSHA PEL [a]	1 ppm
Threshold Limit Value® [a]	1 ppm
Emergency Response Planning Guidelines (ERPG) [c]	
ERPG 1	1 ppm
ERPG 2	10 ppm
ERPG 3	25 ppm

[a] 29 CFR 1910.1048, Permissible Exposure Limit (PEL) 8-Hour Time Weighted Average, 1987.

[b] Threshold Limit Values® and Biological Exposure Indices for 1989-1990. American Conference of Governmental Industrial Hygienists.

[c] American Industrial Hygiene Association, Emergency Response Planning Guidelines. Definitions of ERPG's are in Appendix G.

assumption that visual impairment is a more sensitive indicator of toxic effect than respiratory function, occupational studies determined a level of 3 ppm was adequate for worker safety to protect against noncarcinogenic effects; no vision impairment was found. A Threshold Limit Value® (TLV®) of 1 ppm has been recommended by the American Conference of Governmental Industrial Hygienists (ACGIH, 1989). Threshold Limit Values® are defined in section 9.3.

Carcinogenicity

A 24-month inhalation study performed on rats produced data suggesting formaldehyde is carcinogenic. Nasal tumors were found in rats exposed to 15 ppm (ACGIH, 1986). Formaldehyde has been found to produce nasal cancer in multiple studies on rats and mice at exposures levels ranging from 6.9 to 17.6 mg/m^3. Although the data on mice are limited, in general, the data from the studies is considered adequate to conclude that formaldehyde is carcinogenic in animals (EPA, 1985). No evidence of cancer was found in hamsters. A unit risk value of 1.3×10^{-5} was developed by the EPA in 1987 and is cited in the CAPCOA manual (1989). Human studies regarding carcinogenic potential of formaldehyde have not produced any significant relationship between a particular cancer and formaldehyde exposure (U.S. EPA, 1985). Formaldehyde has a weight-of-evidence classification of B1 suggesting that it is a "probable" human carcinogen having limited human evidence (CAPCOA, 1989).

Reproductive Effects and Developmental Toxicity

No significant reproductive or developmental effects were seen in animal studies (U.S. EPA, 1985). No association was found between formaldehyde exposure and increased spontaneous abortions in a study of hospital equipment-sterilizing personnel (Hemminki et al., 1982).

Genotoxicity

Results have been both positive and negative for the Ames Salmonella reverse-mutation assay. Mutagenicity has been observed in repair deficient E.Coli as well. Formaldehyde has been shown to have mutagenic activity in specific mouse cells when isolated outside of the living animal. No evidence has been demonstrated in intact living organisms possibly because of its rapid biotransformation to other chemicals and its rapid elimination from the body. The mechanism for genotoxicity proposed by numerous scientists is the formation of chemical bridges between DNA molecules and other chemicals in the body (U.S. EPA, 1985).

12.6 Risk Characterization

Risk Characterization combines the results from the emission estimates, modeling analyses, and dose response sections to provide estimates of potential adverse health effects. Two primary adverse health effects were considered: the risk of developing cancer from a prorated exposure to accident emissions and the potential for acute noncancer effects.

Cancer risk was estimated for each exposure scenario by multiplying the prorated exposure determined for each receptor by the unit risk value recommended in the CAPCOA manual (1989). The cancer risk results are summarized in Table 12-5. As noted in the table, the risks range from 2.1×10^{-9} (pedestrian) to a maximum of 3.6×10^{-7} (MEI), assuming a 100-percent probability of the accident actually occurring during a 70-year lifetime.

The potential for short term noncancer effects from exposure to formaldehyde was evaluated by comparing the modeled formaldehyde concentrations in air to available toxicology-based criteria discussed in Section 12.5. The peak concentration at the nearest public road (East Avenue) was estimated to be 14 ppm. This value, which represents the peak plume centerline concentration, exceeds the ERPG-2 of 10 ppm, but is less than the

TABLE 12-5. SUMMARY OF LIFETIME CANCER RISK[a]

Receptor	Risk
Road	
Pedestrian	2.1×10^{-9}
MEI	3.6×10^{-7}
Residence	1.1×10^{-7}

[a] Unit risk value of 1.3×10^{-5} $(\mu g/m^3)^{-1}$ developed by the EPA in 1987 as cited in CAPCOA 1989).

ERPG-3 value of 25 ppm. As discussed under Dose-Response, formaldehyde may cause tissue damage at concentrations exceeding 25 ppm, but exposures under 25 ppm do not appear to cause tissue damage or life threatening effects in most individuals (AIHA, 1988; Brabec, 1981). At a concentration of 14 ppm for one hour, irritation is expected to occur, but once exposure ceases recovery is rapid and complete (Brabec, 1981). With the low odor threshold of less than 1 ppm, it is unlikely that an individual would remain at the plume centerline for the entire one hour period.

In addition to a maximum exposed individual, exposure to a pedestrian or someone escaping the accident plume was examined. Once an individual passing along East avenue detects the strong odor associated with the peak centerline concentration, it is more likely that the individual will move away from the plume than remain in the same location. Movement by the pedestrian 30 meters in either direction perpendicular to the plume centerline will reduce exposure to levels well below those associated with even minor irritation effects or odors. Movement along the plume centerline would be restricted by fences.

The formaldehyde concentration at the nearest residence was estimated to be 4.1 ppm. This level is below the ERPG-2 and levels associated with any significant or irreversible effects.

Based on the accident results and the above discussion, the formaldehyde spill has the potential to cause irritation of the upper respiratory tract, eyes, and exposed skin for an individual exposed at the nearest road to 14 ppm for one hour, but no irreversible effects are expected. An individual passing through the plume, or at the nearest residence, will experience exposures significantly lower than 14 ppm, and hence, below levels associated with irreversible effects.

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