Technical Report
LAX Master Plan EIS/EIR

14a. Human Health Risk Assessment Technical Report

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Prepared for:

Los Angeles World Airports
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EXECUTIVE SUMMARY

Due to the overall increase in activity levels at LAX associated with implementation of any of the Los Angeles International Airport (LAX) three build alternatives, increased emissions of toxic air pollutants (TAPs) are possible. These emissions may come from a variety of sources including aircraft, ground service equipment (GSE), on- and off-airport traffic, and maintenance facilities. Potential impacts associated with increases in releases of TAPs to air in the South Coast Air Basin may include increased cancer risks and non-cancer health hazards from inhalation of TAPs by people working, living, recreating, or attending school on or near the airport. TAPs of greatest concern in emissions from LAX include diesel particulates, 1,3-butadiene, benzene, and acrolein.

Possible impacts to human health can be assessed through development of a human health risk assessment (HHRA). A HHRA for toxic air pollutant (TAP) emissions associated with the proposed LAX Master Plan, as required under State of California statutes and regulations, was conducted in four phases as defined in California Environmental Protection Agency (CalEPA) and U.S. Environmental Protection Agency (USEPA) guidance. These steps included:

- Identification of chemicals (in this case, TAPs) that may be released in sufficient quantities to present a public health risk (Hazard Identification).
- Analysis of ways in which people might be exposed to chemicals (TAPs) (Exposure Assessment).
- Evaluation of the toxicity of chemicals (TAPs) that may present public health risks (Toxicity Assessment).
- Characterization of the magnitude and location of potential health risks for the exposed community (Risk Characterization).

Methods used are in the HHRA are conservative. That is, methods are used that are more likely to overestimate than underestimate possible health risks. For example, risks are calculated for individuals that are likely to be exposed at locations where TAP concentrations are predicted to be highest. Further, individuals are assumed to be exposed for almost all days of the year and for many years to maximize estimates of possible exposure. Resulting incremental risk estimates represent upper-bound predictions of exposure, and therefore health risk, that may be associated with living near, and breathing emissions from, LAX during and after implementation of the Master Plan. By protecting hypothetical individuals that receive the highest exposures, the risk assessment will also be protective for actual members of the population near LAX that are not as highly exposed.

The HHRA was conducted in two phases. First, a screening level assessment was used to focus the final HHRA on TAPs, receptors (people), exposures, and locations of potential concern for the EIS/EIR process. During the screening level assessment, emission sources for TAPs at LAX were identified, emissions of individual TAPs were estimated, and TAPs of concern for LAX were selected. In addition, populations that may be affected by TAPs from LAX and exposure pathways (e.g., inhalation of TAPs in air, deposition of TAPs onto soils) were identified. Screening-level air dispersion modeling was conducted to assist with initial determination of which areas and populations near LAX were the most important for the final risk characterization, and to assist in evaluation of deposition of TAPs onto soils and other surfaces.

During the final HHRA, refined air dispersion modeling was conducted and incremental cancer risks and non-cancer health hazards were characterized for receptor populations (people living near LAX) and exposure pathways identified in the screening level assessment.

Estimates of possible incremental cancer risks associated with the build alternatives indicate that thresholds of significance might be exceeded in horizon year 2005 if no Mitigation Measures are implemented. These risks are associated mainly with exposure to diesel particulates, 1,3-butadiene, and benzene released from various LAX-associated sources, including vehicles involved in construction. Toxicity of particulate matter released with diesel exhaust is uncertain, and estimates for incremental cancer risks could be over- or under-estimated to some degree.

HHRA estimates of possible incremental human health impacts associated with Alternatives A, B, and C indicated that thresholds of significance for non-cancer health hazards could be exceeded in horizon year 2015 prior to implementation of Mitigation Measures. Non-cancer hazard estimates are highly uncertain, however, because of the paucity of data on acrolein emissions from jet aircraft engines. Acrolein is responsible for almost all non-cancer hazard, yet is not generally recognized as a significant TAP in the South Coast Air Basin and was not addressed in the Multiple Air Toxics Exposure Study-II (MATES-II)

conducted by the South Coast Air Quality Management District (SCAQMD).[1] Dependence on regulatory databases with estimated acrolein emissions may have substantially overestimated possible releases of acrolein during LAX operations. After mitigation, however, both cancer risks and non-cancer health hazards would be less than significant for the build alternatives. This finding would hold true both during construction (2005) and after completion of construction (2015).

Further, results of the assessment suggest that the three build alternatives would have smaller adverse health impacts than the No Action/No Project Alternative. In fact, for most areas near the airport, the build alternatives would actually result in a decrease in health risks and hazards because of improved airport operations and traffic flows. The No Action/No Project Alternative, however, would cause maximally exposed individual (MEI)[2] cancer risks and non-cancer hazards to increase in all areas near LAX in both horizon years.

Preliminary assessments of potential Mitigation Measure benefits are projected to reduce all potential MEI incremental cancer risks or non-cancer health hazards to below thresholds of significance for the three build alternatives. After mitigation, no significant impacts are anticipated. Accompanying these reductions in MEI risks and hazards, areas near the airport where risks and hazards might increase incrementally would be dramatically reduced in size, or eliminated, compared to those estimated for the No Action/No Project Alternative. Mitigation Measures would be effective in eliminating significant impacts estimated under pre-mitigation conditions for the build alternatives.

Cumulative impacts of the three build alternatives were evaluated by comparison of possible incremental cancer risks with the results of the MATES-II, which evaluated possible cancer risks associated with air toxics within the South Coast Air Basin. The three build alternatives would reduce possible cancer risks associated with LAX operations for areas near the airport. These reductions in cancer risk -- a maximum of -10 to -20 in one million -- would reduce cumulative impacts of LAX operations compared with baseline conditions. Anticipated reductions in cancer risk are small compared with the range of cancer risks estimated for the Basin (average of about 1,400 in one million). The comparisons indicate that LAX emissions under the build alternatives would result in reduced cumulative cancer risks for some areas nearest the airport. However, because many sources of TAPs not related to LAX operations contribute to total risks within the South Coast Air Basin, potential cancer risks for all populations within the Basin would remain high even after reduction in the contribution from LAX.

The above conclusions are based on several key findings of the human health risk assessment, including:

- Potential incremental cancer risks for the No Action/No Project Alternative under pre-mitigation conditions were higher for both horizon years than estimated risks for the build alternatives by factors of 2 to 4. These differences were due to less efficient aircraft operations and greater traffic congestion as existing LAX facilities become more constrained to accommodate additional passengers and freight in the future.
- Potential incremental cancer risks and non-cancer health hazards for the three build alternatives under post-mitigation conditions would be less than estimated cancer risks and non-cancer health hazards for the No Action/No Project Alternative for both horizon years. Further, all incremental risks and hazards for the build alternatives would be less under post-mitigation conditions than appropriate thresholds of significance.
- Compared to background cancer risks as defined by MATES-II, the three build alternatives and the No Action/No Project Alternative under pre-mitigation conditions would not contribute greatly to current cumulative impacts. Under post-mitigation conditions for 2015, implementation of any of the build alternatives would result in a decrease in cumulative risks for residents living east of the airport.
- The three build alternatives might have significant human health impacts, under pre-mitigation conditions for both horizon years. Possible incremental MEI cancer risks exceed the threshold of significance of 10 in one million for all build alternatives in horizon year 2005. Possible incremental MEI non-cancer health hazards exceed the threshold of significance of 5 for the three build alternatives under pre-mitigation conditions for horizon year 2015.

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1 SCAQMD, Multiple Air Toxics Exposure Study in the South Coast Air Basin (MATES-II), November 1999.

2 MEI is a hypothetical individual that lives, works, or goes to school at a location with the highest predicted concentrations of TAPs in air, and who has other characteristics, such as inhalation rate and years of exposure, that result in maximum intake of TAPs.

- The build alternatives with mitigation would have no significant human health impacts at either horizon year. Possible incremental cancer risks would be below the threshold of significance of 10 in one million; in fact, risks would be reduced compared to baseline conditions for most locations near the airport. Possible non-cancer health hazards would be less than the threshold of significance of 5; in fact, hazards would be reduced compared to baseline conditions for many locations near the airport.
- The area surrounding LAX where incremental cancer risks might be positive (but less than the threshold of significance) is very small for all build alternatives with mitigation when compared to the area where similar incremental cancer risks might be found under the No Action/No Project Alternative.
- The area surrounding LAX where non-cancer health hazard indices greater than 5 are anticipated under the No Action/No Project Alternative completely disappears for the three build alternatives with mitigation. Possible non-cancer risks under the three build alternatives would actually be reduced below baseline estimates following mitigation for horizon year 2005 and, for some cases, at horizon year 2015.
- Emissions of TAPs during LAX operations would not be expected to exceed workplace standards and worker exposures, therefore, are not expected to be significant.

1. INTRODUCTION

This Technical Report presents detailed information on methodology and baseline conditions related to the human health risk associated with implementation of the Los Angeles International Airport (LAX) Master Plan. This report provides data and analysis in support of the Environmental Impact Statement/Environmental Impact Report (EIS/EIR) for the LAX Master Plan prepared pursuant to the National Environmental Policy Act (NEPA) and the California Environmental Quality Act (CEQA).

This Technical Report provides information on the methodology used, as well as the baseline conditions and environmental consequences, that supports material presented in Section 4.24.1, *Human Health Risk Assessment*, of the EIS/EIR. References to this report are provided in Attachment A, *Bibliography*.

2. GENERAL APPROACH

A HHRA for TAP emissions associated with the proposed LAX Master Plan is required under State of California statutes and regulations. Risk assessments are conducted in four phases as defined in California Environmental Protection Agency (CalEPA) and U.S. Environmental Protection Agency (USEPA) guidance. These steps include:

- Identification of chemicals (in this case TAPs) that may be released in sufficient quantities to present a public health risk (Hazard Identification)
- Analysis of ways in which people might be exposed to chemicals (TAPs) (Exposure Assessment)
- Evaluation of the toxicity of chemicals (TAPs) that may present public health risks (Toxicity Assessment)
- Characterization of the magnitude and location of potential health risks for the exposed community (Risk Characterization)

Methods used in the HHRA are conservative. That is, methods are used that are more likely to overestimate than underestimate possible health risks. For example, risks are calculated for individuals that are likely to be exposed at locations where TAP concentrations are predicted to be highest. Further, individuals are assumed to be exposed for almost all days of the year and for many years to maximize estimates of possible exposure. Resulting incremental risk estimates represent upper-bound predictions of exposure, and therefore health risk, that may be associated with living near and breathing emissions from LAX. By protecting hypothetical individuals that receive the highest exposures (i.e., people living at locations for which the highest emissions are predicted), the risk assessment will also be protective for actual members of the population near LAX that are not as highly exposed.

The HHRA for the LAX Master Plan followed closely CalEPA and USEPA guidance, as adapted for the unique environment of the airport. The assessment was conducted in two phases. A screening level assessment was used to focus the final HHRA on TAPs, receptors (people), exposures and locations of potential concern for the EIR process. A screening analysis intentionally exaggerates possible health risks to determine the relative importance of different TAPs and exposure routes (inhalation, ingestion, and

dermal contact). A screening assessment is a valuable tool for focusing subsequent analysis on the most important issues.

During the screening level assessment, emission sources for TAPs at LAX were identified, emissions of individual TAPs were estimated, and TAPs of concern for LAX were selected. In addition, populations that may be impacted by TAPs from LAX and exposure pathways (e.g., inhalation of TAPs in air, deposition of TAPs onto soils) were identified. Screening-level air dispersion modeling was conducted to assist with initial determination of which areas and populations near LAX were the most important for the final risk characterization, and to assist in evaluation of deposition of TAPs onto soils and other surfaces.

During the final HHRA, refined air dispersion modeling was conducted and incremental risks that may be associated with increased emissions from LAX above current baseline emissions were characterized for receptor populations (people living near LAX) and exposure pathways identified in the screening level assessment. This Technical Report presents a summary of the initial tasks conducted in the screening level assessment and presents the results of characterizations of incremental risks conducted for Alternatives A, B, and C and for the No Action/No Project Alternative. Analyses conducted during the screening level assessment are described in detail in Attachment B, *Screening Level Human Health Risk Assessment*. A flow chart for major steps included in the HHRA (screening and final is provided as **Figure 1**, Process Flow Chart for LAX HHRA.

3. SUMMARY OF SELECTION OF TAPS OF CONCERN

TAPs of concern for LAX were selected based on identification of chemicals as TAPs in federal and state regulations, current or future presence in emissions at LAX, magnitude of possible emissions, and toxicity. TAPs of concern for LAX are those chemicals that could be released in sufficient amounts to contribute substantially to overall impacts from airport operations. Inclusion of a chemical as a TAP of concern does not indicate that the TAP will have important impacts; inclusion only suggests that additional analysis is warranted. TAP listings in regulations were used to help guide identification of TAPs of concern, and particular attention was paid to federal and state listings for toxic substances released to the atmosphere.

The process for identifying TAPs of concern included six steps:

- Sources of TAPs at LAX were identified.
- Specific TAPs associated with sources at LAX were identified.
- TAPs potentially released during LAX operations were compared to TAPs listed in state and federal guidance.
- Emissions of individual TAPs were estimated.
- Relative percent impact was estimated for each TAP using toxicity criteria and emissions estimates and TAPs contributing at least 0.1 percent to total relative impacts were selected.
- TAPs selected based on percent impact were further screened through comparisons with Region IX Preliminary Remediation Goals (PRGs) after final air dispersion modeling was completed.

Details of the selection process are provided in Attachment B, *Screening Level Human Health Risk Assessment.* A brief summary is provided below.

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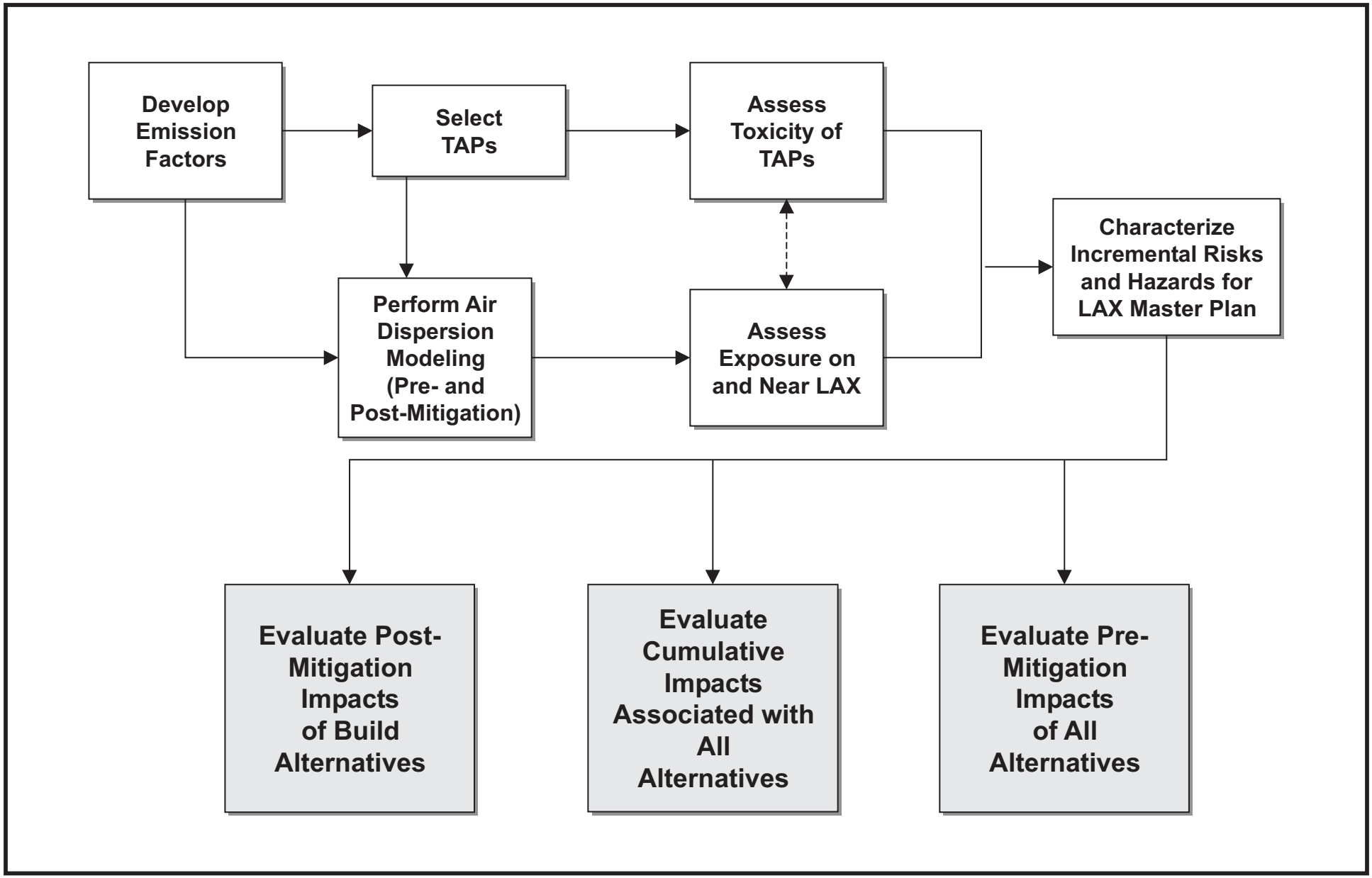

Los Angeles International Airport Master Plan	**Process Flow Chart for LAX HHRA**	Figure 1

3.1 Identification of TAP Sources (Step 1) and TAPs Associated with Sources at LAX (Step 2)

TAP sources were identified during extensive surveys of airport facilities, automotive traffic, air traffic, and typical airport operations. An inventory of potential sources of air pollutants at LAX was conducted in 1997 using the following information:

- Recent surveys of stationary sources at LAX (see *Land Use Technical Report*).
- SCAQMD databases of stationary sources.
- Previous LAX surveys.

TAP sources include exhaust from aircraft and ground vehicles, as well as a variety of other sources related to maintenance operations, airport utilities, and fuel farms.

After TAP sources were identified, chemicals released from each source were characterized. Information sources used for this analysis step included, but were not limited to the following:

- California Air Toxics Emission Factors (CATEF) Database.[3]
- Factor Information Retrieval (FIRE) System Database.[4]
- Volatile Organic Compounds/Particulate Matter (VOC/PM) Speciation Data System (SPECIATE) Database.[5]
- Crosswalk/Air Toxic Emission Factor (XATEF) Database.[6]
- USEPA Memorandum, Re: Source Identification and Base Year 1990 Emission.[7]
- Inventory Guidance for Mobile Source HAPs on the USEPA Office of Air Quality Planning Services (OAQPS) List of 40 Priority HAPs.[8]
- Motor Vehicle-Related Air Toxics Study.[9]
- FAA's Aircraft Engine Emissions Database (FAEED) Version 2.1.[10]
- FAA/U.S. Air Force (USAF) Emissions and Dispersion Modeling System (EDMS) Version 3.02.[11]
- Compilation of Air Pollutant Emission Factors (AP-42).[12]
- EMFAC7 Mobile Emissions Model, Version 7F and 7G.[13]
- TANKS Tank Emissions Estimation Model, Version 3.0.[14]

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3 California Air Resources Board, California Air Toxics Emission Factors Database User's Manual, Version 1.2, October 1993.

4 USEPA Office of Air Quality Planning and Standards, Factor Information Retrieval (FIRE) System: User's Manual, September 1993.

5 USEPA Office of Air Quality Planning and Standards, Volatile Organic Compound (VOC)/Particulate Matter (PM) Speciation Data System (SPECIATE) User's Manual, Version 1.5, February 1993.

6 USEPA Office of Air Quality Planning and Standards, Crosswalk/Air Toxic Emission Factor (XATEF) Database Management System User's Manual, Version 2.0, EPA-450/B-92-011, October 1992.

7 USEPA Office of Mobile Sources, Memorandum from Rich Cook to Anne Pope, Re: Source Identification and Base Year 1990 Emission Inventory Guidance for Mobile Source HAPs on the OAQPS List of 40 Priority HAPs, June 11, 1997.

8 USEPA, Source Identification and Base Year 1990 Emission Inventory Guidance for Mobile Source HAPs on the OAQPS List of 40 Priority HAPs, 1997.

9 USEPA Office of Mobile Sources, Emission Planning and Strategies Division, Motor Vehicle-Related Air Toxics Study, Report Number EPA 420-R-93-005, 1993.

10 Federal Aviation Administration Office of Environment and Energy (AEE-120) and the United States Air Force Armstrong Laboratory Tyndall Air Force Base, Emissions and Dispersion Modeling System (EDMS) Reference Manual, FAA-AEE-97-01, 1997.

11 Federal Aviation Administration and the United States Air Force Armstrong Laboratory, Tyndall Air Force Base and FAA Office of Environment and Energy (AEE-120), Air Quality Procedures for Civilian Airports & Air Force Bases, 1997.

12 USEPA Office of Air Quality Planning and Standards, Compilation of Air Pollution Emission Factors. Volume I: Stationary Point and Area Sources (AP-42, 5th Edition and Supplements), 1997.

13 California Air Resources Board and California Department of Transportation, Methodology for Estimating Emissions from On-Road Motor Vehicles B Volume II: EMFAC7G, November 1996.

- Air Pollution Mitigation Measures for Airports and Associated Activity.[15]
- Air Quality Procedures for Civilian Airports and Air Force Bases.[16]
- CEQA Air Quality Handbook.[17]

TAPs potentially associated with sources at LAX are listed in Attachment B, *Screening Level Human Health Risk Assessment*.

3.2 Comparison of TAPs listed in State and Federal Guidance with LAX-Related TAPS (Step 3)

Chemicals that may be released at LAX were compared to TAPs listed in state and federal regulations to identify TAPs considered potential health threats for air releases by regulatory agencies. Three state lists (SCAQMD Rules 1401 and 1402, AB2588 and AB1807/2728) and one federal list (the Clean Air Act (CAA) were consulted. Almost all chemicals identified as chemicals potentially associated with LAX emissions were also found on state and/or federal lists of TAPs. Because very few chemicals that may be associated with operations at LAX are not listed in state or federal regulations, a decision was made to not eliminate any chemicals based on comparison with regulatory lists. All chemicals identified in releases from LAX sources in Step 2 were carried into Step 4.

3.3 Emissions Estimates for TAPs (Step 4)

Operational data for emissions sources and chemical species in exhausts or other forms of air emission were characterized using the above databases and peer-reviewed literature sources. Emissions factors and operational parameters were then used to estimate annual emissions for TAPs associated with operations at LAX.

Emissions estimates for TAPs at LAX were generated in two phases. In the first phase (Phase I), emissions were estimated for the No Action/No Project Alternative year 2015 using data collected during a previous survey. In the second phase (Phase II), emissions estimates for the No Action/No Project Alternative were refined based on inspections at LAX and interviews with LAX tenants identified by Los Angeles World Airports (LAWA). In addition, projections for future emissions from LAX, based on Alternative B year 2015, were generated from the descriptions of airport operations expected for this alternative. The purpose of screening emissions for Alternative B year 2015 was to identify additional TAPs of concern that may be present in significant quantities in emissions for 2015, but not in emissions for the No Action/No Project Alternative. As indicated in Section 6, *Risk Characterization*, TAP releases, and resulting potential human health impacts are greatest for Alternative B, making this alternative the most appropriate for use in selecting TAPs of concern.

Phase I analyses indicated that aircraft emissions account for about 97 percent of total overall emissions and also contribute most to emissions of individual TAPs. For example, for acrolein, the chemical associated with the greatest potential non-cancer health hazards at LAX, aircraft emissions were estimated to comprise more than 99 percent of total emissions. Phase II screening analyses therefore focused on aircraft emissions. For the Phase II analysis total airport emissions from aircraft and other sources were estimated by adding Phase I non-aircraft emissions to Phase II aircraft emissions. Phase I and II emissions estimates are presented in **Table 1**, Emissions Summary.

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[14] USEPA Office of Air Quality Planning and Standards, User's Guide to Tanks. Storage Tank Emissions Calculation Software, Version 3.1, 1997.

[15] California Air Resources Board Research Division, Air Pollution Mitigation Measures for Airports and Associated Activity, CARB A132-168, 1994.

[16] Federal Aviation Administration and the United States Air Force Armstrong Laboratory, Tyndall Air Force Base and FAA Office of Environment and Energy (AEE-120), Air Quality Procedures for Civilian Airports & Air Force Bases, 1997.

[17] SCAQMD, CEQA Air Quality Handbook, 1993.

Table 1

Emissions Summary
Phase I/II

	Phase I			Phase II					
	No Action/No Project Emission Estimates			Revised No Action/No Project 2015 Emission Estimates			Alternative B 2015 Emission Estimates		
Source Category	AircraftTotals (kg/yr)	Non-aircraft Totals (kg/yr)	Total Operating, kg/year	AircraftTotals (kg/yr)	Non-aircraft Emissions (kg/yr)[1]	Aircraft + Non-aircarft Emissions (kg/yr)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr)[1]	Aircraft + Non-aircraft Emissions (kg/yr)
1,3-Butadiene	20,876	2,567	23,443	8,926	2,567	11,494	10,857	2,567	13,424
Acenaphthene	0	1.92	1.92	7.04	1.92	8.96	9.51	1.92	11
Acenaphthylene	0.0081	8.26	8.26	10	8.26	19	14	8.26	22
Acetaldehyde	53,956	1,574	55,530	14,873	1,574	16,447	18,022	1,574	19,596
Acrolein	26,323	235	26,559	6,909	235	7,144	8,399	235	8,634
Anthracene	0.51	0.96	1.47	62	0.96	63	84	0.96	85
Arsenic	1,208	1.14	1,210	9.51	1.14	10.65	13	1.14	14
Benzaldehyde	0.0030	5.94	5.95	2,360	5.94	2,366	2,845	5.94	2,851
Benzene	22,840	20,412	43,251	10,348	20,412	30,760	12,547	20,412	32,959
Benzo(a)anthracene	0.30	1.08	1.38	5.32	1.08	6.39	7.17	1.08	8.24
Benzo(a)pyrene	0.18	0.48	0.66	1.24	0.48	1.71	1.67	0.48	2.15
Benzo(b)fluoranthene	0.029	0.59	0.62	4.12	0.59	4.71	5.56	0.59	6.15
Benzo(g,h,i)perylene	0.042	0.89	0.94	2.19	0.89	3.08	2.96	0.89	3.85
Benzo(k)fluoranthene	0.029	0.41	0.44	4.12	0.41	4.53	5.56	0.41	5.97
Beryllium	0	0.0044	0.0044	2.56	0.0044	2.56	3.48	0.0044	3.48
Cadmium	114	8.03	122	15	8.03	23	21	8.03	29
Chromium Hexavalent(all sources)	0.0000021	0.00087	0.00087	0.51	0.00087	0.51	0.69	0.00087	0.69
Chromium (total)		0		20	52	72	27	52	79
Chrysene	0.19	0.87	1.06	11	0.87	12	15	0.87	15
Copper	0.064	46	46	47	46	93	64	46	110
Dibenz(a,h)anthracene	0.00017	0.24	0.24	3.14	0.24	3.39	4.24	0.24	4.49
Ethylbenzene	0.059	69	69	1,995	69	2,064	2,425	69	2,494
Fluoranthene	1.47	1.56	3.03	192	1.56	194	259	1.56	261
Fluorene	0	3.03	3.03	0.00	3.03	3.03	0	3.03	3.03
Formaldehyde	174,648	3,969	178,617	48,316	3,969	52,285	58,579	3,969	62,548
Hexane	0.22	96	96	6,493	96	6,589	7,906	96	8,002
Indeno(1,2,3-cd)pyrene	0.00013	0.24	0.24	2.46	0.24	2.71	3.32	0.24	3.57
Lead	1,253	1,687	2,941	29	1,687	1,716	39	1,687	1,726
Manganese	0	120	120	485	120	605	659	120	780
Mercury	0.0013	0.62	0.62	0.13	0.62	0.74	0.17	0.62	0.79
Naphthalene	418	608	1,026	11,668	608	12,276	15,703	608	16,311
Nickel	114	29	143	2,298	29	2,328	3,124	29	3,153
Phenanthrene	6.44	11	17	628	11	639	845	11	856
Propylene	50,747	8,119	58,866	29,036	8,119	37,156	35,313	8,119	43,432

Table 1

Emissions Summary
Phase I/II

	Phase I			Phase II					
	No Action/No Project Emission Estimates			Revised No Action/No Project 2015 Emission Estimates			Alternative B 2015 Emission Estimates		
Source Category	AircraftTotals (kg/yr)	Non-aircraft Totals (kg/yr)	Total Operating, kg/year	AircraftTotals (kg/yr)	Non-aircraft Emissions (kg/yr)[1]	Aircraft + Non-aircarft Emissions (kg/yr)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr)[1]	Aircraft + Non-aircraft Emissions (kg/yr)
Pyrene	1.65	1.43789	3.09	160	1.44	162	216	1.44	217
Selenium	114	0.00068	114	0.40	0.00068	0.40	0.54	0.00068	0.55
Styrene	4,574	1,052	5,626	2,975	1,052	4,027	3,616	1,052	4,668
Toluene	5,101	33,883	38,984	14,511	33,883	48,394	17,663	33,883	51,547
Xylene (total)	4,709	17,645	22,354	10,679	17,645	28,324	12,988	17,645	30,633
Xylene, m- or p-	2,857	0	2,857	7,791	0	7,791	9,474	0	9,474
Xylene, o-	1,872	0	1,872	2,895	0	2,895	3,521	0	3,521
Zinc	1,254	649	1,903	2,534	649	3,183	3,444	649	4,093

[1] Non-aircraft emissions for Phase II estimates were calculated as part of Phase I.

Source: Camp Dresser & McKee Inc., 2000.

3.4 Toxicity Screening for TAPs Released from LAX (Step 5)

Relative impacts for TAPs were estimated using emissions estimates for the No Action/No Project Alternative and Alternative B, Year 2015 (Attachment B, *Screening Level Human Health Risk Assessment*) and toxicity information presented in Section 4, *Exposure Assessment*. Because TAP selection is based on emissions estimates for 2015, the time when all construction associated with the LAX Master Plan is expected to be completed, construction emissions were not considered in the selection of TAPs of concern. Sources of TAPs during construction would be from sources such as diesel construction equipment that are similar to sources currently associated with day-to-day airport activities. Therefore, construction might influence the quantity of TAPs released, but is unlikely to introduce new TAPs not considered in the selection of TAPs of concern.

Chemicals estimated to contribute at least 0.1 percent to overall impacts associated with LAX operations were retained as TAPs of concern in this screening step. Relative impacts were estimated using toxicity criteria developed by regulatory agencies, and toxicity criteria proposed, but not yet adopted, by the State of California (i.e., California Reference Exposure Levels [RELs]). Chemicals with relative impact equal to or exceeding 0.1 percent based on Phase I or Phase II emission estimates and current CalEPA or USEPA toxicity values were retained as TAPs of concern for quantitative risk analyses. A threshold of 0.1 percent was taken from USEPA guidance.[18] USEPA recommends a value of 1 percent for toxicity screening. One-tenth of this value was used in the screening analysis for LAX to ensure that the analysis would be protective for all TAPs that might be released. A few TAPs were also retained as TAPs of concern based on use of California proposed RELs as toxicity criteria. Inclusion of RELs was used to help determine the impact, if any, of future adoption of RELs on the conclusions of the HHRA.

Diesel particulates were not included in the TAP screening analysis (Attachment B, *Screening Level Human Health Risk Assessment*) because diesel emissions estimates were not available at the time the screening was conducted. Diesel was included as a TAP of concern based on MATES-II[19] results that indicated that exposure to diesel particulates may cause the highest cancer risks of all TAPs found in air in the South Coast Air Basin. Inclusion of diesel as a TAP of concern is discussed separately in Section 3.6, *Evaluation of Diesel Exhaust as a TAP of Concern*.

For carcinogenic TAPs, impact factors for each TAP were determined by multiplying annual emissions by an established cancer slope factor. For non-carcinogenic TAPs, impact factors are determined by dividing emissions estimates for each TAP by established reference doses. In all cases, current California cancer slope factors took precedence over federal criteria and inhalation criteria took precedence over oral criteria. The percent contribution for each chemical to overall potential impact was then calculated by dividing the estimated impact factor for individual chemicals by the sum of all impact factors. The analysis was conducted separately for carcinogens and non-carcinogens.

Toxicity screening results using Phase I and II emissions estimates suggested that for carcinogens ten TAPs would likely contribute over 99.9 percent of potential risks. These chemicals included volatile organic compounds (1,3-butadiene, benzene, formaldehyde, and acetaldehyde), one semivolatile (2,3,7,8-tetrachlorodibenzo(p)dioxin equivalents (TCDD)), and several metals (arsenic, beryllium, cadmium, chromium, and manganese). Arsenic and 1,3-butadiene were initially predicted to dominate total impacts. In the screening analysis with Phase I emissions estimates, they accounted for approximately 36.3 and 36.1 percent of total impacts, respectively. In addition, carcinogenic polycyclic aromatic hydrocarbons (PAHs) were not eliminated even though, combined, these seven chemicals were predicted to contribute less than 0.5 percent to overall impacts. Carcinogenic PAHs have been a subject of public concern and, therefore, warrant additional evaluation. Refined emission estimates (Section 3.5, *Refined Taps of Concern (Step 6)*) and air dispersion modeling decreased the projected impact of arsenic as discussed in later sections.

Toxicity screening indicated that, among systemic toxicants, acrolein would be associated with the greatest impacts. Screening against existing USEPA toxicity criteria indicated that acrolein would

[18] USEPA Office of Emergency and Remedial Response, Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual Interim Final, December 1989.

[19] SCAQMD, Multiple Air Toxics Exposure Study in the South Coast Air Basin (MATES-II), November 1999.

contribute over 98.6 percent (Phase I emissions) and 93.8 percent (Phase II emissions) of the cumulative relative impact of all TAPs evaluated. Screening against proposed California RELs suggested approximately 90 percent contribution from acrolein. Because acrolein strongly dominated non-cancer impact estimates, percent contribution for other systemic toxicants was determined after subtracting out the impact factor for acrolein. Screening using USEPA toxicity criteria using Phase I and II emissions estimates indicated that eight TAPs would contribute more than 0.1 percent to total impacts in the absence of acrolein. Screening using RELs suggested that in the absence of acrolein, four additional chemicals may have relative impacts of 0.1 percent or more. Chemicals identified in either screening step were retained as TAPs of concern.

Potential impacts for chemicals for which toxicity criteria are not available were separately evaluated based on estimated magnitude of emissions and qualitative toxicity. Lead (Pb) was eliminated as a TAP of concern because maximum on-airport air concentrations predicted with screening-level and dispersion modeling were less than the ambient air quality standard of 1.5 $\mu g/m^3$.

3.5 Refined TAPs of Concern (Step 6)

One additional screening step was conducted to further evaluate TAPs of potential concern identified in Steps 1 through 6. Once final air dispersion modeling results were available for Alternative B, maximum predicted annual average air concentrations were compared to USEPA Region IX PRGs, as modified for use in California. Where maximum predicted concentrations were minimal compared to these PRGs, the TAP was eliminated from further quantitative evaluation. In all cases where TAPs were eliminated, the maximum concentration predicted in the modeling for Alternative B was orders of magnitude less than the PRG, indicating that no human health impacts, even those resulting from exposure to multiple chemicals would be possible. TAPs eliminated in this step included copper, nickel, selenium, toluene, and zinc.

TAPs selected for quantitative evaluation in Steps 1 through 6 are presented in **Table 2**, Toxic Air Pollutants of Concern for LAX.

3.6 Evaluation of Diesel Exhaust as a TAP of Concern

Diesel exhaust was not screened as a TAP of concern in the above analysis because emissions estimates for diesel were not available at the time the TAP screening was conducted. Diesel is, however, included as a TAP of concern for quantitative evaluation because diesel exhaust is expected to be emitted in large quantities from LAX under the three build alternatives and the No Action/No Project Alternative.

Diesel exhaust is emitted from several ground sources (predominantly trucks and buses). Aircraft use a lighter fuel and a substantially different combustion process than diesel engines. The result is dramatically lower emissions of particulates in exhaust, and probably much different toxicological properties. The HHRA considered only diesel exhaust from ground sources in estimating risks. Justification for this approach is provided below.

Diesel exhaust, in the form of particulate matter (PM), is evaluated as a TAP of concern in the HHRA using the approach presented by SCAQMD in the MATES-II Study.[20] Toxicity criteria used in California consider the unique toxicity and physical and chemical characteristics of diesel exhaust. Particulate matter present in jet exhaust is not considered chemically, physically, or toxicologically similar to diesel exhaust based on inherent differences in fuel composition, combustion properties, and exhaust composition and toxicity.

[20] SCAQMD, Multiple Air Toxics Exposure Study in the South Coast Air Basin (MATES-II), November 1999.

Table 2

Toxic Air Pollutants of Concern for LAX

Substance	CAS Number	Chemical Class
Acetaldehyde	5-07-0	Volatile organic
Acrolein	107-02-8	Volatile organic
Arsenic	7440-38-2	Metalloid
Benz(a)anthracene [1]	556-55-3	Carcinogenic PAH
Benzene	71-43-2	Volatile organic
Benzo(b)fluoranthene [1]	205-99-2	Carcinogenic PAH
Benzo(k)fluoranthene [1]	207-08-9	Carcinogenic PAH
Benzo(a)pyrene [1]	50-32-8	Carcinogenic PAH
Beryllium[2]	7440-41-7	Metal
1,3-Butadiene	106-99-0	Volatile organic
Cadmium	7440-43-9	Metal
Chromium (total) (evaluated as Cr(VI))	7440-47-3	Metal
Chrysene[1]	218-01-9	Carcinogenic PAH
Dibenz(a,h)anthracene [1]	53-70-3	Carcinogenic PAH
Particulates in diesel exhaust		Particulate
Formaldehyde	50-00-0	Volatile organic
Indeno(1,2,3-cd)pyrene [1]	193-39-5	Carcinogenic PAH
Manganese	7439-96-5	Metal
Naphthalene[4]	91-20-3	PAH
Xylene[3]	1330-20-7	Volatile organic
2,3,7,8-TCDD equivalents	1746-01-6	Chlorinated Dioxins and Furans

[1] Carcinogenic PAHs were retained as TAPs of concern, even though they were not identified in the toxicity screening. This group of chemicals was retained due to public concern with PAHs.
[2] Greater than 0.1 percent relative impact under No Action/No Project Alternative Year 2015 emissions and only if CalEPA RELs adopted.
[3] Selected only if proposed CalEPA RELs are adopted.
[4] Greater than 0.1 percent relative impact under No Action/No Project Alternative Year 2015 emissions scenario.

Source: Camp Dresser & McKee Inc., 1998.

Diesel fuel is a complex mixture of thousands of individual compounds, most with carbon numbers between 10 and 22. Most of these compounds are members of the paraffinic, naphthenic, or aromatic class of hydrocarbons. Generally speaking, more than half of the molecules in diesel fuels contain at least 15 carbon atoms.[21] Jet fuel differs significantly from diesel fuel both physically and chemically, being significantly lighter with shorter carbon chains, smaller molecules (generally), and more uniform composition. Commercial jet fuel is similar to kerosene in composition and contains an array of carbon chain-lengths from 4 to 16 carbons long.[22]

Diesel engines and jet engines also differ in their combustion mechanisms and fuel combustion efficiencies. Most diesel engines are based on the compression/ignition principle. In a typical four-stroke compression/ignition four-stroke cycle, air is drawn into the cylinder in the intake stroke and then compressed, creating space for finely atomized diesel fuel to be sprayed into the hot air, initiating auto-ignition of the mixture. During the subsequent power stroke, the expanding hot mixture forces the piston down. The final exhaust stroke purges the burnt gases. The diesel cycle relies upon warm vapor for combustion of fuel injected in pulses into cylinders. As a result, the combustion process is often incomplete or inefficient, creating a large amount of partially oxidized carbon-containing particulate matter in the exhaust. Hazardous components of diesel exhaust include, but are not limited to: benzene, arsenic, nickel, benzo(a)pyrene, 1,3-butadiene, formaldehyde, a variety of hydrocarbons, carbon monoxide, sulfur oxides, nitrogen oxides, and particulate matter (PM). Concentrations of PM and other hazardous components in diesel exhaust vary significantly depending on factors such as engine type and condition, fuel grade, and combustion efficiency.

[21] Chevron Company, Information about Diesel Fuel Chemistry, Available: http//www.chevron.com/prodserv/bulletin/diesel [June 2000].

[22] Agency for Toxic Substances and Disease Registry (ATSDR), Toxicological Profile for Jet Fuel, CD-ROM, 1997.

Toxicological research indicates that the component of diesel exhaust responsible for most toxicological effects is PM.[23] Diesel PM typically consists of a solid core, composed mainly of elemental carbon, with a coating of various organic and inorganic compounds. More than 75 percent of diesel exhaust particles have diameters smaller than 1 micrometer (µm), with typical particles sized between 0.1 and 0.25 µm.[24] For reference, particles 10 µm and smaller are generally respirable, meaning that they deposit into the deepest and often most sensitive areas of the lung (the alveoli). Particles that deposit in the deep lung are not removed in mucus that protects much of the respiratory tree and may reside in the lung for long periods of time.

According to USEPA's Integrated Risk Information System (IRIS), the systemic (non-cancer) toxicity of diesel emissions is due to the insoluble carbon core of diesel particles. Long-term effects seen with whole diesel are not found or are much less evident in laboratory animals exposed to similar dilutions of diesel exhaust filtered to remove most of the PM. As a result, USEPA's reference concentration (RfC) for diesel exhaust is based entirely upon PM. In addition, the California Air Resources Board (CARB) has identified diesel exhaust PM as a "toxic air contaminant" under the state's air toxics program, based on the information available on cancer and non-cancer health effects. California limited its findings to diesel PM, as opposed to diesel exhaust.

Jet engines operate through use of turbines continuously injected with carefully controlled amounts of fuel. Basically, in a jet turbine engine, turbine blades suck air in at tremendous speeds, causing higher pressure on the inside of the turbine. The engine is so hot (up to 3,500 degrees Fahrenheit) that the fuel ignites in a constant flame. The thrust provides the huge force necessary to propel commercial airliners. The high temperatures and continuous fuel injection act to combust fuel more completely and efficiently than diesel engines. Burning of jet fuel in engines using modern turbine technology creates much less particulate matter than is created during diesel fuel combustion. The combination of different fuel compositions and combustion technologies result in exhausts which differ chemically and physically, and, as a result, toxicologically from diesel exhaust.

Relatively little is currently known about the actual amount of PM present in jet exhaust or especially about the toxicity of jet exhaust. The following is an excerpt from USEPA's 1999 document, *Evaluation of Air Pollutant Emissions from Subsonic Commercial Jet Aircraft*:

> PM emissions result from the incomplete combustion of fuel. High power operation, such as takeoff and climbout, produce the highest PM emission rates due to the high fuel consumption under those conditions. PM emission test data for aircraft engines are sparse, and engine-specific PM emission factors are available for only a few engine models.[25]

As a result, PM emission factors are not reported in the document. Estimates of PM emissions for use in this report were made using a variety of sources. No data were available for many types of engines and estimates were based on fuel consumption for similar engines in many cases.

Because of (a) differing fuels, (b) very different combustion processes in jet engines and diesel engines, and (c) to a lesser extent uncertainties in PM emissions from jet engines, extrapolation of PM emissions from diesel exhaust to jet exhaust is not considered appropriate or scientifically justifiable for the LAX HHRA. Accordingly, a January 2000 CARB Advisory Committee draft report on commercial airport activities states that, when assessing toxic impacts associated with particulate emissions from aircraft, it may not be appropriate to use the CalEPA Unit Risk Factor for diesel PM (3.0×10^{-4} µg/m^3).[26] Although PM from jet exhaust is not quantified in the HHRA, various investigations[27] have been performed which provide information about emission factors for other toxic air contaminants in jet exhaust. As a result, carcinogenic risks and hazard quotients are calculated in the HHRA for specific jet exhaust components with known emission factors (e.g., chlorinated dioxins, various PAHs, and 1,3-butadiene).

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[23] USEPA, Integrated Risk Information System (IRIS) Online Database, 2000.

[24] CalEPA, Non-cancer Chronic Reference Exposure Levels (RELs), Air Toxicology and Epidemiology Section, Draft for Public Review, 1997.

[25] USEPA, Evaluation of Air Pollutant Emissions from Subsonic Commercial Jet Aircraft, 1999.

[26] CalEPA, Non-cancer Chronic Reference Exposure Levels (RELs), Air Toxicology and Epidemiology Section, Draft for Public Review, 1997.

[27] Spicer et al., Chemical Composition and Photochemical Reactivity of Exhaust from Aircraft Turbine Engines, Annalues Geophysicae, May 25, 1994.

3.7 TAPs of Concern for Deposition onto Soils

TAPs in emissions for LAX may deposit onto soils. From soils, TAPs could theoretically be incidentally ingested, dermally contacted, or absorbed into garden vegetables. The potential for chemicals to accumulate in soil was therefore evaluated.

Screening of TAPs of concern for soil consisted of the following steps:

- Volatile chemicals were eliminated.
- Concentrations for TAPs in soil were estimated.
- Estimated TAP concentrations were compared to background concentrations.

Screening level deposition modeling was conducted using ISCST3, a USEPA-approved air dispersion model, to evaluate whether operations at LAX could result in substantial deposition onto soil, and, if so, which TAPs would be important to further evaluate. Air dispersion modeling used to evaluate TAPs of concern for soil are based on dated emissions estimates, unrealistic aircraft operational assumptions, and do not consider plume rise. All of these parameters will tend to overestimate deposition rates onto soil. The results of air dispersion modeling are therefore not appropriate for exposure estimates in the HHRA, but are conservative upper bound air concentrations suitable for screening of TAPs of concern for soil (Attachment B, *Screening Level Human Health Risk Assessment*). Refined modeling was conducted to support quantitative exposure estimates in the HHRA.

Background concentrations used in the analysis of deposition to soil were geometric mean concentrations and associated geometric standard deviations for the western United States, including California.[28] Using these values to define a distribution for possible background levels, the percentile for the estimated contribution from LAX emissions was calculated. Analysis of soil deposition suggests that estimated contributions from LAX emissions would make no measurable difference in expected background concentrations of metals, dioxins, or PAHs. A measurable, but small, contribution could be seen for arsenic on-airport in the areas of highest deposition. Resulting arsenic concentrations of soils would still be well within common background levels in the western U.S. Further, deposition is exaggerated because of the conservatism of the air-dispersion and deposition modeling. Therefore, no TAPs of concern were selected for soil, and soil-associated pathways were not evaluated in the HHRA.

The minimal predicted deposition of TAPs onto soils indicated that potential impacts to local surface water would also be minimal. TAP concentrations in sediments, from either direct deposition to surface water or from runoff from surrounding soils, would not exceed the negligible impacts predicted for soils. TAPs that deposit onto surface water and enter the dissolved phase would be rapidly carried away in stream flows and no long-term build up of dissolved concentrations would be possible. Deposition of TAPs to soils followed by runoff to surface water, and direct deposition into surface waters or watercourses, are predicted to be of minimal concern and were not further addressed in the risk assessment.

3.8 Summary of TAPs of Concern

TAPs of concern for emissions from LAX are listed in **Table 2**, Toxic Air Pollutants of Concern for LAX. No TAPs of concern were selected for deposition onto soils or into surface water.

4. EXPOSURE ASSESSMENT

In the exposure assessment, populations potentially exposed to TAPs associated with LAX operations were identified and chemical intakes estimated for individuals within these populations A combination of a receptor population and potential exposure pathways comprises an exposure scenario. Exposure scenarios for the LAX HHRA were selected to provide the most conservative, and therefore, protective, health impact assessment. By protecting the most highly exposed and sensitive populations, the general population is also protected.

Identification of potentially exposed populations is based on current and potential future land uses near LAX, and on exposure to TAPs via inhalation. Current land use near the airport consists of low- to medium-density residential housing immediately adjacent to the airport to the north in Playa del Rey and

[28] Shacklette and Boerngen, Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States, US Geological Survey Professional Paper 1270, 1984.

Westchester, and a mixture of low- and high-density residential housing to the south in El Segundo. High-density housing is found closest to the ends of runways to the east of the LAX north runways and also in Westchester. Residents at the ends of runways would be expected to experience the greatest impacts from jet exhaust because of winds that blow predominantly from west to east.

Certain subpopulations may be more sensitive or susceptible to negative health impacts caused by environmental contaminants than the population at large.[29] These critical subpopulations were also considered in the exposure assessment. During this evaluation, the following sensitive receptor locations were identified:

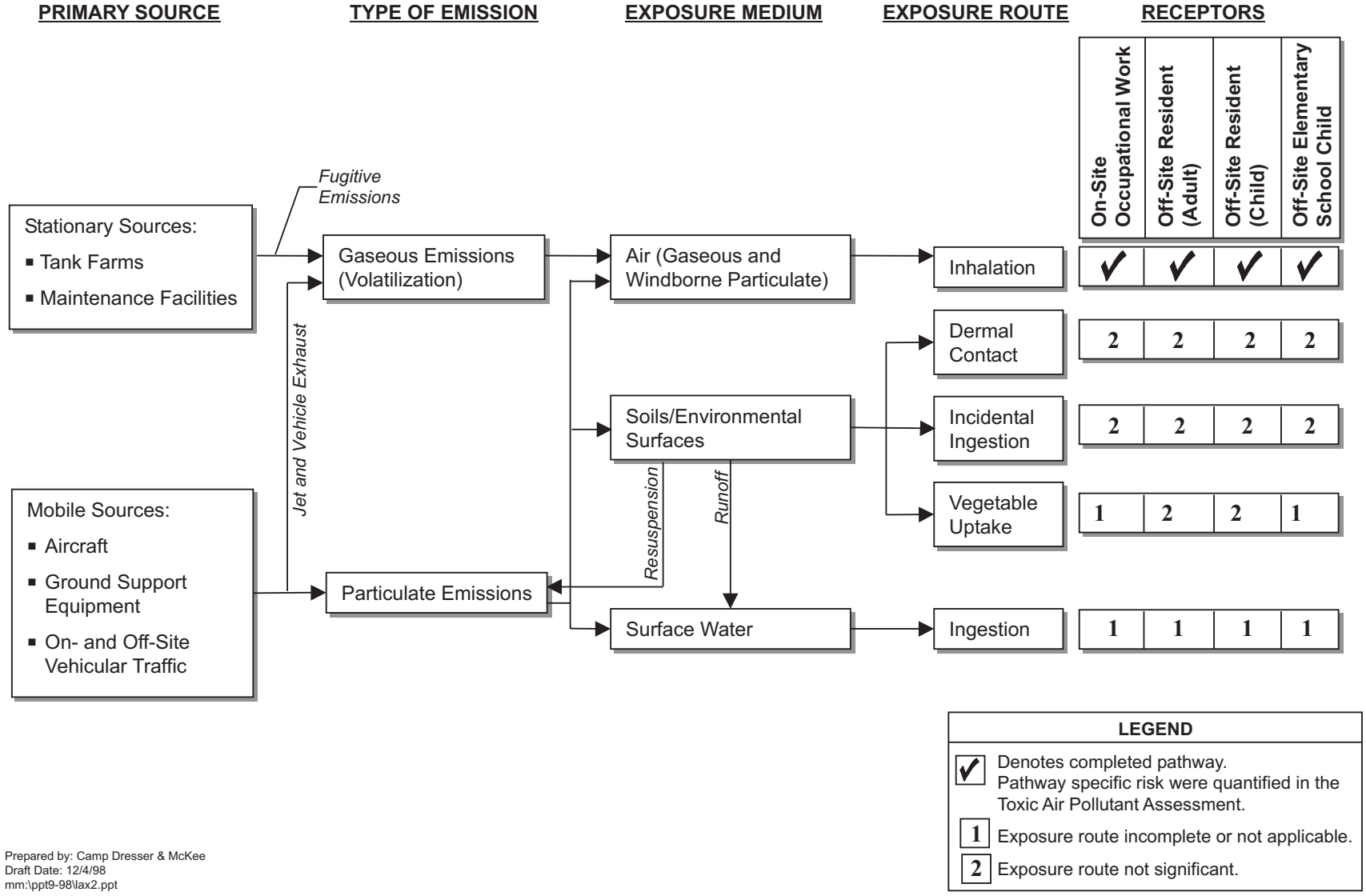
- Schools: School children include all students enrolled in kindergarten through high school. A survey was conducted of the study area in 1996, and 99 schools were identified. Of these, approximately 20 schools lie within one mile of the LAX fence line. Oak Street Elementary School and Escuela de Montessori were identified as the schools where the highest concentrations of TAPs released from LAX were predicted (see Section 6, *Risk Characterization*). Elementary school children are also the most critical population for evaluation of non-cancer health hazards for exposure to TAPs of concern for LAX operations. Elementary school children at the Oak Street School and Escuela de Montessori were, therefore, quantitatively evaluated for cancer risks and health hazards. Cancer risks are proportional to the duration of exposure. Thus, cancer risks to adult and child residents, exposed for many years, will be higher than for any school population. Protection of residents living adjacent to LAX for carcinogenic effects will also protect all school populations.
- Day care centers and preschools: Day care centers and preschools within the noise impact area for LAX were also identified. Forty-one preschool/day care centers were identified. Of these centers, 14 facilities are located within one mile of the LAX fence line. The center nearest the LAX fence line is St. John's Lutheran Child Development Center, at 16111 East Sycamore Avenue in El Segundo.
- Hospitals, nursing homes, and retirement communities: Patients and residents in hospitals, nursing homes, and retirement communities are critical subpopulations with possibly increased sensitivity to environmental contaminants. According to the 1990 census, 8 percent of the local population is in excess of 65 years of age in the area surrounding LAX. No hospitals are, however, located within one mile of LAX. The nearest hospital, Centinela Hospital, lies approximately 1.6 miles to the east.
- Residential areas with children: Children living in the immediate vicinity of the site or within the potential impact zones are probably more sensitive or susceptible to effects of many TAPs. The area surrounding LAX includes mixed use and residential communities. The 1990 census reported a population of 441,375 within an area subject to noise impacts from LAX. Of this population, 131,794 people were less than 16 years of age.

Of these several sensitive receptors, school children and children in residential areas are assessed quantitatively. Methods to estimate exposures and risks for these populations are well defined in guidance. Methods to separately assess populations in hospitals, nursing homes, and retirement communities have not been defined in guidance and methods for quantitative evaluation are not readily available. Instead, toxicity criteria (cancer slope factors and reference doses) are defined by CalEPA or USEPA to be protective for sensitive subpopulations of people. Thus, if protection based on these toxicity criteria is provided for the most heavily exposed people, sensitive subpopulations should also be protected. Children in day care centers and preschools are not separately evaluated because children in this age range are evaluated as residents living immediately adjacent to the airport. When these children are protected, children in day cares and preschools close to LAX, who spend only part of their day at the school in these locations, will also be protected.

4.1 Site Conceptual Exposure Model

Potential exposures associated with emissions from LAX are illustrated in the site conceptual exposure model (SCEM) in **Figure 2**, Conceptual Exposure Model for LAX Master Plan – Toxic Air Pollution Exposure Assessment. The SCEM provided a basis for identifying and evaluating pathways by which human receptors may be exposed to TAP emissions from LAX. Some of the exposure pathways depicted in the model either do not exist or are unlikely to contribute substantially to overall exposures. For example, no TAPs of concern were selected for deposition onto soil, and pathways associated with soil are not quantitatively evaluated.

[29] USEPA Office of Emergency and Remedial Response, Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual Interim Final, December 1989.



Prepared by: Camp Dresser & McKee
Draft Date: 12/4/98
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Los Angeles International Airport Master Plan

Conceptual Exposure Model for LAX Master Plan - Toxic Air Exposure Assessment

Figure 2

Analyses of populations and exposure pathways identified the following scenarios as appropriate for conservative assessment of human health risks.

- On-airport worker
- Off-airport elementary school child
- Off-airport residents (adults and young children)

Each receptor represents a unique population and set of exposure conditions. As a whole, they cover a range of exposure scenarios for the potentially most affected human receptors. Evaluation of these scenarios will also be protective of others that may exist at the site.

Workers at LAX may represent the population for which exposures to TAPs may be greatest. LAX workers, especially baggage handlers at the gates and on the aprons, spend large amounts of their time at work in areas where exhaust from jet engines, GSE, and other sources may reach their highest concentrations.

Children may attend school at locations close to LAX where impacts may be greater than those at their residences. Further, children may be more susceptible to air toxics because of relatively high inhalation rates and low body weights. School children were selected for quantitative evaluation over other sensitive receptor populations, because evaluation of other populations would either be redundant or quantitative methods for evaluation of these populations are not available. For example, day care or nursery school children could live at the "fence line" and would therefore be adequately represented by the young child resident. Methods for separate quantitative assessment of nursing home residents and many other potentially sensitive receptors are not available. These populations are, however, evaluated qualitatively in the uncertainty assessment.

Adult residents living at locations near the airport, especially in areas downwind, (i.e., east of the east end of the runways) could be exposed to TAPs from LAX, possibly for long periods of time. Long periods of exposure are appropriate for evaluating carcinogenic risks, because exposures to carcinogens are averaged over an entire lifetime. In this assessment, cancer risks were estimated for people who grow up and spend most of their adult life near the airport. Children are separately evaluated for non-cancer health hazards because non-cancer impacts are evaluated on the basis of exceeding a threshold of exposure. Exposures for children are likely to be higher than those for adults because child body weights are lower and chemical intakes rates relatively high.

All populations are evaluated for exposure to volatile organic compounds (VOCs), metals (including arsenic), PAHs, and TCDD via inhalation only.

Potential exposures are summarized in **Table 3**, Scenarios Evaluated in the HHRA.

Table 3

Scenarios Evaluated in the HHRA

Receptor	Pathways
On-Airport Worker	Inhalation of TAPs
Off-Airport Adult Resident	Inhalation of TAPs
Off-Airport Child Resident	Inhalation of TAPs
Off-Airport School Child	Inhalation of TAPs

Source: Camp Dresser & McKee Inc., 2000.

4.2 Exposure Assumptions and Methods Used to Quantify Exposures

Each exposure scenario is has a unique set of exposure parameters (inhalation rates, exposure frequencies, body weights, etc.). These parameters are discussed below for each scenario.

4.2.1 Exposure Assumptions

4.2.1.1 On-Airport Worker

The on-airport worker was assumed to be in contact with TAPs related to LAX operations during a normal workday. Because worker exposures are occupational and not incidental, workers are assessed appropriately through comparison of the maximum average air concentrations of TAPs (conservative predictor of exposure) to thresholds of significance determined for workers by relevant governing bodies. Permissible Exposure Limits – Time-Weighted Average (PEL-TWA) are air concentrations for chemicals adopted by CalOSHA[30] to represent maximum concentrations (8-hour time-weighted average) to which workers may be repeatedly exposed during business hours without developing adverse health effects. Occupational exposures are thus assessed by comparing maximum 8-hour concentrations of TAPs near gates and aprons, estimated through air dispersion modeling, with PEL-TWAs. Under ACGIH guidelines, if TAP concentrations are below PEL-TWAs, health impacts are unlikely for LAX workers.

4.2.1.2 Off-Airport Adult and Child Residents and Elementary School Students

To estimate potential cancer risks and the potential for adverse non-cancer health hazards for off-airport residential receptors and elementary school children, chronic daily intakes (CDIs) for the inhalation pathway are estimated as follows:[31]

$$CDI = (C \times IR \times EF \times ED) / (BW \times AT)$$

Where:

	CDI	=	chronic daily intake (mg/kg body weight/day)
	C	=	chemical concentration in air (mg/m^3)
	IR	=	inhalation rate with exposure medium (m^3/day)
	EF	=	exposure frequency and duration (days/year)
	ED	=	exposure duration (years)
	BW	=	body weight (kg)

AT=average time; e.g., the period over which exposure is averaged (days)

Two types of CDI are calculated. Lifetime Average Daily Dose (LADD) is calculated for exposure to carcinogens. Cancer risk is thought to be cumulative over a lifetime, and chemical exposures are averaged for an average lifetime instead of over the duration of exposure. Average Daily Dose (ADD) is calculated for exposure to non-carcinogens and for carcinogens with significant non-cancer health effects. Non-cancer health impacts are more closely related to average daily intake than cumulative exposure, and chronic intakes are evaluated only over the duration of the exposure.

Exposure parameters used to calculate LADD and ADD for each of these pathways are summarized in **Table 4**, Parameters Used to Estimate Exposures to TAPs of Concern. Exposure parameters are based on the CalEPA Supplemental Guidance for Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities,[32] USEPA Exposure Factors Handbook,[33] and California Air Pollution Control Officers Association (CAPCOA) Air Toxics Assessment Manual.[34]

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[30] CalOSHA (California Occupational Safety and Health Administration). 2000. Table AC-1, Permissible Exposure Limits for Chemical Contaminants. http:/www.dir.ca.gov/title8/5155a.htm.

[31] USEPA Office of Emergency and Remedial Response, Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual Interim Final, December 1989.

[32] California Environmental Protection Agency (CalEPA), CalEPA Supplemental Guidance for Human Heath Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities, 1993.

[33] USEPA, Exposure Factors Handbook, ORD, EPA/600/P-95/002Bc, Review Draft, 1996.

[34] California Air Pollution Control Officers Association, Air Toxics Assessment Manual, Volume 1 Toxic Air Pollutant Source Assessment Manual for California Air Pollution Control Districts and Applicants for Air Pollution Control District Permits, October 1987.

Table 4

Parameters Used To Estimate Exposures to TAPs of Concern

Exposure Pathway	Off-Airport Receptors		
Inhalation of Particulates and Gases	Offsite Resident		Off-Site Elementary School Child
	Adult	Child	
IR (m^3/d)	20	15	6
EF (d/yr)	350	350	200
ED (yr)	30	6	6
BW (kg)	70	15	40
AT Non-cancer (days)	10,950	2,190	2,190
AT Cancer (days)	25,550	25,550	25,550

Acronyms used in this table:
IR =Average inhalation rate
EF =Exposure frequency
ED =Exposure duration
BW =Body weight
AT =Averaging time

Source: Camp Dresser & McKee Inc., 2000.

4.2.2 Exposure Concentrations/Air Dispersion Modeling

The CDM team prepared a written plan for developing the toxic air pollutant emissions inventories and for performing the toxic air pollutant dispersion modeling for the LAX Master Plan (see Attachment F, *Air Quality Modeling Protocol for Toxic Air Pollutants*).

Exposure concentrations in air were annual average concentrations estimated by ISC3 modeling. Exposure concentrations were calculated for a modeling grid that becomes coarser with increasing distance from sources. These concentrations are used in the geographic presentation of risks (see Section 6, *Risk Characterization*). In addition, air concentrations were modeled for locations that will represent maximally exposed receptors (MEI) for the four scenarios defined in **Table 3**, Scenarios Evaluated in the HHRA. These locations were identified as the school and residence locations where the highest off-airport concentrations were predicted by the ISC3 modeling.

4.2.3 Definition of the Study Area

An initial prediction of the extent of the study area to be used in the geographic description of risks was made in the screening assessment and the preliminary study area was refined during the final HHRA. The study area was defined initially by considering the area where impacts from chemicals associated with LAX were estimated to be equal to or greater than one-half the background concentration. Urban background concentrations of 1,3-butadiene, benzene, and toluene were estimated from recent data.[35] ISC3 modeling was performed using emissions for the No Action/No Project Alternative for 2015 to estimate annual average concentrations near the airport for these three TAPs. Isolines of concentrations corresponding to one-half the background concentrations of the above TAPs were then plotted. In all three cases, isopleths representing a concentration of half of the expected urban background, stayed within the LAX boundary. The results of the preliminary evaluation suggested that concentrations of TAPs from LAX would merge with background relatively quickly in areas outside the LAX boundary. Results also indicated an east-west orientation of the isopleths.

An elliptical area extending 2 km (1.25 mi.) north and south, and 4 km (2.5 mi.) east of the current LAX fence line for the No Action/No Project Alternative was selected as the preliminary study area in the screening level assessment (Attachment B, *Screening Level Human Health Risk Assessment)*. During the risk characterization, it became apparent that the preliminary study area would not include all areas in which cancer risk estimates exceed 10 in one million for the No Action/No Project Alternative. The study area was therefore enlarged to capture all risks above 10 in one million, and a substantial additional area

[35] California Environmental Protection Agency Office of Environmental Health Hazard Assessment (OEHHA) Risk Assessment Advisory Committee, A Review of the California Environmental Protection Agency's Risk Assessment Practices, Policies, and Guidelines. Appendix B, 1996.

east of the airport. The final study area is shown in **Figure 3**, Definition of Study Area for the Human Health Risk Assessment for Releases of TAPs during LAX operations.

5. TOXICITY ASSESSMENT

The toxicity assessment evaluates potential human health effects from exposure to TAPs related to aircraft and airport operations at LAX. Potential adverse effects from exposure to such pollutants include both carcinogenic and non-carcinogenic health effects. This section presents toxicity criteria for all TAPs of concern. A detailed discussion regarding development of these criteria is provided in Attachment B, *Screening Level Human Health Risk Assessment*. Toxicity profiles for all TAPs of concern are provided in Attachment C, *Toxicological Profiles*.

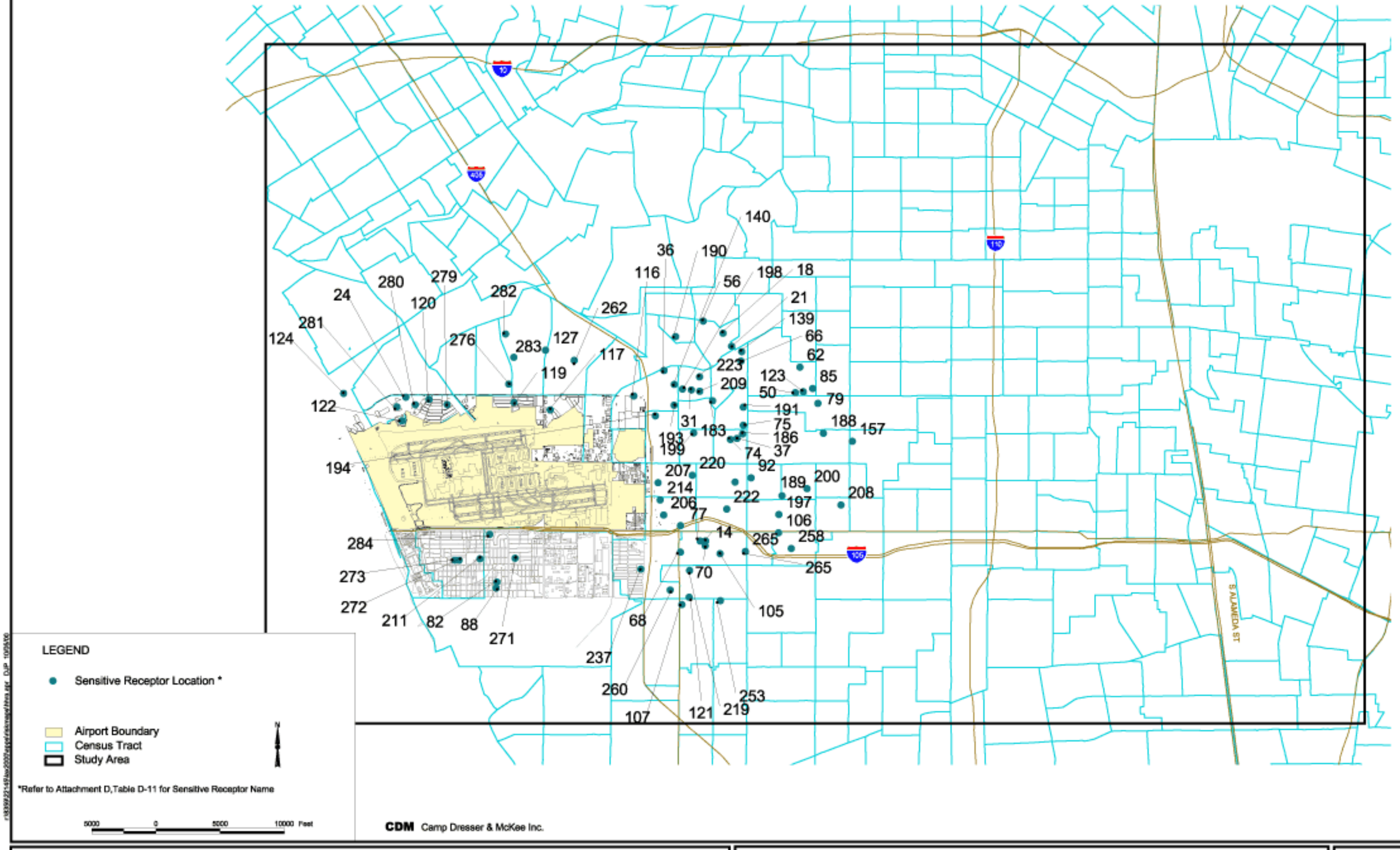
The primary sources of toxicity information used in this assessment were CalEPA Office of Environmental Health Hazard Assessment (OEHHA) Cancer Potency Factors, USEPA's IRIS, Health Effects Assessment Summary Tables (HEAST), Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles, USEPA criteria documents, and occupational standards from ACGIH.[36]

5.1 Cancer Slope Factors

Toxicity criteria for carcinogens are slope factors expressed as per milligram per kilogram-day $(mg/kg\text{-}day)^{-1}$. The cancer slope factor (CSF) describes the increase in an individual's risk of developing cancer over a 70-year lifetime per unit of exposure where exposure is expressed as mg/kg-day. CSFs for carcinogenic TAPs of concern for the LAX Master Plan are listed in **Table 5**, Cancer Slope Factors. Cancer slope factors from USEPA were used only if no criterion from CalEPA OEHHA was available.[37]

[36] American Conference of Governmental Industrial Hygienists (ACGIH), Documentation of the Threshold Limit Values and Biological Exposure Indices, 8th ed., Cincinnati, Ohio, 1998.

[37] California Environmental Protection Agency Office of Environmental Health Hazard Assessment (OEHHA) Risk Assessment Advisory Committee, A Review of the California Environmental Protection Agency's Risk Assessment Practices, Policies, and Guidelines, Appendix B, 1996.



Los Angeles International Airport Master Plan	**Definition of Study Area for the Human Health Risk Assessment for Releases of TAPs During LAX Operations**	Figure 3

Table 5

Cancer Slope Factors

TAP of Concern	Oral Cancer Slope Factor [(mg/kg/day)$^{-1}$]^{1}	Inhalation Cancer Slope Factor [(mg/kg/day)$^{-1}$]	Tumor Site		Cancer Classification^{2}
			Oral	Inhalation	
Organic					
Acetaldehyde	NA	0.00945	NA	Nasal, Larynx	B2
Acrolein	NA	NA	NA	NA	C
Benzene	0.029[1]	0.102	NA	Blood	A
1,3-Butadiene	NA	0.0595	NA	Reproductive Sys., Blood, Lung, GI	B2
Formaldehyde	NA	0.021	NA	Respiratory	B1
2,3,7,8 TCDD Equivalents	156,000	133,000	GI, Immune System, Reproductive System, Kidney	GI, Immune System, Reproductive System, Kidney	A
Polycyclic Aromatic Hydrocarbons (PAHs)					
Benzo(a)anthracene	1.2	0.39	NA	NA	B2
Benzo(b)fluoranthene	1.2	0.39	NA	NA	B2
Benzo(k)fluoranthene	1.2	0.39	NA	NA	B2
Benzo(a)pyrene	12	3.9	GI	GI, Respiratory	B2
Chrysene	0.12	0.039	NA	NA	B2
Dibenz(a,h)anthracene	4.1	4.2	Respiratory	NA	B2
Indeno(1,2,3-cd)pyrene	1.2	0.39	NA	NA	B2
Diesel					
Diesel Particulates	NA	1.1	NA	Lung	D
Inorganic					
Arsenic	1.5[1]	11.6	Skin, Lung	Respiratory System	A
Beryllium	4.3[1]	7.0[4]	NA	Lung	B1
Cadmium	NA	14.7	NA	Respiratory System	B1
Chromium VI	42	525	NA	Lung	A
Manganese	NA	NA	NA	NA	D

Notes: NA - Not available

GI - Gastrointestinal System

All Toxicity Criteria from CalEPA Office of Environmental and Human Health Assessment, Cancer Potency Factors, 1994, except as noted

1 USEPA, Integrated Risk Information System (IRIS) Online Database, 1998.

2 Carcinogen classifications identify the confidence USEPA has in evidence for carcinogenicity for a given chemical, where Class A is a known human carcinogen and Class E is a known non-carcinogen. Definitions are provided in detail in Attachment B, *Screening Level Human Health Risk Assessment*, Section 5.1.1, *Evidence of Carcinogenity*.

3 For nickel in refinery dust

4 Beryllium oxide value

Cancer Classification
Group A – Human Carcinogen
Group B (B1 and B2) – Probable Human Carcinogen
Group C – Possible Human Carcinogen
Group D – Not classified

Source: Camp Dresser & McKee Inc., 1998.

Further, CSFs are developed for both inhalation and oral exposure for many chemicals. Oral CSFs were used only if no inhalation CSF was available from either CalEPA or USEPA (i.e., inhalation slope factors took precedence over oral factors regardless of source).

5.2 Non-Cancer Reference Doses

Reference doses (RfDs) are toxicity values developed by USEPA for chemicals exhibiting non-carcinogenic effects, or for carcinogens that also have important non-cancer effects. CalEPA has not separately developed RfDs. The RfD is intended as an estimate of the daily exposure to a chemical that would not cause adverse effects even if the exposure occurs continuously over a lifetime. RfDs are

presented in units of mg/kg-day for comparison with estimated chronic daily intake into the body. Intakes that are less than the RfD are not likely to cause adverse health effects. Chronic daily intakes that are greater than the RfD indicate a possibility for adverse effects. RfDs are developed for both inhalation and oral exposure for many chemicals. For this HHRA, oral RfDs were used only if no inhalation RfD was available. RfDs for chemicals of potential concern (COPCs) for the LAX Master Plan are presented in **Table 6**, Toxicity Criteria for Systemic Toxicants.

5.3 California Reference Exposure Levels (RELs)

CalEPA has proposed RELs, which are analogous to USEPA Reference Concentrations (RfCs) (i.e., inhalation RfDs expressed in units of mg/m^3). RfDs for inhalation are generally calculated from RfCs. RfCs are derived in a fashion analogous to that for oral RfDs, but are expressed in units of mg/m^3. RfCs are intended as estimates of ambient air concentrations that could be present for a lifetime without causing adverse effects. An RfC is converted to an inhalation RfD by multiplying by inhalation rate and dividing by body weight. Standard CalEPA and USEPA parameters, 20 cubic meters per day (m^3/day) for inhalation rate and 70 kilograms for body weight, were used for these calculations. Currently, RELs are in review and are subject to change. For this reason, potential impacts from future regulatory adoption of the RELs are considered only in the uncertainties section of the HHRA (Section 7, *Uncertainties*). RELs, converted to units of mg/kg-day, for COPCs for the LAX Master Plan are presented in **Table 6**, Toxicity Criteria for Systemic Toxicants.

6. RISK CHARACTERIZATION

In the risk characterization, information developed in the exposure and toxicity assessments was combined to generate risk estimates for quantitatively evaluated populations. Separate calculations were performed to estimate both cancer risks and non-cancer health effects from exposure to chemicals with non-cancer toxicity. Cumulative impacts were evaluated, following state and federal guidance, by adding cancer risks for exposure to all carcinogenic TAPs, and by adding health hazards from all non-carcinogenic TAPs that affect the same target organ or tissue. Target organs and tissues for chronic exposure to TAPs are provided in Attachment B, *Screening Level Human Health Risk Assessment*, Section 5, *Toxicity Assessment*.

Risk evaluations were conducted for 2005 and 2015 and for pre- and post mitigation conditions. Year 2005 was chosen as a reasonable interim date during implementation of the LAX Master Plan where human health impacts during construction could be evaluated. 2015 is the year projected for completion of the LAX Master Plan and is thus the first year when full operations are expected after implementation.

Table 6

Toxicity Criteria for Systemic Toxicants

TAP of Concern	USEPA Chronic Oral RfD (mg/kg-day)[1]	USEPA Chronic Inhalation RfD (mg/kg-day)[1]	Proposed CalEPA Chronic Inhalation RfD[2] (mg/kg-day)	Target Organ		Uncertainty Factor		
				Oral	Inhalation	Oral	Inhalation (USEPA RfD)	Inhalation (CalEPA RfD)
Organics								
1,3-Butadiene	NA	NA	$2.3x10^{-3}$	NA	Reproductive System	NA	NA	300
Acetaldehyde	NA	$2.57x10^{-3}$	NA	NA	Nasal	NA	1,000	NA
Acrolein	$2x10^{-2}$ (3)	$5.71x10^{-6}$	NA	NA	Nasal	NA	1,000	NA
Benzene	NA	$1.71 x^{-3}$ (3)	$1.7x10^{-2}$	NA	NA	NA	NA	NA
Formaldehyde	$2x10^{-1}$	NA	$5.7x10^{-4}$	Body Weight	NA	100	NA	NA
Hexane	$6x10^{-2}$ (2)	$5.7x10^{-2}$	NA	NA	CNS	10,000	300	NA
Naphthalene	$2x10^{-2}$	$8.57x10^{-4}$	$2.6x10^{-3}$	Body Weight	Resp. System, Blood	3,000	3,000	1,000
Toluene	$2x10^{-1}$	$1.1x10^{-1}$	NA	Liver, Kidney	CNS	1,000	300	NA
Diesel								
Diesel Particulates	NA	$1.43x10^{-3}$	NA	NA	Lung	NA	30	NA
Xylene	$2x10^{0}$	NA	$5.7x10^{-2}$	Body Weight	CNS, Resp.System	100	NA	100
Inorganics								
Arsenic	$3x10^{-4}$	NA	$8.57x10^{-6}$	Skin	NA	3	NA	3
Beryllium	$2x10^{-3}$	$5.7x10^{-6}$	$2.9x10^{-7}$	GI	Resp. System	300	10	300
Cadmium	$1x10^{-3}$ (Food)	$5.7x10^{-5}$ (5)	$2.9x10^{-6}$	Kidney	Kidney, Resp. System	10	NA	NA
Chromium (VI)	$3x10^{-3}$	$2.86x10^{-5}$	$2.3x10^{-7}$	NA	NA	300	NA	300
Copper	$4x10^{-2}$ (4)	NA	$5.7x10^{-6}$	GI	Resp. System	NA	NA	100
Manganese	$1.4x10^{-1}$(Food)	$1.4x10^{-5}$	NA	CNS	CNS	1	1,000	NA
Nickel	$2x10^{-2}$	NA	$1.4x10^{-5}$	Body, Organ Weight	Lung	300	NA	30
Selenium	$5x10^{-3}$	NA	$2.3x10^{-5}$	NA	NA	3	NA	3,000
Zinc	$3x10^{-1}$	NA	$2.6x10^{-4}$	Blood	NA	3	NA	100

NA – Not Available
CNS – Central Nervous System
GI – Gastrointestinal System
Resp – Respiratory System

1 Calculated from proposed CalEPA Reference Exposure Levels (RELs)
2 From the Integrated Risk Information System (IRIS) unless otherwise noted
3 from Health Effects Assessment Tables (HEAST)
4 National Center for Environmental Assessment (NCEA) Regional Support Provisional Value
5 Withdrawn from IRIS

Source: Camp Dresser & McKee Inc., 2000.

ISC3 air dispersion modeling for post-mitigation conditions was performed assuming the following Mitigation Measures:

- Reduced engine taxi/queue
- GSE replacement
- Cargo ramp incentives
- Flyaways/Restructured Short-Term Parking Payment and Fee Schedules/Traffic Mitigation

Cancer risks and non-cancer health effects estimates for post-mitigation conditions were generated for comparisons with predicted impacts under the No Action/No Project Alternative and Alternatives A, B, and C without mitigation. These comparisons were conducted to evaluate the potential for reduction of human health risks and hazards using reasonable and implementable Mitigation Measures.

6.1 Calculation of Risk Estimates

Cancer risks are estimated by multiplying exposure estimates for carcinogenic chemicals by corresponding cancer slope factors. The result is a risk estimate expressed as the odds of developing cancer. Commonly, risks (or odds) of developing cancer of one to ten in one million (1×10^{-6} to 10×10^{-5}) or less are considered *de minimis*. Higher risks may be deemed unacceptable in some instances. In such instances, mitigation of risks may be considered necessary. Estimation of chemical exposure to carcinogens is discussed in Section 4.2.1, *Exposure Assumptions*. Development of cancer slope factors is discussed in Section 5.1, *Cancer Slope Factors*.

Non-cancer risk estimates are calculated by dividing exposure estimates (Section 4.2.1, *Exposure Assumptions*) by reference doses. As discussed in Section 5.2, *Non-Cancer Reference Doses*, reference doses are estimates of highest exposure levels that would not cause adverse health effects even if exposures continue over a lifetime. The ratio of exposure to reference dose is termed the hazard quotient (HQ). A HQ greater than one indicates an exposure greater than that considered safe. Risks or odds of adverse effects cannot be estimated using references doses. However, because reference doses are developed in a conservative fashion, HQs only slightly higher than one are generally accepted as being associated with low risks (or even no risk) of adverse effects, and that potential for adverse effects increases as the HQ gets larger.

Impacts of exposure to multiple chemicals are accounted for by adding cancer risk estimates for exposure to all carcinogenic chemicals, and by adding estimated HQs for non-carcinogenic chemicals that affect the same target organ or tissue in the body. Addition of HQs for TAPs that produce effects in similar organs and tissues results in a Hazard Index (HI) that reflects possible cumulative hazards. Several TAPs have effects on the respiratory system including acetaldehyde, acrolein, formaldehyde, xylenes, and diesel particulates. Cumulative exposure to these chemicals accounts for essentially all potential non-cancer hazards. Therefore, the only HI calculated is that for the respiratory system.

6.2 Presentation of Risk Estimates

Risk estimates are presented in several different ways to provide a comprehensive illustration of the relative health impacts among the build alternatives, and between the build alternatives and the No Action/No Project Alternative.

The risk characterization includes the following risk presentations:

- Comparison of on-airport air concentrations with OSHA standards for workers
- Incremental cancer risks and non-cancer hazards for MEI – adult and child residents and school children
- Incremental cancer risks and non-cancer hazards depicted geographically (using contours)
- Cumulative impacts associated with the build alternatives and the No Action/No Project Alternative

Exposure of workers at the airport would be subject to OSHA regulations. On-airport workers are therefore evaluated for exposure to TAPs by comparing estimated air concentrations on-airport with OSHA standards for TAPs in the air (see Section 4.2.1.1, *On-Airport Worker*).

MEI calculations represent maximum possible health threats to the community near LAX. MEI estimates were generated for future residents and school children in locations of maximum incremental annual

average TAP concentrations predicted by ISC3 air dispersion modeling (Section 4.2.3, *Definition of the Study Area*).

For the geographic depiction of risks, estimates of risk and hazard were calculated for each grid node in the air dispersion modeling domain (Section 4.2.3, *Definition of the Study Area*), and these estimates were used to generate contours of risks and hazards for communities surrounding LAX. This presentation of risks allows visualization of the area of potential impact and provides a frame of reference for interpreting MEI calculations.

The last type of risk evaluation consists of a comparison of "background" risks from all sources within the South Coast Air Basin to those estimated to be associated with operations at LAX. This analysis provides a means to estimate the incremental impact of emission of TAPs from LAX on communities surrounding the airport. The MATES-II study,[38] which provides a general evaluation of cancer risks associated with TAPs within the South Coast Air Basin, is used in this comparison. Incremental impacts on human health are only evaluated for carcinogenic TAPs, because the MATES-II study does not report estimates for non-cancer effects.

Spreadsheets used in risk calculations are presented in Attachment D, *Risk Calculations for Maximally Exposed Resident and School Child*. Spreadsheets used in the comparison with background risks are presented in Attachment E, *Risks Associated with the Build Alternatives Compared to Background*.

6.3 Risk Estimates for Pre-Mitigation Conditions for Horizon Year 2005

For 2005, the No Action/No Project Alternative and Alternative A, B and C were quantitatively evaluated. Emissions for all alternatives were predicted to be so similar that a single modeling run could be used to predict air quality impacts of TAP releases for all three scenarios.

6.3.1 Comparison of On-Airport Air Concentrations with OSHA Standards for Workers

Workers are evaluated by comparing estimated annual air concentrations of TAPs for the different alternatives to eight-hour PEL-TWAs. Estimated on-airport air concentrations and PEL-TWAs for TAPs of concern for LAX are presented in **Table 7**, Comparison of CalOSHA Permissible Exposure Limits (PEL-TWAs) to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2005 Pre-Mitigation Conditions.

Estimated on-airport concentrations for the No Action/No Project Alternative and the build alternatives are not greatly different for most TAPs, but locations where maximum concentrations are predicted on the airport vary (**Figure 4**, Locations for Maximally Exposed Workers for Horizon Year 2005, Pre-Mitigation Conditions and **Table 8**, Legend for Figure 4, Locations for Maximally Exposed Workers for Horizon Year 2005 Pre-Mitigation Conditions). Estimated concentrations for all TAPs except arsenic, cadmium, and manganese are higher for the No Action/No Project Alternative than the build alternatives. Higher concentrations associated with the No Action/No Project Alternative are due to increased motor vehicle traffic in the absence of airport expansion as defined by the build alternatives.

Estimated maximum 8-hour air concentrations for the build alternatives and the No Action/No Project Alternative, are well below PEL-TWAs for all TAPs. This result suggests that air concentrations from airport emissions with or without implementation of the LAX Master Plan will not exceed those considered "acceptable" by CalOSHA.

6.3.2 Risks for Maximally Exposed Individuals (MEI)

MEI risks and health hazards were calculated for adult residents, resident children ages 0 to 6 years, and for elementary-aged school children at off-airport locations where maximum air concentrations for TAPs were predicted. Generally, predicted off-airport TAP concentrations were similar for the build alternatives and the No Action/No Project Alternative, but some minor differences in the locations of maximum concentrations were observed (**Figure 5**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2005, Pre-Mitigation Conditions).

[38] SCAQMD, Multiple Air Toxics Exposure Study in the South Coast Air Basin (MATES-II), November 1999.

Table 7

Comparison of CalOSHA Permissible Exposure Limits (PEL-TWA) to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2005 Pre-Mitigation Conditions

	Alternative (mg/m^3)		
TAP[1]	No Action/ No Project	Alternatives A, B, and C	CalOSHA PEL-TWA (mg/m^3)[2]
Acetaldehyde	$5.5x10^{-3}$	$4x10^{-3}$	$1.8x10^{2}$
Acrolein	$1.4x10^{-3}$	$1.1x10^{-3}$	$2.5x10^{-1}$
Benzene	$1.3x10^{-2}$	$8.4x10^{-3}$	$3.2x10^{-1}$ [3]
1,3-Butadiene	$3.9x10^{-3}$	$2.7x10^{-3}$	$2.2x10^{0}$
Diesel Particulates	$1.1x10^{-2}$	$4.7x10^{-3}$	NA
Formaldehyde	$1.7x10^{-2}$	$1.3x10^{-2}$	$3.7x10^{-1}$ [3]
Naphthalene	$1.9x10^{-3}$	$1.5x10^{-3}$	$5.0x10^{1}$
PAHs as Benzo(a) pyrene equivalents	$1.2x10^{-6}$	$9.1x10^{-7}$	NA
TCDD equivalents	$1.2x10^{-9}$	$7.3x10^{-10}$	NA
Xylenes	$2.8x10^{-2}$	$1.7x10^{-2}$	$4.34x10^{2}$
Arsenic	$1.1x10^{-6}$	$1.2x10^{-6}$	$1.0x10^{-2}$
Beryllium	$1.6x10^{-7}$	$1x10^{-7}$	$2.0x10^{-3}$
Cadmium	$3.1x10^{-6}$	$3.2x10^{-6}$	$5.0x10^{-3}$
Chromium (as Cr(VI))	$3.2x10^{-8}$	$2x10^{-8}$	$5.0x10^{-2}$
Manganese	$6.1x10^{-5}$	$6.6x10^{-5}$	$5.0x10^{0}$

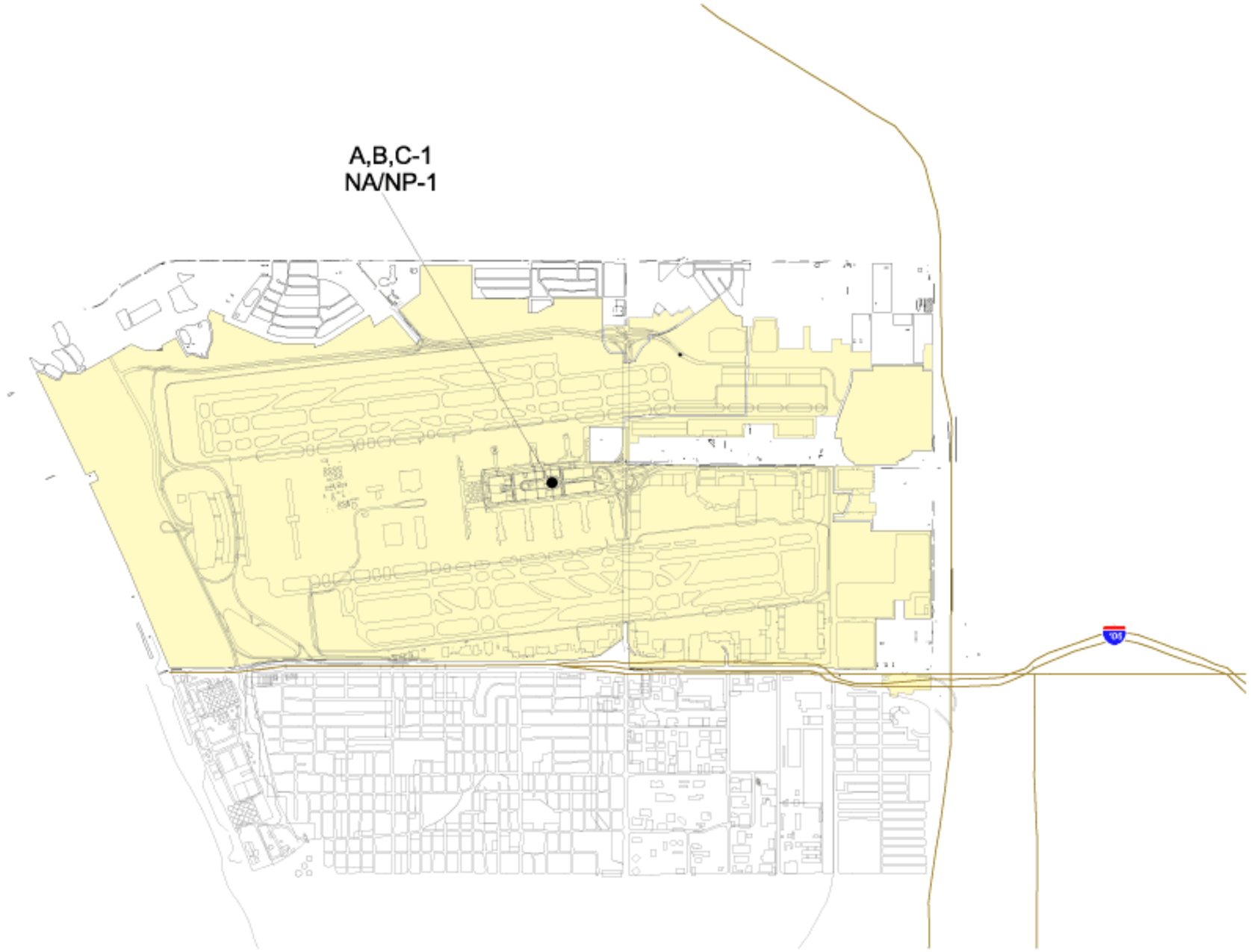
NA = Not Available

[1] All TAPs for which PEL-TWAs are available are listed. PEL-TWAs are not available for diesel exhaust, chromium VI and PAHs other than naphthalene.

[2] CalOSHA (California Occupational Safety and Health Administration). 2000. Table AC-1, Permissible Exposure Limits for Chemical Contaminants. http:\www.dir.ca.gov/title8/5155a.htm.

[3] CalOSHA Value not available; value is from American Conference of Governmental Industrial Hygienists (ACGIH), Documentation of the Threshold Limit Values and Biological Exposure Indices, 8th ed., Cincinnati, Ohio, 1998.

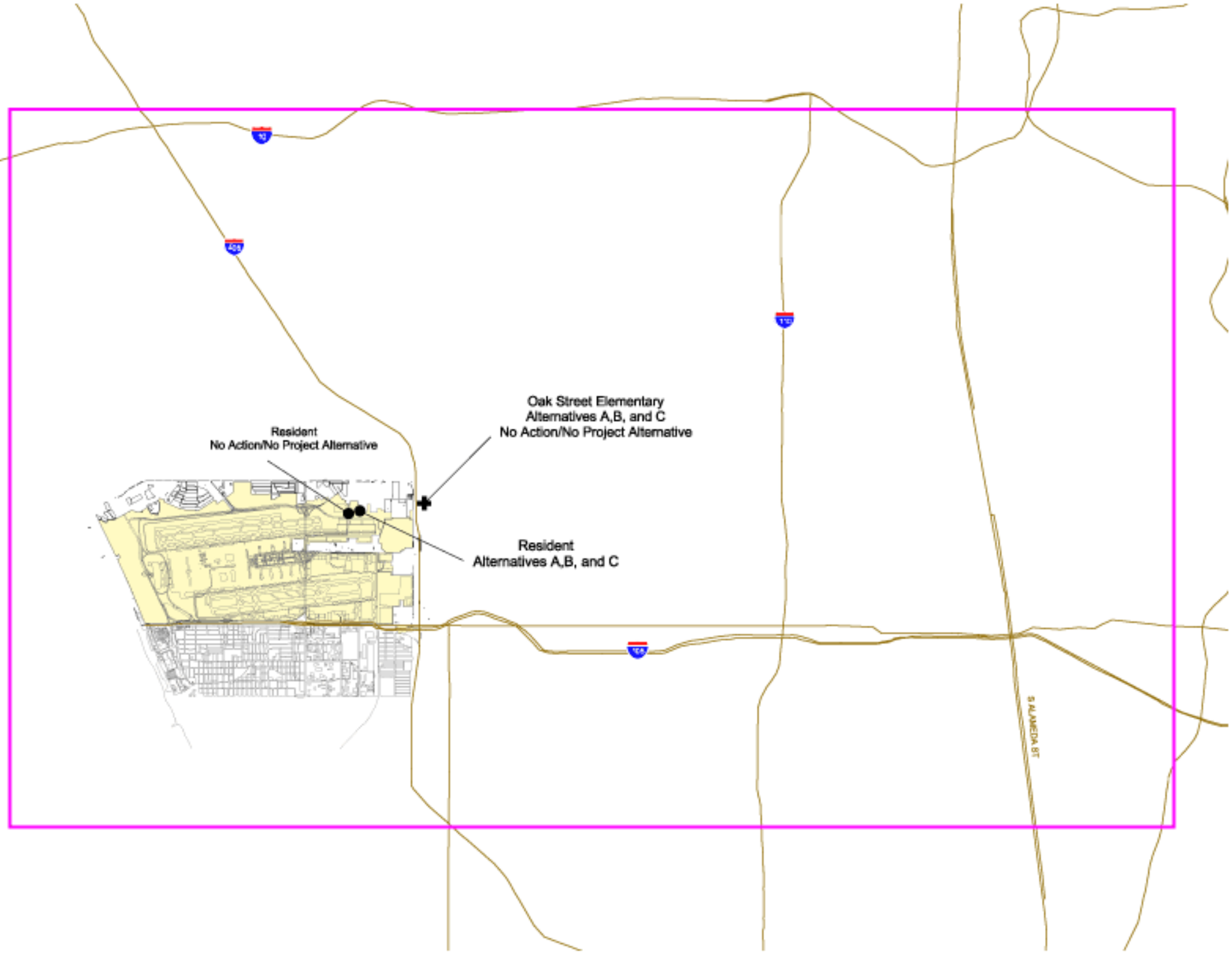
Source: Camp Dresser & McKee Inc., 2000.



Los Angeles International Airport Master Plan

Locations for Maximally Exposed Workers for Horizon Year 2005 Pre-Mitigation Conditions

Figure 4



Note: Airport Boundary Footprint shown is for Alternative C.

CDM Camp Dresser & McKee Inc.

Los Angeles International Airport Master Plan	**Locations for Maximally Exposed Residents and School Children for Horizon Year 2005 Pre-Mitigation Conditions**	Figure 5

Table 8

Legend for Figure 4, Locations for Maximally Exposed Workers for Horizon Year 2005 Pre-Mitigation Conditions

Label	X_stateplane (ft)	Y_stateplane (ft)	Model_X (m)	Model_Y (m)
No Action/No Project, NA/NP-1	6439597.00	1802295.00	0	0
Alternatives A, B, and C, ABC-1	6439597.00	1802295.00	0	0
	No Action/No Project		**Alternatives A, B, and C**	
TAP	**Label**	**Maximum On-Site Concentration (ng/m^3)**	**Label**	**Maximum On-Site Concentration (ng/m^3)**
Acetaldehyde	NA/NP-1	629	ABC-1	458
Acrolein	NA/NP-1	155	ABC-1	129
Benzene	NA/NP-1	1,500	ABC-1	965
1,3-Butadiene	NA/NP-1	447	ABC-1	306
Formaldehyde	NA/NP-1	1,900	ABC-1	1,420
Xylene (total)	NA/NP-1	3,170	ABC-1	1,940
TCDD equivalents	NA/NP-1	0.000136	ABC-1	0.0000834
PAHs as Benzo(a)pyrene equivalents	NA/NP-1	0.134	ABC-1	0.103
Naphthalene	NA/NP-1	215	ABC-1	168
Diesel Particulates	NA/NP-1	1,230	ABC-1	541
Arsenic	NA/NP-1	0.123	ABC-1	0.135
Beryllium	NA/NP-1	0.0185	ABC-1	0.0116
Cadmium	NA/NP-1	0.359	ABC-1	0.362
Chrommium VI	NA/NP-1	0.00369	ABC-1	0.00231
Manganese	NA/NP-1	7.02	ABC-1	7.53

Two sets of coordinates were used in the analyses. Stateplane coordinates are commonly used to describe locations along an east-west (X) axis and north-south (Y) axis. ISC3 air dispersion modeling used a separate coordinate system with the 0, 0 point along east-west and north-south axis at the LAX theme building. Stateplane coordinates that correspond with this location are provided to allow model results to be translated easily to other coordinate systems.

Source: Camp Dresser & McKee Inc., 2000.

6.3.2.1 Residents (Adults and Young Children)

Estimated cancer risks for maximally exposed residents for the build alternatives at 2005 were about one-half of those calculated for the No Action/No Project Alternative. Residences with the highest concentrations of TAPs were predicted to be in the same general area but at slightly different locations for the build alternatives and the No Action/No Project Alternative (**Figure 5,** Locations for Maximally Exposed Residents and School Children for Horizon Year 2005, Pre-Mitigation Conditions). Total incremental estimated cancer risks for adult residents were 20 in one million and 40 in one million, for build alternatives and No Action/No Project Alternative, respectively (**Table 9**, Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2005 Pre-Mitigation Conditions). Total estimated cancer risks for young children living at residences with maximum predicted TAP concentrations were 10 in one million and 30 in one million, respectively for the build alternatives and No Action/No Project Alternative. Estimated cancer risks are higher for adults than for children because exposure to carcinogens is cumulative through both childhood and adult years.

Risks predicted for the No Action/No Project Alternative were about twice those for the build alternatives. Greater traffic congestion predicted under No Action/No Project Alternative is responsible for much of this disparity.

Cancer risks for adults and children were mostly due to predicted exposure to diesel particulates and 1,3-butadiene. Diesel contributed 70 percent to estimated cancer risks under the No Action/No Project Alternative and 80 percent under the build alternatives. The contribution from 1,3-butadiene was 16 percent for the No Action/No Project Alternative and 13 percent under the build alternatives.

Table 9

Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2005 Pre-Mitigation Conditions

	Cancer Risk (per million individuals)					
	No Action/No Project Alternative			Alternatives A, B, and C		
TAP	Child Resident	School Child	Adult Resident[1]	Child Resident	School Child	Adult Resident
VOCs						
Acetaldehyde	0.1	0.003	0.1	0.05	0.002	0.07
Acrolein	NA	NA	NA	NA	NA	NA
Benzene	3	0.07	4	0.4	0.04	0.6
1,3-Butadiene	4	0.1	6	2	0.1	2.5
Formaldehyde	0.7	0.02	0.9	0.4	0.02	0.5
Xylene (total)	NA	NA	NA	NA	NA	NA
PAHs						
PAHs as Benzo(a)pyrene equivaltents	0.01	0.0003	0.01	0.004	0.0002	0.006
Naphthalene	NA	NA	NA	NA	NA	NA
Diesel						
Diesel Particulates	19	1	28	11	0.08	16
Dioxins						
TCDD equivalents	0.3	0.007	0.4	0.02	0.003	0.03
Metals						
Arsenic	0.01	0.0006	0.02	0.015	0.0006	0.02
Beryllium	0.001	0.00005	0.001	0.002	0.00007	0.002
Cadmium	0.03	0.002	0.05	0.04	0.002	0.05
Chromium (VI)	0.01	0.0007	0.02	0.02	0.001	0.03
Manganese	NA	NA	NA	NA	NA	NA
Total	**27**	**1**	**39**	**14**	**0.2**	**19**

[1] Adult resident includes exposure for 6 years as a child (0-6 years of age) and 24 years as an adult.
All values rounded to one significant figure or a whole number.
NA - Not Available

Source: Camp Dresser & McKee Inc., 2000.

The estimated HI for children living at locations with maximum TAP concentrations was five for both the build alternatives and the No Action/No Project Alternative (**Table 10,** Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2005 Pre-Mitigation Conditions). HI estimates include cumulative exposures to all TAPs that are toxic to the respiratory system at low chronic daily exposure. The HI for maximally exposed adult residents was two for the No Action/No Project Alternative and one for the build alternatives. HI estimates are higher for children than adults because HIs are normalized to body weight, which is lower for children than for adults.

Acrolein is the only chemical for which the HQ exceeds one. Acrolein contributes more than 95 percent to the total HIs for all alternatives. The major source of acrolein at LAX is jet exhaust. Since air traffic for the build alternatives and the No Action/No Project Alternative are predicted to be very similar in 2005, little difference in non-cancer health hazards is anticipated.

Table 10

Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2005 Pre-Mitigation Conditions

	Hazard Quotient					
	No Action/No Project Alternative			Alternatives A, B, and C		
TAP	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident
VOCs						
Acetaldehyde	0.05	0.001	0.01	0.02	0.001	0.007
Acrolein	5.0	0.2	1.4	5	0.2	1
Benzene	NA	NA	NA	NA	NA	NA
1,3-Butadiene	NA	NA	NA	NA	NA	NA
Formaldehyde	0.002	0.0001	0.0005	0.001	0.00005	0.0003
Xylene (total)	0.0003	0.000007	0.00009	0.00002	0.000004	0.000007
PAHs						
Naphthalene	0.05	0.002	0.01	0.03	0.001	0.007
Diesel						
Diesel Particulates	0.1	0.008	0.04	0.08	0.0006	0.02
Dioxins						
TCDD equivalents	NA	NA	NA	NA	NA	NA
Metals						
Arsenic	0.00004	0.000002	0.00001	0.00005	0.000002	0.00001
Beryllium	0.0003	0.00001	0.00008	0.0005	0.00002	0.0001
Cadmium	0.0005	0.00002	0.0001	0.0005	0.00003	0.0001
Chromium (VI)	0.00001	0.0000006	0.000003	0.00002	0.0000008	0.000005
Manganese	0.04	0.002	0.01	0.05	0.002	0.02
Total Hazard Index	**5**	**0.2**	**2**	**5**	**0.2**	**1**
Total HI for Respiratory Effects	**5**	**0.2**	**2**	**5**	**0.2**	**1**

All values rounded to one significant figure or a whole number.
NA - Not Available

Source: Camp Dresser & McKee Inc., 2000.

6.3.2.2 School Children

Maximum TAP concentrations under the build alternatives and the No Action/No Project Alternative were predicted for the Oak Street Elementary School near the current eastern LAX fence line (**Figure 5**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2005, Pre-Mitigation Conditions).

Estimated cancer risks for children attending the above schools are 0.2 in one million and 1 in one million for the build alternatives and the No Action/No Project Alternative, respectively (**Table 9**, Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2005, Pre-Mitigation Conditions). Estimated risks for the No Action/No Project Alternative are higher than those for the build alternatives; this difference is due to increased traffic congestion predicted for the No Action/No Project Alternative in the future.

The estimated total HIs for chemicals affecting the same target (i.e., the respiratory system) for MEI school children are 0.2 for both the build alternatives and the No Action/No Project Alternative. Estimated HIs are predominantly due to exposure to acrolein in jet exhaust.

6.3.2.3 Risks Described Geographically

For the build alternatives and the No Action/No Project Alternative, total incremental cancer risks and hazard indices (HIs) for all respiratory irritants (acetaldehyde, acrolein, formaldehyde, diesel particulates, beryllium, chromium VI, and manganese) were calculated for each grid node in the ISC3 modeling domain. Risks and HIs were then used to generate estimates of risks and hazards on a spatial basis as overlays for maps of LAX and surrounding communities. All risk and hazard estimates used to describe risk geographically were for residents living within the study area. Further, cancer risk maps reflect adult residents, while non-cancer health hazard maps reflect exposure to young children, ages 0 to 6. Results for pre-mitigation conditions in Project Year 2005 are presented in **Figure 6**, Geographic Extent of

Incremental Cancer Risks for Horizon Year 2005, Pre- and Post-Mitigation Conditions. This figure depicts the extent of risks between Alternatives A, B, and C and the No Action/No Project Alternative. Negative incremental risks or HIs are calculated when risks from LAX operations are lower than baseline risks.

Incremental upper range cancer risks exceeding 10 in one million are estimated for an area extending approximately 13,000 feet east-northeast from the LAX boundary for the No Action/No Project Alternative. For Alternatives A, B, and C, cancer risks exceeding one in one million were predicted for an area extending approximately 18,000 feet east northeast from the LAX boundary. Cancer risks exceeding 10 in one million were estimated for Alternatives A, B, and C only in a small area immediately adjacent to the east end of the north runways. Cancer risks are much reduced under airport operation assumptions for the build alternatives.

Incremental non-cancer health effects for the build alternatives and the No Action/No Project Alternative are depicted in **Figure 7**, Geographic Extent of Incremental Non-Cancer Health Hazards for Horizon Year 2005, Pre- and Post-Mitigation Conditions. Incremental non-cancer HIs above unity are not predicted for most off-site locations for Alternatives A, B, and C and the No Action/No Project Alternative. This suggests little potential for impacts from either implementation of the build alternatives or the No Action/No Project Alternative.

6.4 Risk Estimates for Pre-Mitigation Conditions for Horizon Year 2015

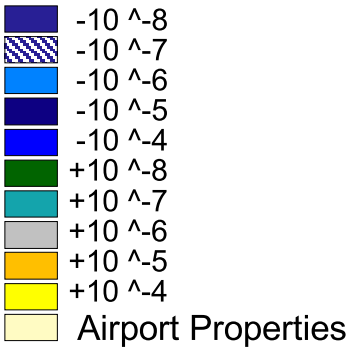
For 2015, the build alternatives and the No Action/No Project Alternative were quantitatively evaluated. Separate modeling results for all alternatives were provided, allowing separate calculations for each alternative.

6.4.1 Comparison of On-Airport Air Concentrations with OSHA Standards for Workers

Workers are evaluated by comparing estimated annual air concentrations of TAPs for the different alternatives to eight-hour PEL-TWAs. Estimated on-airport air concentrations and TLV-TWAs for TAPs of concern for LAX are presented in **Table 11**, Comparison of CalOSHA Permissible Exposure Limits to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2015 Pre-Mitigation Conditions.

FIGURE LEGENDS

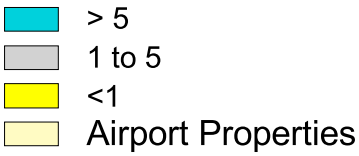
LEGEND - Incremental Cancer Risk (Figures 6, 10, 14)



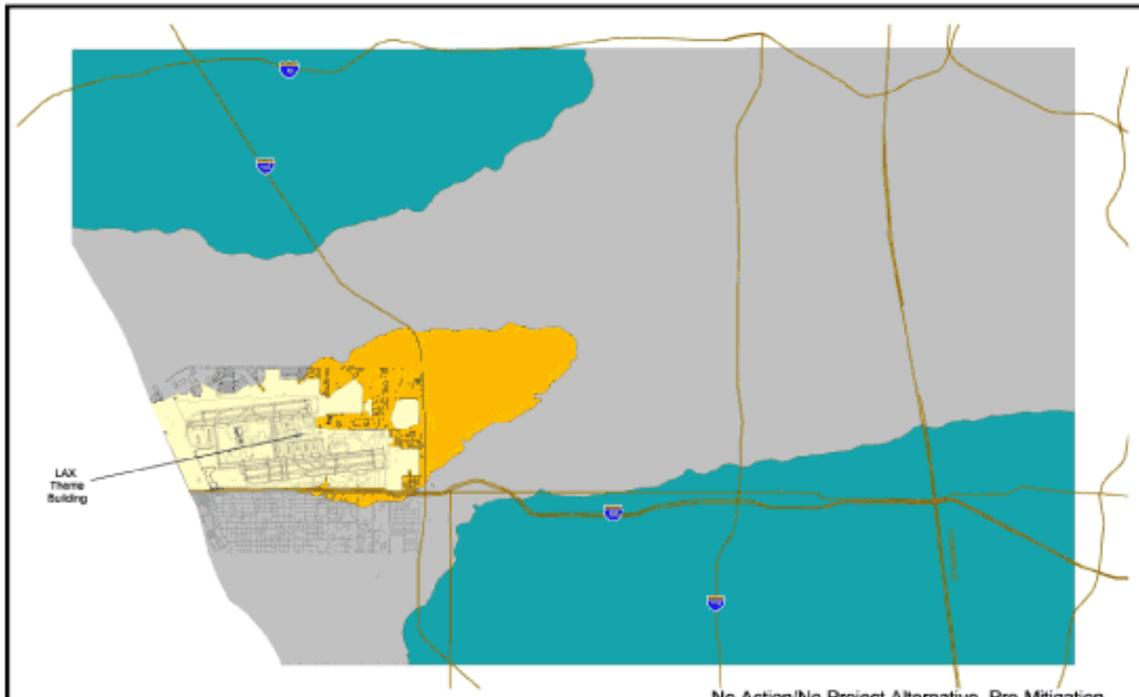
Negative Incremental Risks indicate that risks associated with an alternative are less than baseline risks.

Positive Incremental Risks indicate that risks associated with an alternative are greater than baseline risks.

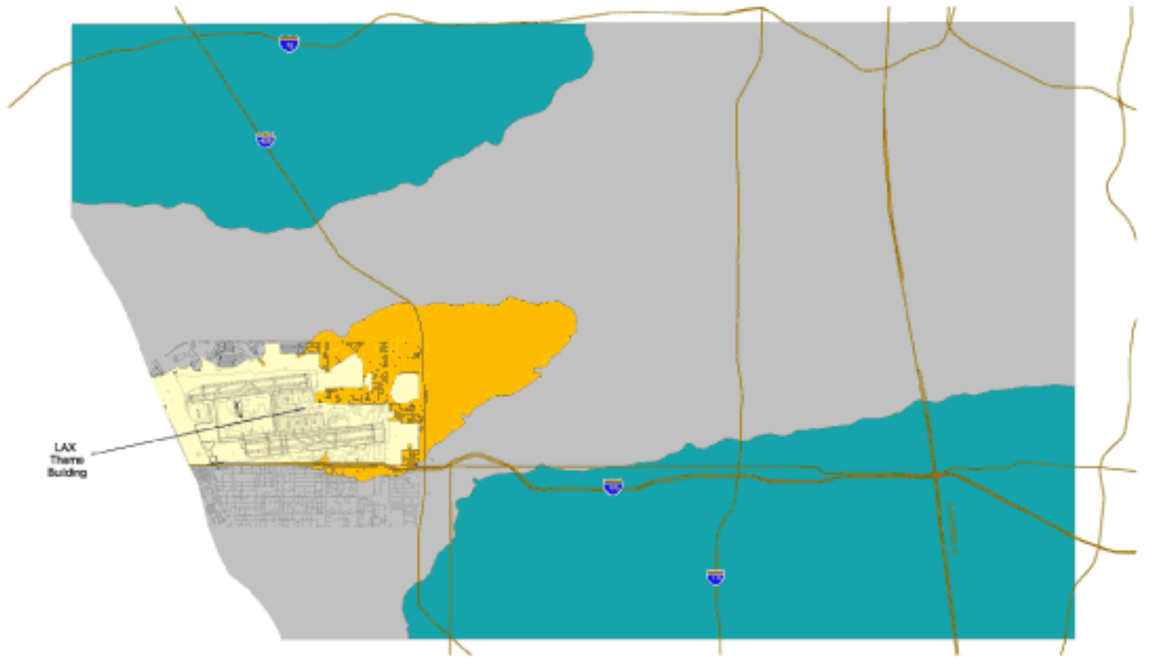
LEGEND - Incremental Non-Cancer Health Hazards (Figures 7, 11, 15)



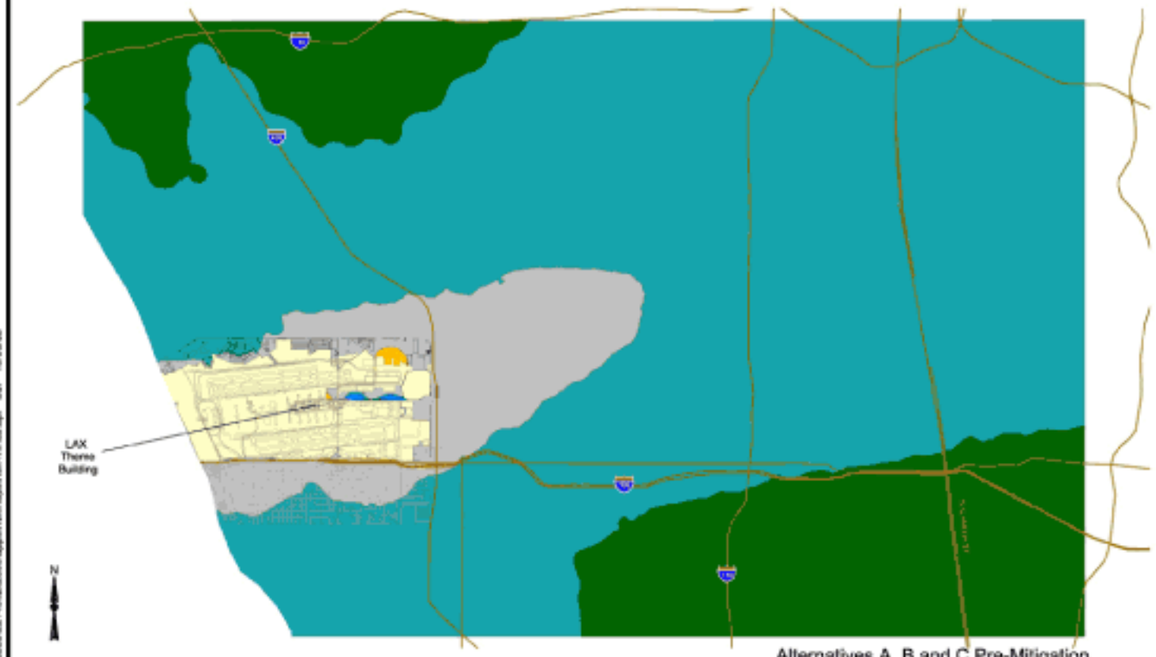
Positive Incremental Hazard Indices indicate that the Hazard Indices associated with an alternative are greater than the baseline hazard index.



No Action/No Project Alternative, Pre-Mitigation

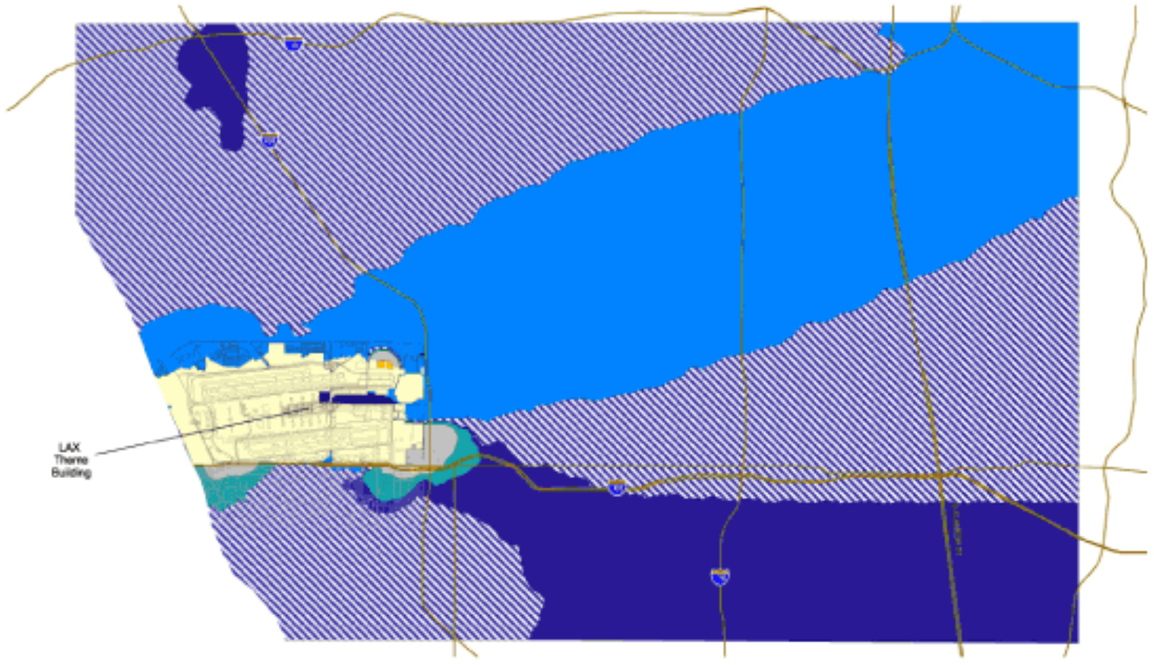


No Action/No Project Alternative, Post-Mitigation



Alternatives A, B and C Pre-Mitigation

Note: Airport Boundary Footprint shown is for Alternative C.



Note: Airport Boundary Footprint shown is for Alternative C.

Alternatives A, B and C Post-Mitigation

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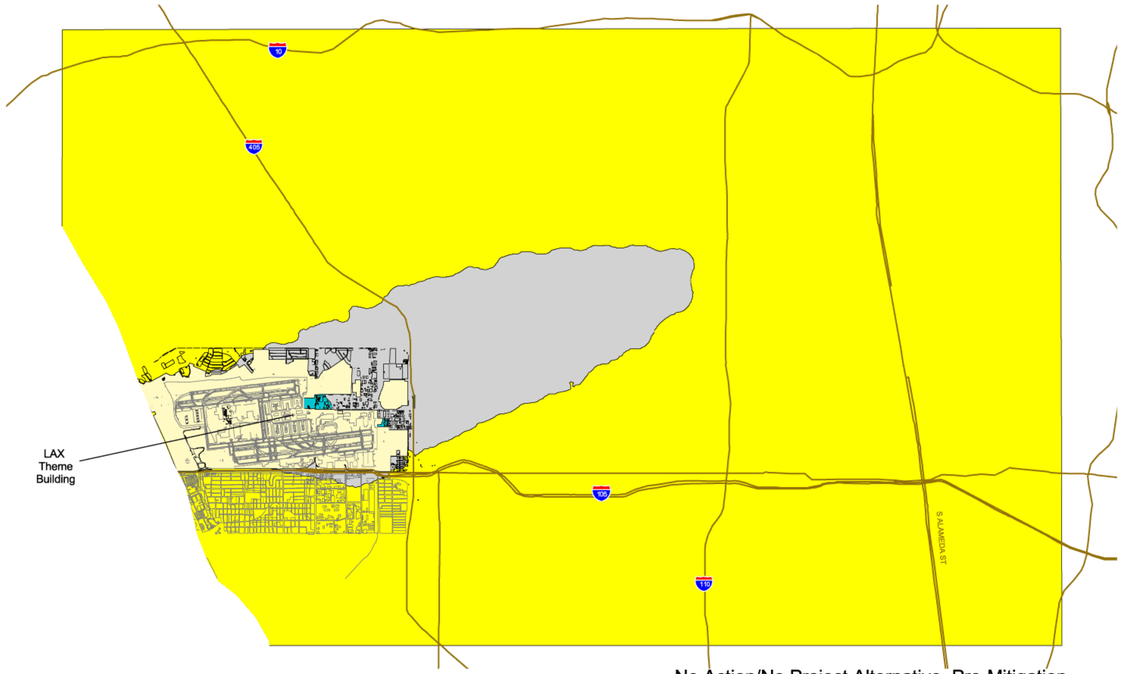
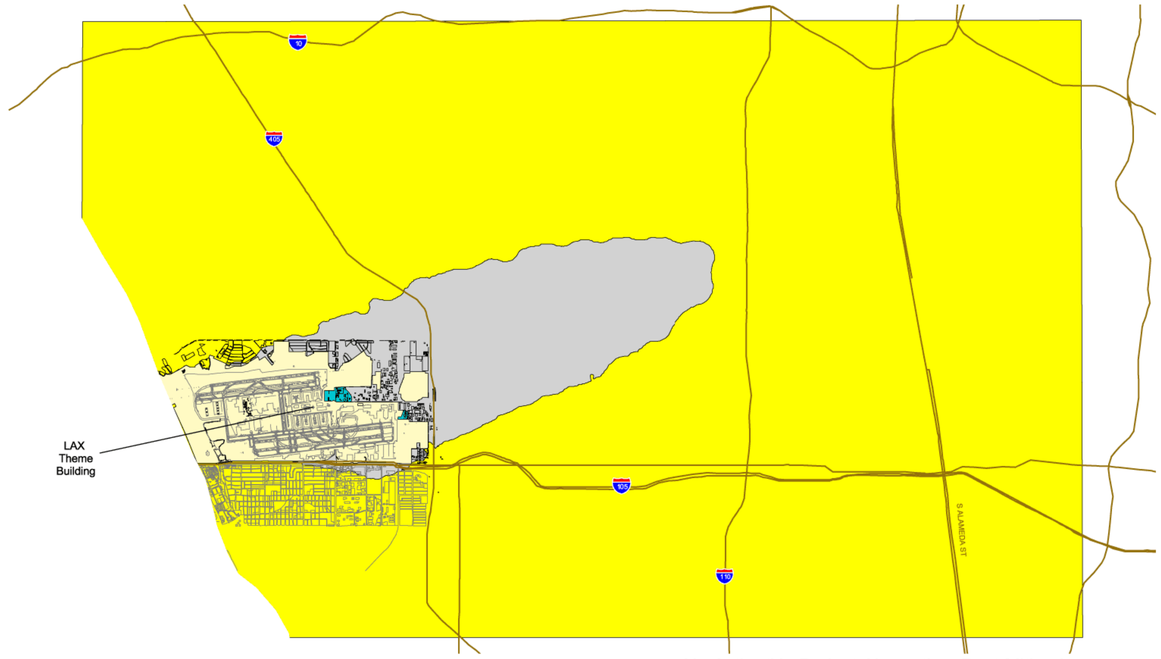
Geographic Extent of Incremental Cancer Risks for Horizon Year 2005 Pre- and Post-Mitigation Conditions

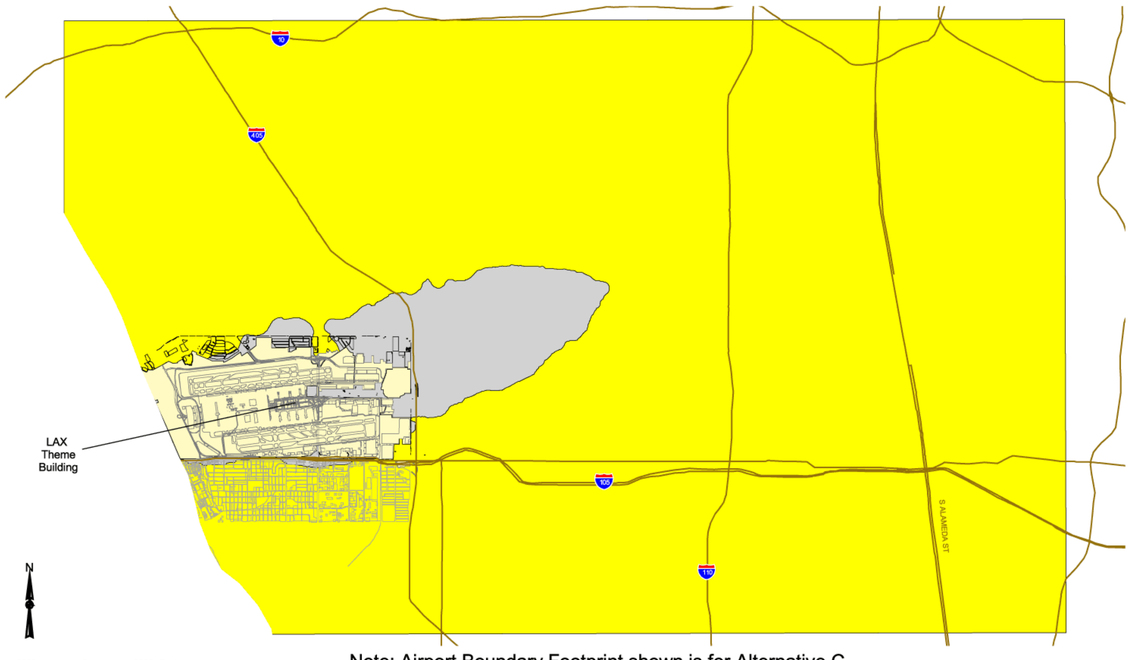
Figure 6



No Action/No Project Alternative, Pre-Mitigation

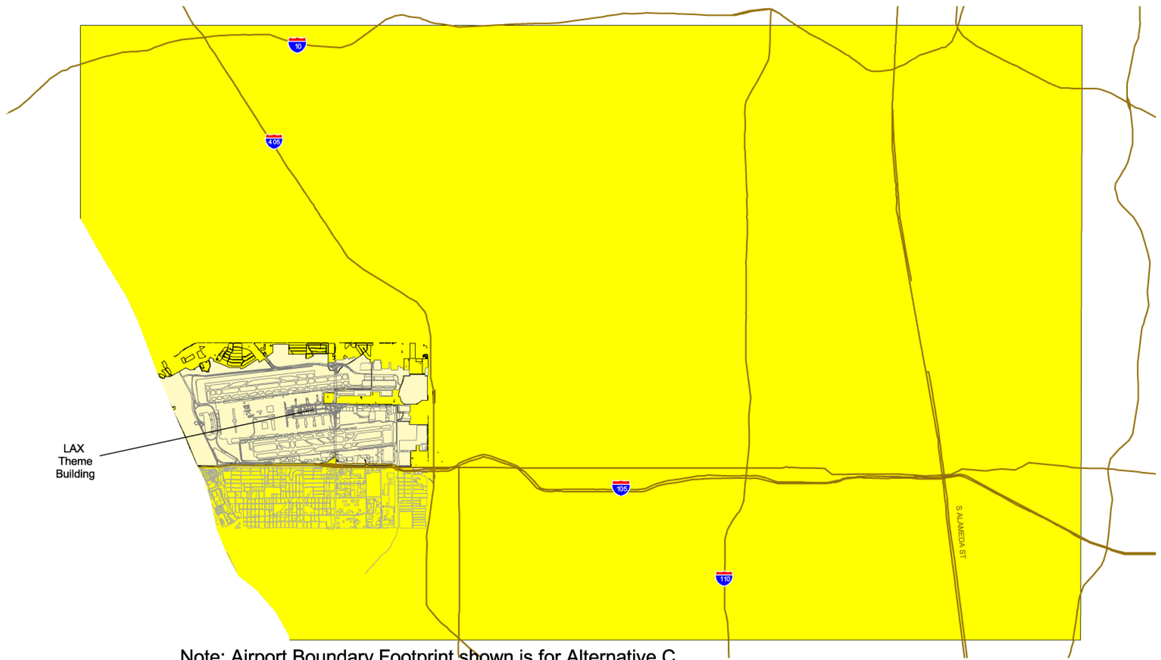


No Action/No Project Alternative, Post-Mitigation



Note: Airport Boundary Footprint shown is for Alternative C.

Alternatives A, B and C Pre-Mitigation



Note: Airport Boundary Footprint shown is for Alternative C.
All Incremental Health Hazards are less than 1.

Alternatives A, B and C Post-Mitigation

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Geographic Extent of Incremental Non-Cancer Health Hazards for Horizon Year 2005 Pre- and Post-Mitigation Conditions

Figure 7

Table 11

Comparison of CalOSHA Permissible Exposures Limits to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2015 Pre-Mitigation Conditions

	Alternative (mg/m^3)				CalOSHA PEL-TWA
TAP[1]	No Action/No Project	A	B	C	(mg/m^3)[2]
Acetaldehyde	$4.4x10^{-3}$	$2.3x10^{-3}$	$3.0x10^{-3}$	$2.6x10^{-3}$	$1.8x10^{2}$
Acrolein	$1.2x10^{-3}$	$1.0x10^{-3}$	$1.4x10^{-3}$	$1.2x10^{-3}$	$2.5x10^{-1}$
Benzene	$8.4x10^{-3}$	$3.7x10^{-3}$	$4.0x10^{-3}$	$3.9x10^{-3}$	$3.2x10^{-1, 3}$
1,3-Butadiene	$2.6x10^{-3}$	$1.4x10^{-3}$	$1.9x10^{-3}$	$1.7x10^{-3}$	$2.2x10^{0}$
Diesel Particulates	$9.6x10^{-3}$	$1.1x10^{-3}$	$2.0x10^{-3}$	$1.1x10^{-3}$	NA
Formaldehyde	$1.4x10^{-2}$	$7.3x10^{-3}$	$9.8x10^{-3}$	$8.6x10^{-3}$	$3.7x10^{-1, 3}$
Naphthalene	$1.5x10^{-3}$	$1.4x10^{-3}$	$1.8x10^{-3}$	$1.6x10^{-3}$	$5.5x10^{1}$
PAHs as Benzo(a)pyrene equivalents	$1.2x10^{-6}$	$7.0x10^{-7}$	$9.4x10^{-7}$	$8.3x10^{-7}$	NA
TCDD equivalents	$6.3x10^{-10}$	$2.8x10^{-10}$	$2.1x10^{-10}$	$3.3x10^{-10}$	NA
Xylenes	$1.4x10^{-2}$	$6.0x10^{-3}$	$6.6x10^{-3}$	$7.5x10^{-3}$	$4.34x10^{2}$
Arsenic	$1.1x10^{-6}$	$9.4x10^{-7}$	$1.1x10^{-6}$	$1.1x10^{-6}$	$1.0x10^{-2}$
Beryllium	$2.0x10^{-7}$	$1.7x10^{-7}$	$2.3x10^{-7}$	$2.0x10^{-7}$	$2.0x10^{-3}$
Cadmium	$3.2x10^{-6}$	$3.1x10^{-6}$	$3.7x10^{-6}$	$2.7x10^{-6}$	$5.0x10^{-3}$
Chromium (as Cr(VI))	$3.9x10^{-8}$	$3.4x10^{-8}$	$4.6x10^{-8}$	$4.1x10^{-8}$	$5.0x10^{-2}$
Manganese	$6.4x10^{-5}$	$5.1x10^{-5}$	$6.3x10^{-5}$	$6.3x10^{-5}$	$5.0x10^{0}$

NA - Not Available

1 All TAPs for which PEL-TWAs are available are listed. PEL-TWAs are not available for diesel exhaust, chromium VI and PAHs other than naphthalene.

2 CalOSHA (California Occupational Safety and Health Administration). 2000. Table AC-1, Permissible Exposure Limits for Chemical Contaminants. http:\www.dir.ca.gov/title8/5155a.htm.

3 CalOSHA Value not available; value is from American Conference of Governmental Industrial Hygienists (ACGIH), Documentation of the Threshold Limit Values and Biological Exposure Indices, 8th ed., Cincinnati, Ohio, 1998.

Source: Camp Dresser & McKee Inc., 2000.

Estimated maximum 8-hour on-airport concentrations for all build alternatives and the No Action/No Project are similar for most TAPs, but locations where maximum concentrations are predicted vary. (**Figure 8**, Locations for Maximally Exposed Workers for Horizon Year 2015, Pre-Mitigation Conditions and **Table 12**, Legend for **Figure 8**, Locations for Maximally Exposed Workers for Horizon Year 2015, Pre-Mitigation Conditions. Estimated concentrations for all TAPs of concern except acrolein, naphthalene, beryllium, cadmium and chromium(VI) are, however, higher for the No Action/No Project Alternative than the build alternatives. This finding is repeated throughout all hazard analyses for on-airport workers. Higher concentrations associated with the No Action/No Project Alternative are due, in part, to increased motor vehicle traffic predicted in the absence of airport expansion as defined by the build alternatives. Additionally, aircraft operations under the build alternatives would be more efficient, resulting in decreased releases of jet engine exhaust during taxi and queue times when compared to the No Action/No Project Alternative.

Estimated maximum 8-hour air concentrations for the build alternatives and the No Action/No Project Alternative are well below PELs for all TAPs. This result suggests that air concentrations from airport emissions with or without implementation of the LAX Master Plan would not exceed those considered "acceptable" by CalOSHA.

6.4.2 Risks for Maximally Exposed Individuals (MEI)

6.4.2.1 Residents (Adults and Children)

Estimated cancer risks for maximally exposed residents vary at most by a factor of 4 among Year 2015 build alternatives and the No Action/No Project Alternative. Residents living at the location of the highest estimated TAP concentrations were found at different locations in the three build alternatives and the No Action/No Project Alternative. (**Figure 9**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2015, Pre-Mitigation Conditions). Total estimated cancer risks for adult residents were

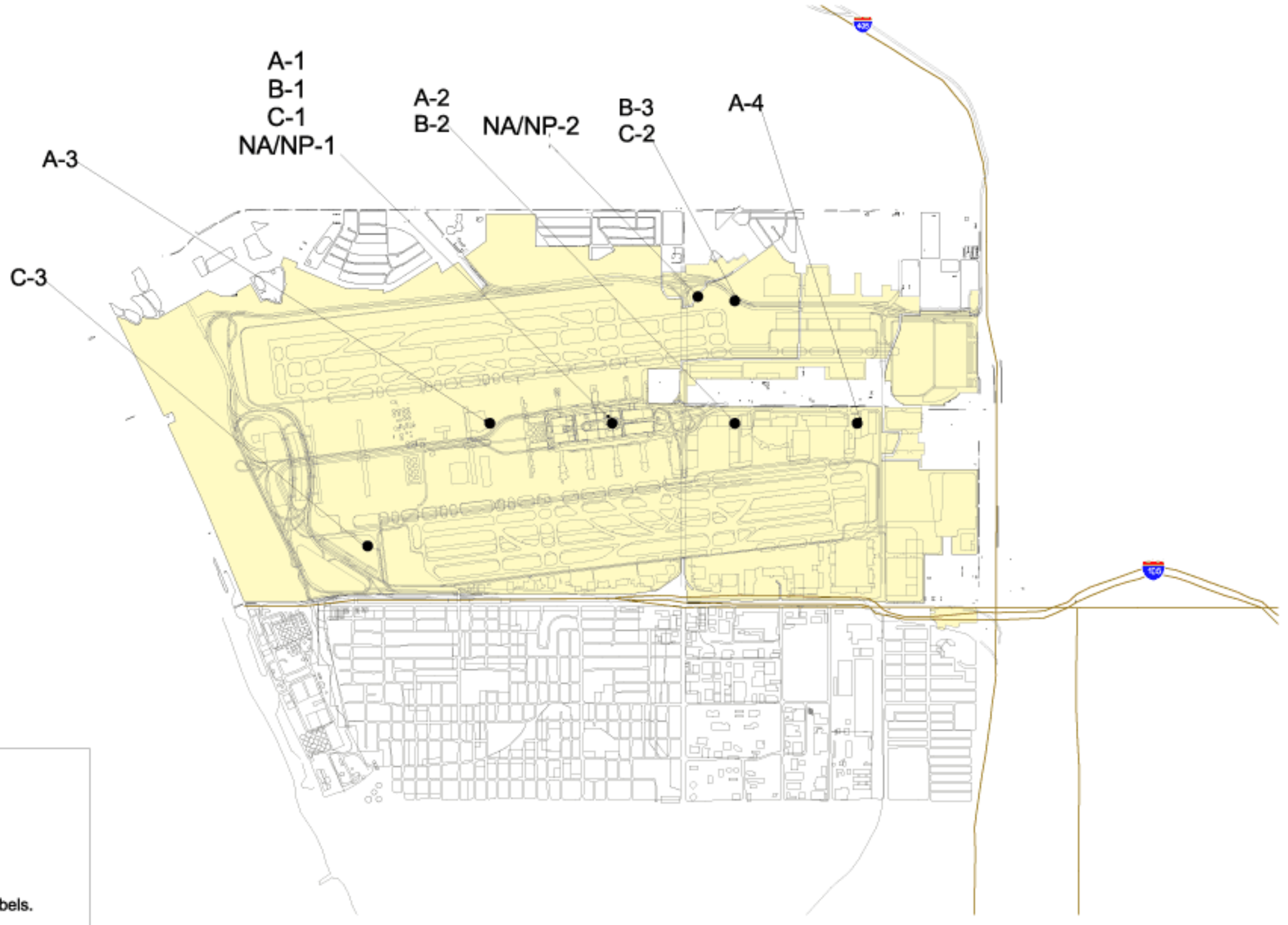
Table 12

Legend for Figure 8, Locations for Maximally Exposed Workers for Horizon Year 2015, Pre-Mitigation Conditions

Label	X_stateplane (ft)	Y_stateplane (ft)	Model_X (m)	Model_Y (m)
A-1	6439597.00	1802295.00	0	0
A-2	6442877.84	1802295.00	1,000	0
A-3	6436316.16	1802295.00	-1,000	0
A-4	6446158.68	1802295.00	2,000	0
B-1	6439597.00	1802295.00	0	0
B-2	6442877.84	1802295.00	1,000	0
B-3	6442877.84	1805575.84	1,000	1,000
C-1	6439597.00	1802295.00	0	0
C-2	6442877.84	1805575.84	1,000	1,000
C-3	6433035.32	1799014.16	-2,000	-1,000
NP-1	6439597.00	1802295.00	0	0
NP-2	6441893.59	1805690.67	700	1,035

	Alternative A		Alternative B		Alternative C		No Action/No Project	
TAP	Label	Maximum Onsite Concentration (ng/m^3)	Label	Maximum Onsite Concentration (ng/m^3)	Label	Maximum Onsite Concentration (ng/m^3)	Label	Maximum Onsite Concentration (ng/m^3)
Acetaldehyde	A-1	259	B-3	342	C-2	301	NP-1	496
Acrolein	A-1	95.4	B-3	162	C-2	142	NP-1	144
Benzene	A-3	428	B-1	452	C-1	445	NP-1	961
1,3-Butadiene	A-1	163	B-3	220	C-2	192	NP-1	302
Formaldehyde	A-4	829	B-3	1120	C-2	983	NP-1	1590
Xylene (total)	A-1	680	B-1	757	C-3	853	NP-1	1590
TCDD equivalents	A-1	0.0000319	B-1	0.0000357	C-3	0.0000373	NP-1	0.0000716
PAHs as Benzo(a)pyrene	A-3	0.0803	B-3	0.107	C-2	0.0943	NP-1	0.143
Naphthalene	A-4	156	B-3	205	C-2	183	NP-2	173
Diesel Particulates	A-2	121	B-1	125	C-1	122	NP-1	1120
Arsenic	A-4	0.0748	B-1	0.13	C-1	0.13	NP-1	0.127
Beryllium	A-4	0.0187	B-3	0.0263	C-2	0.0232	NP-2	0.0227
Cadmium	A-2	0.359	B-2	0.42	C-1	0.307	NP-1	0.362
Chrome VI	A-3	0.00229	B-3	0.00526	C-1	0.00463	NP-2	0.00452
Manganese	A-1	5.77	B-1	7.15	C-1	7.19	NP-1	7.27

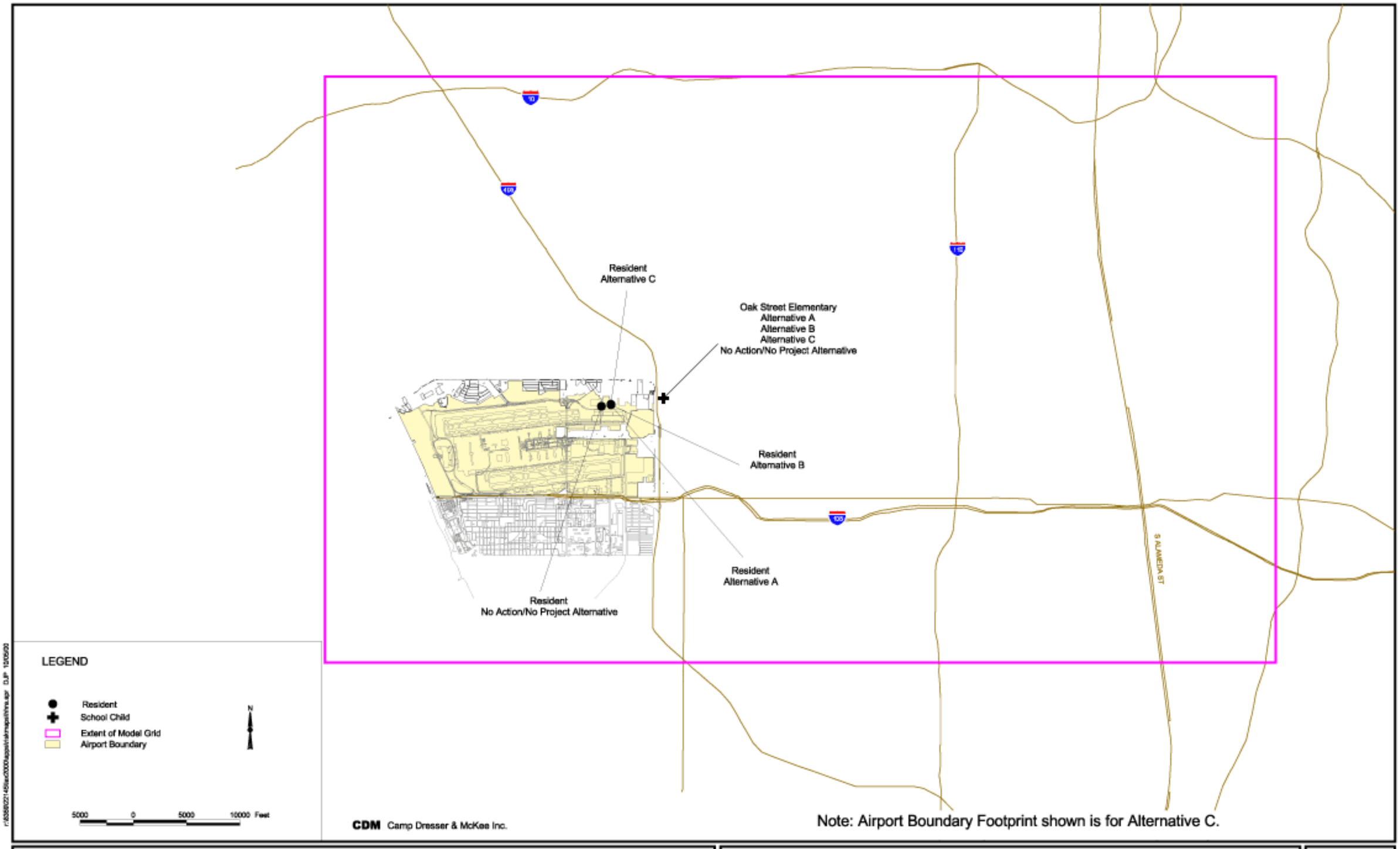
Source: Camp Dresser & McKee Inc, 2000.



Note: Airport Boundary Footprint shown is for Alternative C.

CDM Camp Dresser & McKee Inc.

Los Angeles International Airport Master Plan	**Locations for Maximally Exposed Workers for Horizon Year 2015 Pre-Mitigation Conditions**	Figure 8



Los Angeles International Airport Master Plan	**Locations for Maximally Exposed Residents and School Children for Horizon Year 2015 Pre-Mitigation Conditions**	Figure 9

-11 in one million, 10 in one million, 0.9 in one million, and 33 in one million for Alternatives A, B, and C and the No Action/No Project Alternative, respectively (**Table 13**, Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2015, Pre-Mitigation Conditions). Total estimated cancer risks for young children living at residences with maximum predicted TAP concentrations were -7 in one million, 7 in one million, 0.6 in one million, and 23 in one million, respectively, for the No Action/No Project Alternative and Alternatives A, B, and C, respectively. Estimated cancer risks are higher for adults than for children, because exposure duration for adults is longer. Negative incremental cancer risk for Alternative A indicate a reduction in risk below those associated with baseline conditions. Implementation of Alternative A would result in a beneficial impact on LAX-associated cancer risk.

Risks predicted for the No Action/No Project Alternative were higher than those for the build alternatives. Greater traffic congestion predicted under the No Action/No Project Alternative is responsible for much of this disparity. Cancer risks for adults and children under the No Action/No Project Alternative were mostly due to predicted exposure to diesel particulates and 1,3-butadiene. Diesel contributed 77 percent to estimated cancer risks and 1,3-butadiene contributed 14 percent.

Cancer risks from exposure to diesel particulates for Alternatives A, B, and C were less than those for baseline, and total cancer risk from Alternative A was less than under baseline conditions. For Alternatives B and C, increased incremental cancer risks were due mainly to exposure to 1,3-butadiene. The findings are explained by decreased diesel emissions predicted for the build alternatives, and by increased emissions of 1,3-butadiene in exhaust from aircraft engines. The latter will increase as air traffic increases after implementation of the LAX Master Plan.

Estimated HIs for children living at locations with maximum TAP concentrations 6, 14, 11, and 6 for Alternatives A, B, and C, and the No Action/No Project Alternative, respectively (**Table 14**, Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2015, Pre-Mitigation Conditions). HIs for maximally exposed adult residents are 2, 4, 3, and 2 for the Alternatives A, B, and C, and the No Action/No Project Alternative, respectively. HI estimates are higher for children than adults, because they are normalized to body weight, which is lower for children than for adults. HI estimates are slightly higher for Alternative B than for the No Action/No Project Alternative and for Alternatives A and C.

Acrolein is the only chemical for which the HQ exceeds one. Acrolein contributes 97 percent or more to total HIs for all alternatives. Since the source of acrolein is mainly jet engine exhaust, the somewhat greater HIs associated with Alternatives B and C are apparently due to a higher volume of aircraft traffic predicted for 2015.

6.4.2.2 School Children

Maximum TAP concentrations under the three build alternatives were predicted to be highest for the Oak Creek Elementary School. Maximum TAP concentrations under the No Action/No Project Alternative were predicted for the Escuela de Montessori to the north of the airport near the LAX fence line. Locations of schools are shown in (**Figure 9**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2015, Pre-Mitigation Conditions).

Estimated cancer risks for children attending the above schools are -0.3 in one million, 0.2 in one million, - 0.2 in one million, and one in one million for Alternatives A, B, and C and the No Action/No Project Alternative, respectively (**Table 13**, Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2015 Pre-Mitigation Conditions). Negative cancer risks indicate a reduction in risk compared to baseline conditions. Estimated risks for the No Action/No Project Alternative are higher than those for the three build alternatives because of increased traffic congestion and less efficient aircraft operations.

Cancer risks for school children are substantially lower than risks for adult or child residents. This finding is due in part to the locations of schools, which are not expected to experience maximum offsite TAP concentrations, and in part to shorter exposure times for children that spend only part of each day at school.

Estimated total HIs for chemicals affecting the same target organ (i.e., the respiratory system) for MEI school children are 0.3, 0.5, 0.4, and 0.3 for Alternatives A, B, and C, and the No Action/No Project Alternative, respectively (**Table 14**, Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2015 Pre-Mitigation Conditions). All estimated HIs are predominantly from acrolein (i.e., more than 97 percent). The similar HI estimates reflect similar impacts of jet exhaust at the two school locations where annual concentrations are predicted to be highest.

Table 13

Estimated Incremental Cancer Risks for Maximally Exposed individuals for 2015 Pre-Mitigation Conditions

	Cancer Risk (per million individuals)											
	No Action/No Project Alternative			Alternative A			Alternative B			Alternative C		
TAP	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident
VOCs												
Acetaldehyde	0.1	0.003	0.1	0.1	0.003	0.1	0.1	0.01	0.2	0.1	0.004	0.1
Acrolein	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	2	0.1	2	0.2	0.02	0.2	0.7	0.04	1	0.4	0.03	0.5
1,3-Butadiene	3	0.1	5	2	0.1	3	5	0.2	7	4	0.2	5
Formaldehyde	1	0.02	1	0.4	0.02	0.5	0.9	0.04	1	1	0.03	1
Xylene (total)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PAHs												
PAHs as Benzo(a)pyrene equivalents	0.01	0.0004	0.01	0.01	0.001	0.01	0.02	0.001	0.02	0.01	0.001	0.02
Naphthalene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dioxins												
TCDD equivalents	0.2	0.004	0.2	0.01	0.003	0.02	0.1	0.01	0.1	0.04	0.004	0.1
Diesel												
Diesel Particulates	18	1	25	-10	-0.5	-14	-0.01	-0.2	-0.01	-4	-0.4	-6
Metals												
Arsenic	0.02	0.001	0.03	0.03	0.002	0.04	0.1	0.002	0.1	0.05	0.002	0.1
Beryllium	0.002	0.0001	0.003	0.01	0.0002	0.01	0.01	0.0003	0.01	0.01	0.0003	0.01
Cadmium	0.1	0.003	0.07	0.1	0.01	0.1	0.2	0.01	0.3	0.1	0.01	0.1
Chromium (VI)	0.03	0.002	0.05	0.1	0.004	0.1	0.1	0.01	0.2	0.1	0.004	0.1
Manganese	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total	**23**	**1**	**33**	**-7**	**-0.3**	**-11**	**7**	**0.2**	**10**	**0.6**	**-0.2**	**0.9**

All values rounded to one significant figure.
NA – Not Available

Source: Camp Dresser & McKee Inc., 2000.

Table 14

Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2015 Pre-Mitigation Conditions

	Hazard Quotient											
	No Action/No Project Alternative			Alternative A			Alternative B			Alternative C		
	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident
TAP												
VOCs												
Acetaldehyde	0.04	0.001	0.01	0.02	0.001	0.007	0.061	0.003	0.02	0.05	0.002	0.01
Acrolein	6	0.3	2	6	0.3	2	13	0.5	4	11	0.4	3
Benzene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,3-Butadiene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Formaldehyde	0.002	0.0001	0.0004	0.0010	0.0001	0.0003	0.003	0.0001	0.001	0.002	0.0001	0.001
Xylene (total)	0.0001	0.000004	0.00004	-0.00002	0.000002	-0.00001	0.00002	0.000003	0.00001	-0.000002	0.000002	-0.000001
PAHs												
Naphthalene	0.05	0.002	0.01	0.06	0.004	0.02	0.12	0.01	0.04	0.10	0.00	0.03
Dioxins												
TCDD equivalents	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Diesel												
Diesel Particulates	0.1	0.007	0.04	-0.07	-0.004	-0.02	-0.0001	-0.001	-0.00002	-0.032	-0.003	-0.009
Metals												
Arsenic	0.00006	0.000003	0.00002	0.0001	0.00001	0.00003	0.0002	0.00001	0.00005	0.0002	0.00001	0.00004
Beryllium	0.0006	0.00004	0.0002	0.001	0.00007	0.0004	0.002	0.0001	0.0007	0.002	0.0001	0.0006
Cadmium	0.0007	0.00004	0.0002	0.0007	0.00007	0.0002	0.003	0.0001	0.0008	0.0011	0.0001	0.0003
Chromium (VI)	0.00003	0.000001	0.00001	0.00005	0.000003	0.00002	0.00009	0.000004	0.00003	0.00008	0.000003	0.00002
Manganese	0.07	0.00383	0.02	0.12	0.01	0.03	0.20	0.01	0.06	0.17	0.01	0.05
Total Hazard Index	**6**	**0.3**	**2**	**6**	**0.3**	**2**	**14**	**0.5**	**4**	**11**	**0.4**	**3**
Total HI for Respiratory Effects	**6**	**0.3**	**2**	**6**	**0.3**	**2**	**14**	**0.5**	**4**	**11**	**0.4**	**3**

Values rounded to one significant figure, or to nearest whole number.
NA – Not Available

Source: Camp Dresser & McKee Inc., 2000.

6.4.2.3 Risks Described Geographically

For each build alternative and the No Action/No Project Alternative, total cancer risks, and HIs for all respiratory irritants (acetaldehyde, acrolein, formaldehyde, diesel particulates, beryllium, chromium VI, and manganese) were calculated for each grid node in the ISC3 modeling domain. Risks and HIs were then used to generate estimates of risks and hazards on a spatial basis as overlays for maps of LAX and surrounding communities. All risk and hazard estimates used to describe risk geographically were for residents living within the study area.

Cancer risks under pre-mitigation conditions in 2015 are presented in **Figure 10**, Geographic Extent of Incremental Cancer Risks for Horizon Year 2015 Pre-Mitigation Conditions. This figure depicts ranges of incremental cancer risks from -1 in 100 million to 10 in one million. Negative values indicate that cancer risks would be reduced when compared to risks associated with baseline (1996) conditions; positive values indicate an incremental increase in cancer risk compared to the baseline.

The areal extent of upper range incremental cancer risk estimates that exceed 10 in one million is largest for the No Action/No Project Alternative (**Figure 10**, Geographic Extent of Incremental Cancer Risks for Horizon Year 2015, Pre-Mitigation Conditions). Risks may be as high as 10 in one million at distances of over 7,000 feet east-northeast from the LAX boundary. The areas where risks could equal or exceed 10 in one million are substantially smaller for Alternatives A, B, and C.

Incremental non-cancer health effects for the No Action/No Project Alternative and Alternatives A, B, and C are depicted in **Figure 11**, Geographic Extent of Incremental Non-Cancer Health Hazards for Horizon Year 2015, Pre-Mitigation Conditions. This figure shows areas where incremental HIs are less than 5, between 5 and 1, and less than 1.

Non-cancer health hazards might exceed an HI of 5 in a small area east of the LAX fenceline under Alternatives A, B, and C. The extent of the areas where HIs may exceed five is largest for Alternative B and smallest for Alternative A. HIs greater than five were not estimated for the No Action/No Project Alternative. Impacts associated with the build alternatives are apparently due to increased emissions of acrolein in jet exhaust as a result of increased air traffic associated with LAX expansion.

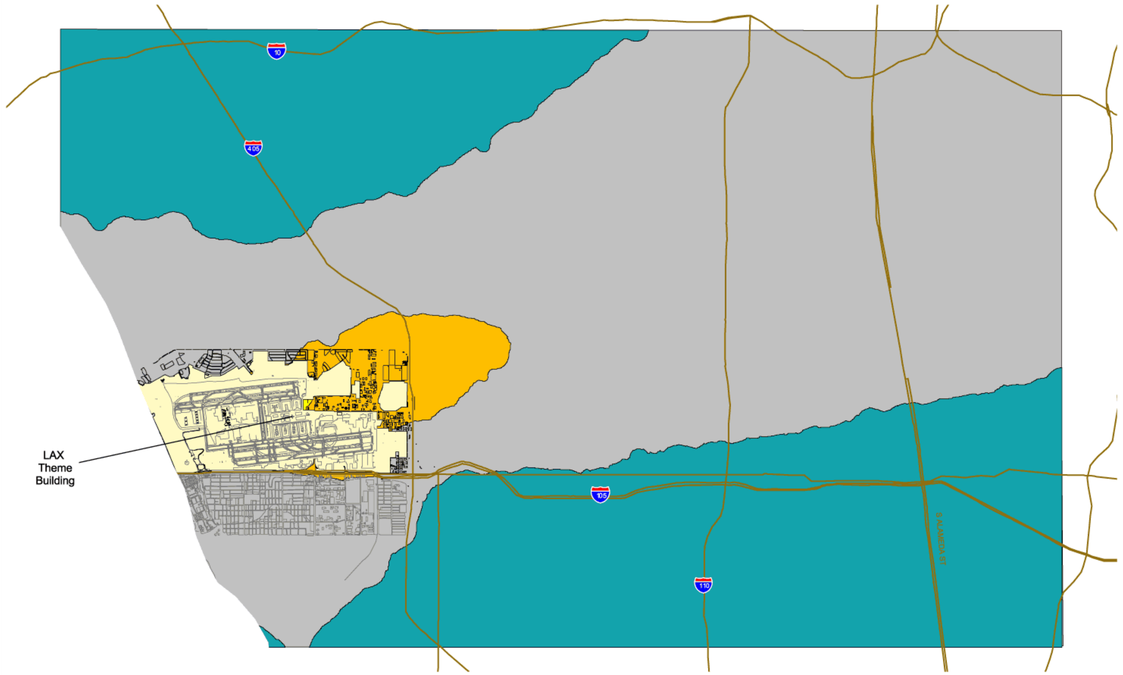
6.5 Risk Estimates for Post-Mitigation Conditions for Horizon Year 2005

The preliminary risk estimates for 2005 Post-Mitigation conditions for Alternative C were quantitatively evaluated. Emissions for Alternatives A and B were estimated to be essentially identical to those for Alternative C. Therefore, all conclusions that apply to Alternative C also apply to Alternatives A and B. See Section 7.5, *Uncertainties in Mitigation Impacts*, for a discussion of the uncertainties in the preliminary post-mitigation impacts.

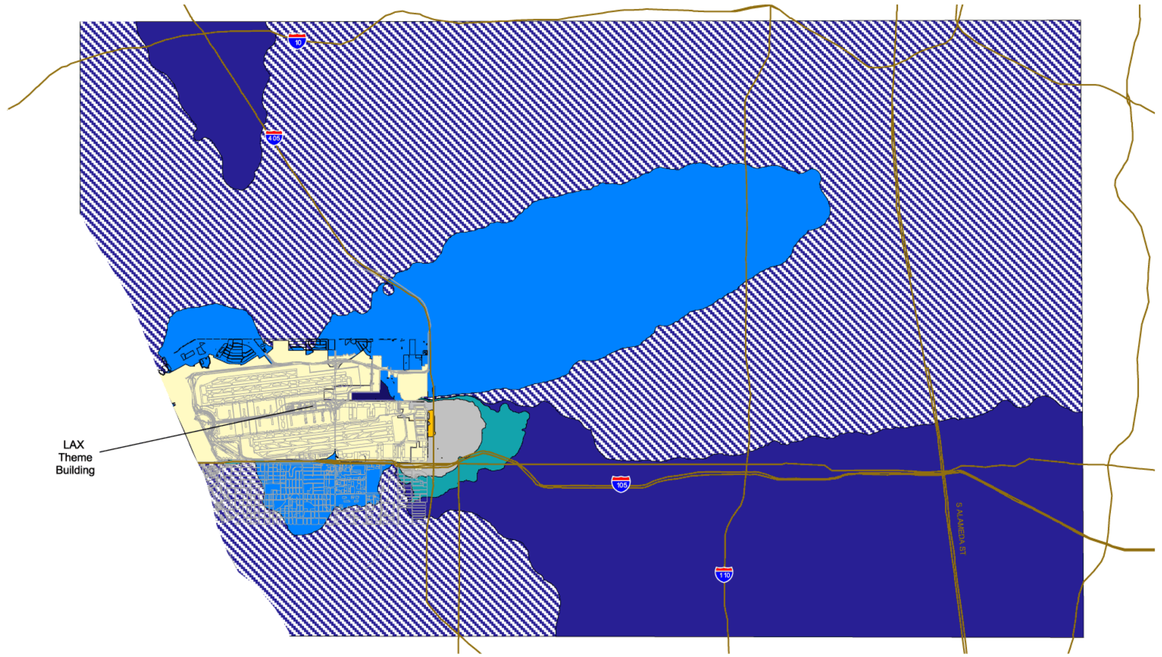
6.5.1 Comparison of On-Airport Air Concentrations with OSHA Standards for Workers

Workers were evaluated by comparing estimated annual air concentrations of TAPs for the build alternatives to eight-hour PEL-TWAs. Estimated on-airport air concentrations and PEL-TWAs for TAPs of concern for LAX are presented in **Table 15,** Comparison of CalOSHA Permissible Exposure Limits to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2005 Post-Mitigation Conditions.

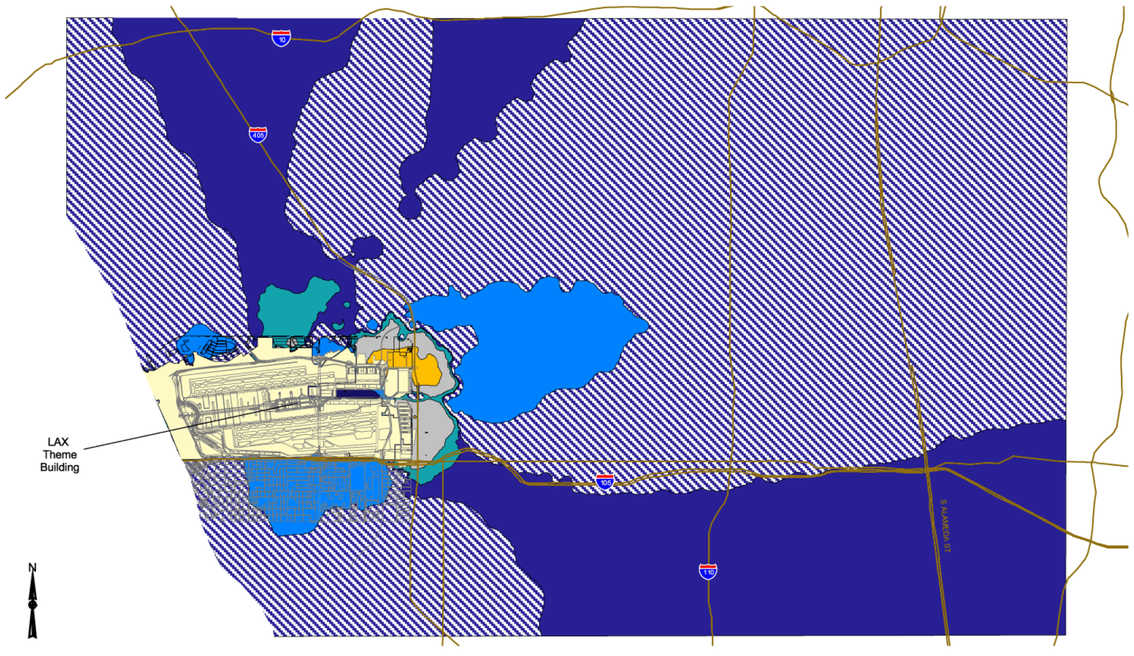
Estimated average annual air concentrations for the build alternatives are well below PEL-TWAs for all TAPs. This result suggests that air concentrations from airport emissions with or without implementation of the LAX Master Plan would not exceed those considered "acceptable" by CalOSHA.



No Action/No Project Alternative



Alternative A



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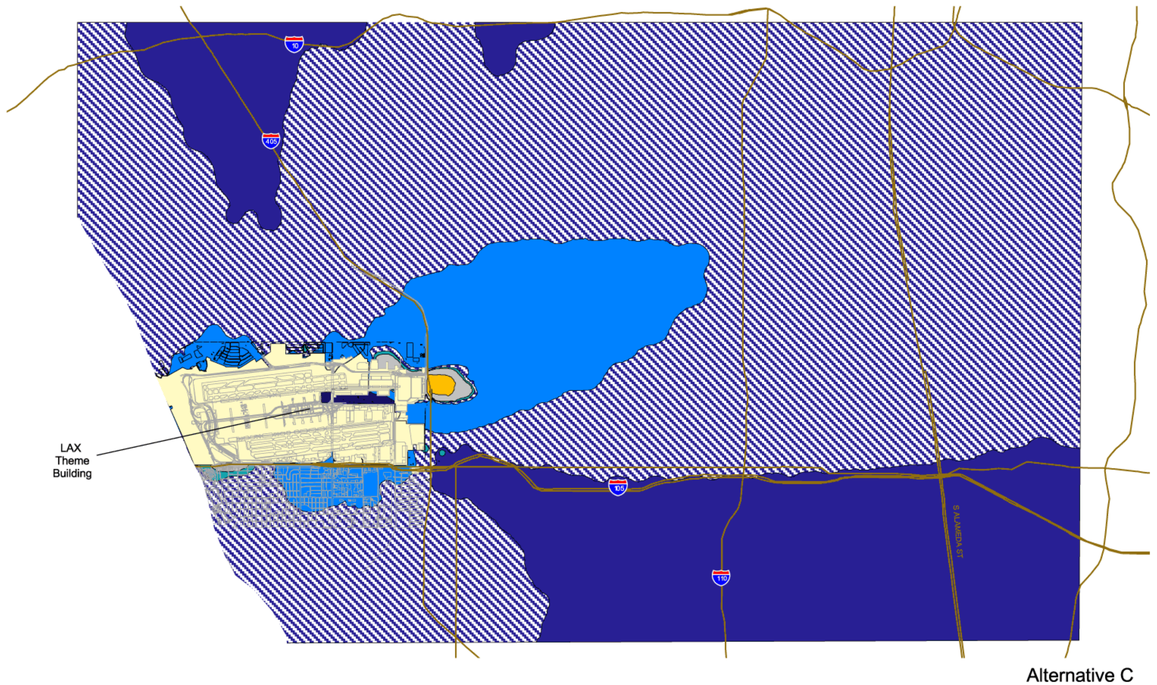

Alternative C

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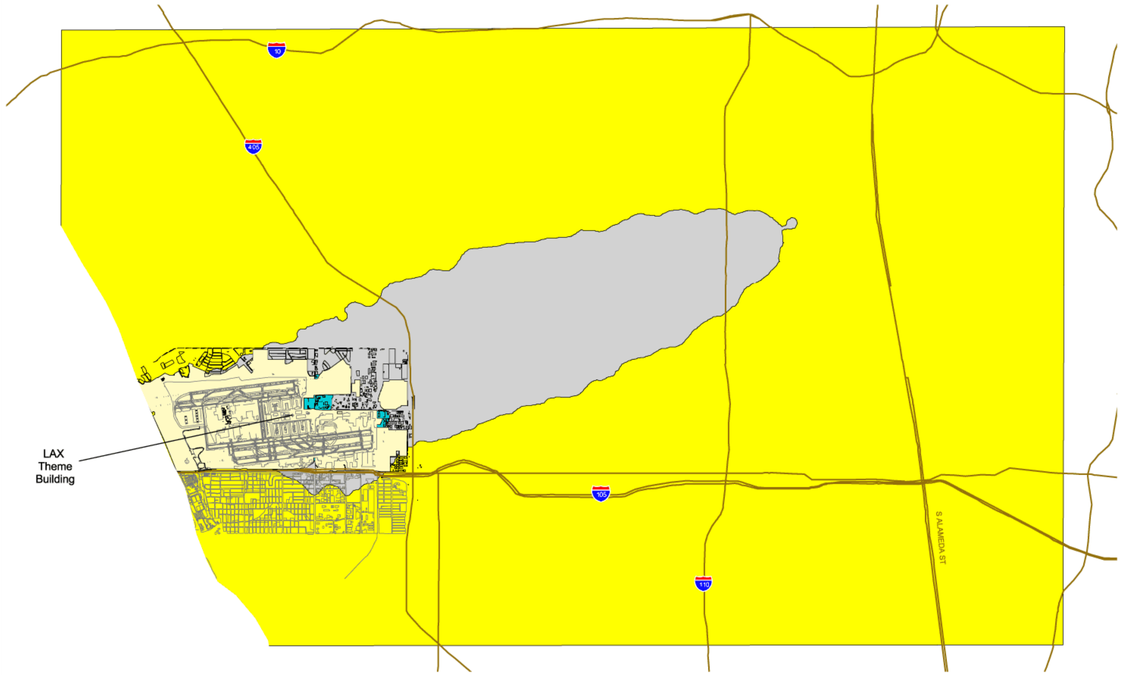
Geographic Extent of Incremental Cancer Risks for Horizon Year 2015 Pre-Mitigation Conditions

Figure 10



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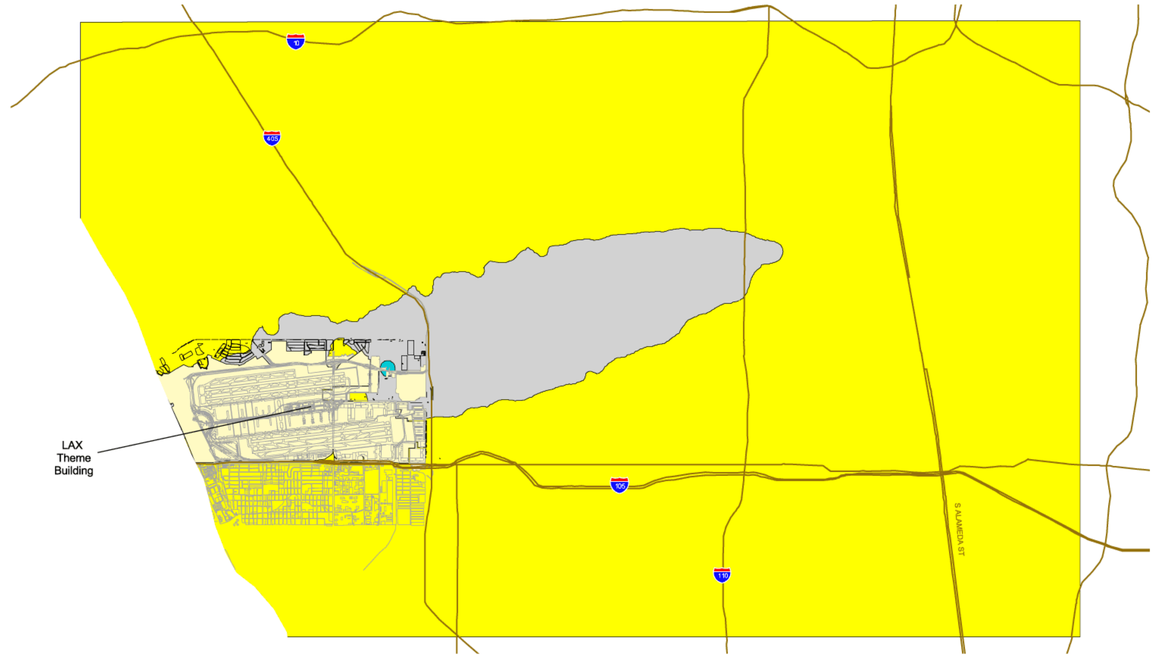
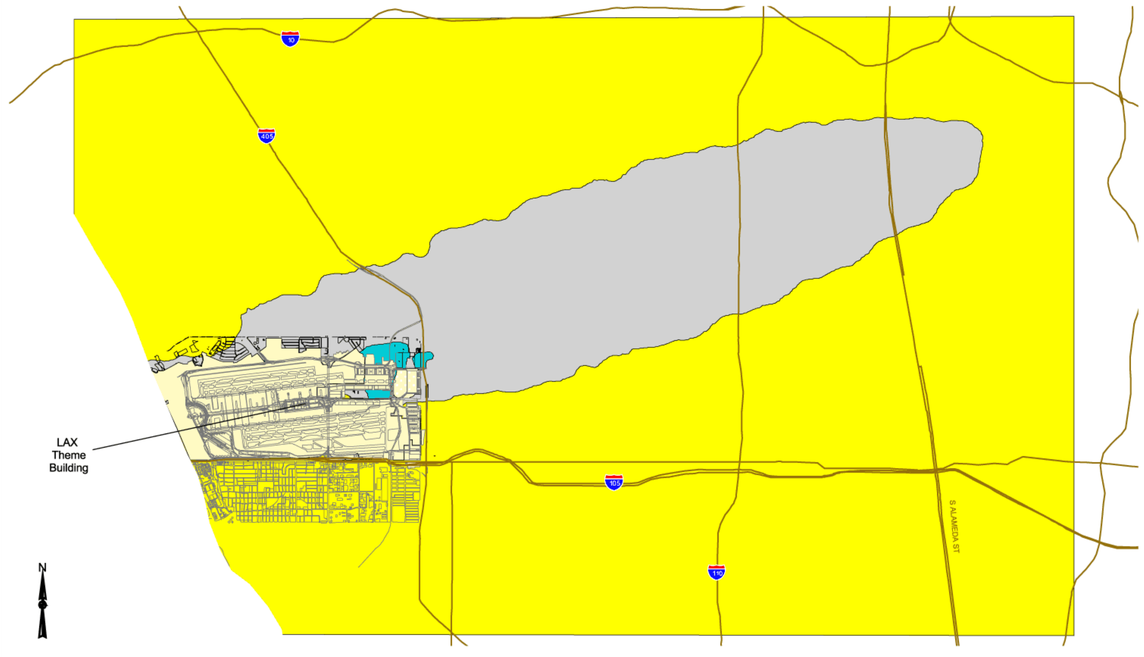
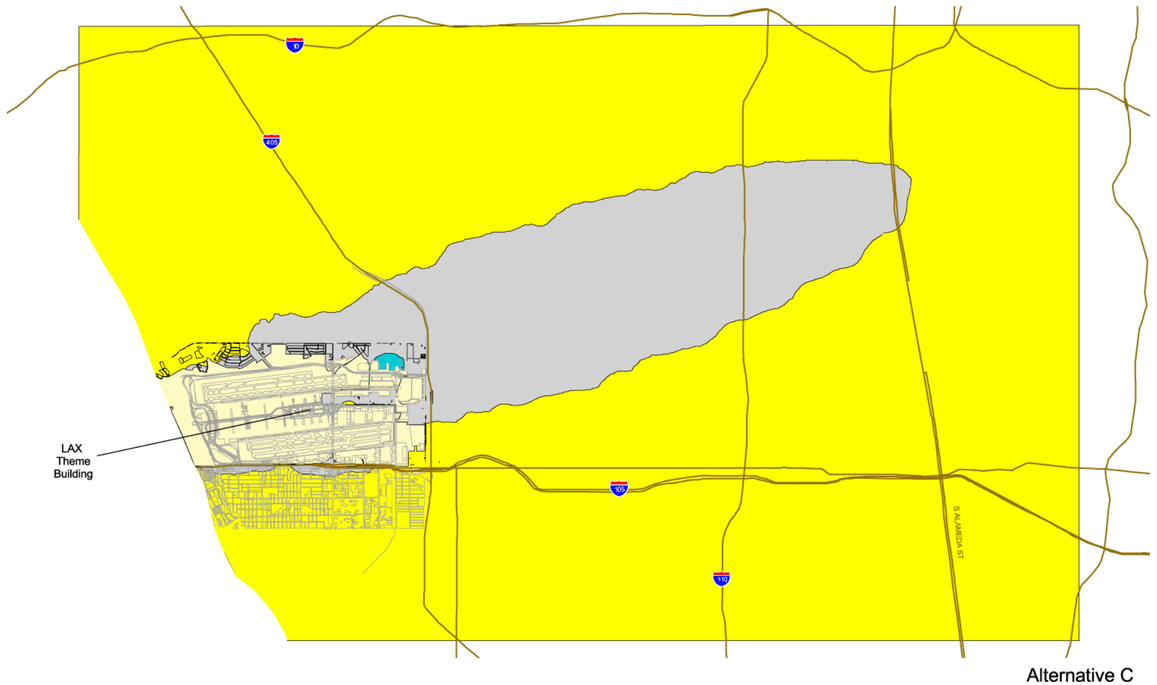


Alternative A



CDM Camp Dresser & McKee Inc.

Alternative B



Alternative C

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Los Angeles International Airport Master Plan

Geographic Extent of Incremental Non-Cancer Health Hazards for Horizon Year 2015 Pre-Mitigation Conditions

Figure 11

Table 15

Comparison of CalOSHA Permissible Exposure Limits to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2005 Post-Mitigation Conditions

TAP[1]	Alternatives A, B, and C (mg/m^3)	CalOSHA PEL-TWA (mg/m^3)[2]
Acetaldehyde	$3.0x10^{-3}$	$1.8x10^{-2}$
Acrolein	$7.3x10^{-4}$	$2.5x10^{-1}$
Benzene	$5.2x10^{-3}$	$3.2x10^{-1}$ [3]
1,3-Butadiene	$1.8x10^{-3}$	$2.2x10^{0}$
Diesel Particulates	$2.9x10^{-3}$	NA
Formaldehyde	$9.2x10^{-2}$	$3.7x10^{-1}$ [3]
Naphthalene	$9.7x10^{-4}$	$5.0x10^{1}$
PAHs as Benzo(a)pyrene equivalents	$5.7x10^{-7}$	NA
TCDD equivalents	$5.4x10^{-10}$	NA
Xylenes	$1.3x10^{-2}$	$4.34x10^{2}$
Arsenic	$9.1x10^{-7}$	$1.0x10^{-2}$
Beryllium	$8.4x10^{-8}$	$2.0x10^{-3}$
Cadmium	$2.6x10^{-6}$	$5.0x10^{-3}$
Chromium (as Cr(VI))	$1.7x10^{-8}$	$5.0x10^{-2}$
Manganese	$5.1x10^{-5}$	$5.0x10^{0}$

NA – Not Available

[1] All TAPs for which PEL-TWAs are available are listed. PEL-TWAs are not available for diesel exhaust, chromium VI and PAHs other than naphthalene.

[2] CalOSHA (California Occupational Safety and Health Administration). 2000. Table AC-1, Permissible Exposure Limits for Chemical Contaminants. http:\www.dir.ca.gov/title8/5155a.htm.

[3] CalOSHA Value not available; value is from American Conference of Governmental Industrial Hygienists (ACGIH), Documentation of the Threshold Limit Values and Biological Exposure Indices, 8th ed., Cincinnati, Ohio, 1998.

Source: Camp Dresser & McKee Inc., 2000.

6.5.2 Incremental Risks for Maximally Exposed Individuals (MEI)

6.5.2.1 Residents (Adults and Children)

For the build alternatives in 2005 with mitigation, the residences with the highest concentrations of TAPs were predicted to be close to the airport boundary near the east end of the north runways (**Figure 12**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2005, Post-Mitigation Conditions). Cancer risk estimates are summarized in **Table 16**, Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2005 Post-Mitigation Conditions.

The total estimated incremental cancer risk for adult residents at this location was 6 in one million. The total estimated cancer risk for young children living at the residence with the maximum predicted TAP concentrations was 4 in one million. Estimated cancer risks are higher for adults than for children, because adult risks included cumulative risks from exposure during childhood and as adults. Incremental risk estimates for post-migration conditions are reduce by about 70 percent from those estimated for pre-mitigation conditions. Mitigation Measures substantially reduce potential cancer risks by reducing jet exhaust release by reducing taxi and queue times and by eliminating diesel GSE.

Cancer risks for adults and children were still mostly due to predicted exposure to diesel particulates and 1,3-butadiene. Diesel contributed 70 percent to estimated cancer risks and 1,3-butadiene contributed 19 percent.

The estimated incremental HI for children living at locations with maximum TAP concentrations was -2, and the HI for maximally exposed adult residents was -1 (**Table 17**, Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2005 Post-Mitigation Conditions). HI estimates are higher for children than adults, because they are normalized to body weight, which is lower for children than for adults. Still HIs for both groups are negative indicating non-cancer health hazards are reduced below baseline.

HI estimates for the build alternatives are reduced over those for pre-mitigation conditions. This reduction is due primarily due to reduction in acrolein emissions predicted for Alternatives A, B, and C. Mitigation, therefore, is predicted to result in build alternatives that have less impact for both cancer risk and non-cancer health hazards than the No Action/No Project Alternative. Mitigation would, in fact, result in a beneficial impact on non-cancer health hazards.

Table 16

Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2005 Post-Mitigation Conditions

	Cancer Risk (per million individuals)		
	Alternatives A, B, and C		
TAP	**Child Resident**	**School Child**	**Adult Resident**
VOCs			
Acetaldehyde	-0.02	-0.001	-0.02
Acrolein	NA	NA	NA
Benzene	-0.7	-0.02	-0.95
1,3-Butadiene	-1.3	-0.07	-1.8
Formaldehyde	-0.1	-0.01	-0.2
Xylene (total)	NA	NA	NA
PAHs			
PAHs as Benzo(a)pyrene equivalents	-0.004	-0.0002	-0.01
Naphthalene	NA	NA	NA
Diesel			
Diesel Particulates	6.0	-0.20	8.6
Dioxins			
TCDD equivalents	-0.06	-0.001	-0.08
Metals			
Arsenic	-0.005	-0.0004	-0.01
Beryllium	-0.001	-0.0001	-0.002
Cadmium	-0.01	-0.0001	-0.01
Chromium (VI)	-0.02	-0.0015	-0.03
Manganese	NA	NA	NA
Total	**4**	**-0.3**	**6**

NA – Not Available

Source: Camp Dresser & McKee Inc, 2000.

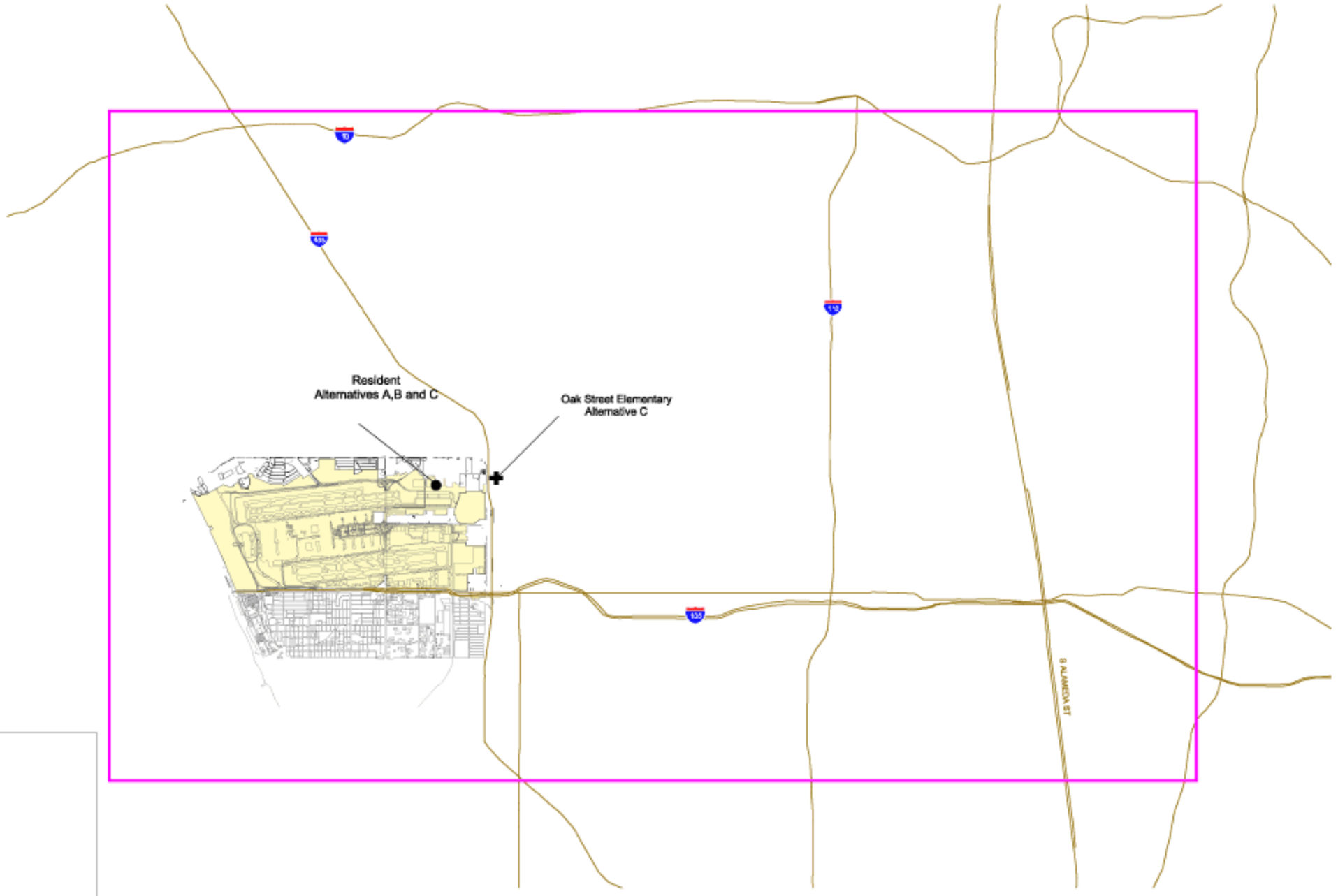
6.5.2.2 School Children

Maximum TAP concentrations under the build alternatives were predicted for the Oak Street School near the current eastern LAX fence line (**Figure 12**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2005, Post-Mitigation Conditions).

The estimated incremental cancer risk for children attending this school is -0.3 in one million. The estimated HI for chemicals affecting the same target organ (i.e., the respiratory system) for school children is -0.2. Both cancer risks and HIs are lower for the build alternatives, than those predicted for the No Action/No Project Alternative for 2005. In fact, beneficial impacts are predicted for both cancer risks and non-cancer hazards. Negative incremental risks or hazards indicated that risks for the build alternatives are less than the risks or hazards for baseline conditions.

6.5.2.3 Risks Described Geographically

For the build alternatives and the No Action/No Project Alternative, total cancer risks and HIs for all respiratory irritants (acetaldehyde, acrolein, formaldehyde, diesel particulates, beryllium, chromium VI, and manganese) were calculated for each grid node in the ISC3 modeling domain. Risks and HIs were then used to generate estimates of risks and hazards on a spatial basis as overlays for maps of LAX and surrounding communities. All risk and hazard estimates used to describe risk geographically were for residents living within the study area.



Note: Airport Boundary Footprint shown is for Alternative C.

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Locations for Maximally Exposed Residents and School Children for Horizon Year 2005 Post-Mitigation Conditions

Figure 12

Table 17

Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2005 Post-Mitigation Conditions

	Hazard Quotient		
	Alternatives A, B, and C		
TAP	**Child Resident**	**School Child**	**Adult Resident**
VOCs			
Acetaldehyde	-0.008	-0.0005	-0.002
Acrolein	-1.8	-0.2	-0.5
Benzene	NA	NA	NA
1,3-Butadiene	NA	NA	NA
Formaldehyde	-0.0003	-0.00002	-0.0001
Xylene (total)	-0.00005	-0.0000004	-0.00001
PAHs			
Naphthalene	-0.03	-0.002	-0.008
Dioxins			
TCDD equivalents	NA	NA	NA
Diesel			
Diesel Particulates	0.04	-0.001	0.01
Metals			
Arsenic	-0.00002	-0.000002	-0.000005
Beryllium	-0.0004	-0.00003	-0.0001
Cadmium	-0.0001	-0.000002	-0.00002
Chromium (VI)	0.0000	-0.000001	-0.000005
Manganese	-0.016	-0.001	-0.005
Total Hazard Index	**-2.0**	**-0.2**	**-1.0**
Total HI for Respiratory Effects	**-2.0**	**-0.2**	**-1.0**

NA – Not Available

Source: Camp Dresser & McKee Inc., 2000.

Results for post-mitigation conditions in 2005 are presented in **Figure 6**, Geographic Extent of Incremental Cancer Risks for Horizon Year 2005, Pre- and Post-Mitigation Conditions. This figure depicts ranges of incremental cancer risks from -1 in 100 million to 10 in one million. Negative values indicate that cancer risks would be reduced when compared to risks associated with baseline (1996) conditions; positive values indicate an incremental increase in cancer risk compared to this baseline. Estimated cancer risks for the No Action/No Project Alternative under pre-mitigation conditions are presented for comparison. For this alternative, modeling under post-mitigation conditions was not conducted because mitigation opportunities are severely limited by existing facilities. Results are therefore compared to pre-mitigation conditions for the No Action/No Project Alternative.

Incremental upper range cancer risk estimates exceeding 10 in one million were not estimated for the build alternatives. Estimated risks associated with post mitigation conditions ranged from -100 in one million to -0.01 in one million (**Figure 6**, Geographic Extent of Incremental Cancer Risks for Horizon Year 2005, Pre- and Post-Mitigation Conditions). Risks are thus substantially reduced over those estimated for pre-mitigation conditions, for which risks greater than one in one million extended approximately 20,000 feet east, northeast from the LAX fenceline.

Risks associated with the three build alternatives are substantially smaller than those estimated for the No Action/No Project Alternative. Cancer risks that exceed 10 in one million under the No Action/No Project Alternative are predicted in an area extending east-northeast from the eastern LAX boundary a distance of approximately 24,000 feet (4.5 miles) from the LAX theme building. In contrast, incremental cancer risks in virtually all of this area would be reduced by a factor of 1 in one million or more under Alternative C after mitigation. The effect of implementing the LAX Master Plan in any of its three forms would convert a substantial adverse impact predicted in the absence of airport expansion into a beneficial impact.

Non-cancer health effects for the No Action/No Project Alternative and Alternatives A, B, and C are depicted in **Figure 7**, Geographical Extent of Non-Cancer Health Hazards for Horizon Year 2005, Pre- and Post-Mitigation Conditions. This figure depicts the extent of non-cancer HIs greater than 5, between 5 and 1 and less than 1. Estimated HIs for all gridpoints were less than one for the build alternatives. HI

estimates are thus greatly reduced over pre-mitigation conditions and the No Action/No Project Alternative.

Importantly, for a large area extending east-northeast from the LAX boundary almost to Interstate Highway 110 (I-110), incremental hazard indices between 1 and 5 under the No Action/No Project Alternative are completely eliminated under Alternatives A, B, and C post-mitigation. Implementing one of the three build alternatives with mitigation would have a substantial beneficial impact when compared with predicted health impacts from LAX under the No Action/No Project Alternative.

6.6 Risk Estimates for Post-Mitigation Conditions for Horizon Year 2015

The preliminary risk estimates for 2015 Post-Mitigation conditions were quantitatively evaluated for Alternatives A, B, and C. See Section 7.5, *Uncertainties in Mitigated Impacts*, for a discussion of the uncertainties in the preliminary post-mitigation impacts.

6.6.1 Comparison of On-Airport Air Concentrations with OSHA Standards for Workers

Workers are evaluated by comparing estimated annual air concentrations of TAPs for the different alternatives to eight-hour PEL-TWAs. Estimated on-airport air concentrations and PEL-TWAs for TAPs of concern for LAX are presented in **Table 18**, Comparison of CalOSHA Permissible Exposure Limits to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2015 Post-Mitigation Conditions.

Post-Mitigation Conditions were not evaluated for the No Action/No Project Alternative. Estimated maximum 8-hour on-airport concentrations for the three build alternatives are similar for most TAPs.

Estimated average annual air concentrations for the three build alternatives are well below PELs for all TAPs. This result suggests that air concentrations from airport emissions with or without implementation of the LAX Master Plan would not exceed those considered "acceptable" by CalOSHA.

Table 18

Comparison of CalOSHA Permissible Exposure Limits to Maximum Estimated 8-Hour On-Airport Air Concentrations for 2015 Post-Mitigation Conditions

TAP[1]	Alternative				CalOSHA PEL-TWA (mg/m^3)[2]
	No Action/No Project	A	B	C	
Acetaldehyde	NC	$1.7x10^{-3}$	$1.9x10^{-3}$	$1.6x10^{-3}$	$1.8x10^{2}$
Acrolein	NC	$8.1x10^{-4}$	$8.8x10^{-4}$	$7.3x10^{-4}$	$2.5x10^{-1}$
Benzene	NC	$1.6x10^{-3}$	$2.2x10^{-3}$	$2.8x10^{-3}$	$3.2x10^{-1}$ [3]
1,3-Butadiene	NC	$1.1x10^{-3}$	$1.2x10^{-3}$	$9.5x10^{-4}$	$2.2x10^{0}$
Diesel Particulates	NC	$1.3x10^{-3}$	$7.7x10^{-4}$	$7.5x10^{-4}$	NA
Formaldehyde	NC	$5.7x10^{-3}$	$6.2x10^{-3}$	$5.0x10^{-3}$	$3.7x10^{-1}$ [3]
Naphthalene	NC	$1.1x10^{-3}$	$1.1x10^{-3}$	$8.8x10^{-4}$	$5.0x10^{1}$
PAHs as Benzo(a)pyrene equivalents	NC	$5.5x10^{-7}$	$5.6x10^{-7}$	$4.7x10^{-7}$	NA
TCDD equivalents	NC	$1.3x10^{-10}$	$2.0x10^{-10}$	$3.1x10^{-10}$	NA
Xylenes	NC	$2.3x10^{-3}$	$4.2x10^{-3}$	$7.1x10^{-3}$	$4.34x10^{2}$
Arsenic	NC	$5.5x10^{-7}$	$8.9x10^{-7}$	$8.9x10^{-7}$	$1.0x10^{-2}$
Beryllium	NC	$1.4x10^{-7}$	$1.5x10^{-7}$	$1.2x10^{-7}$	$2.0x10^{-3}$
Cadmium	NC	$5.1x10^{-6}$	$3.4x10^{-6}$	$2.3x10^{-6}$	$5.0x10^{-3}$
Chromium (as Cr(VI))	NC	$2.8x10^{-8}$	$2.9x10^{-8}$	$2.4x10^{-8}$	$5.0x10^{-2}$
Manganese	NC	$2.8x10^{-5}$	$4.9x10^{-5}$	$5.0x10^{-5}$	$5.0x10^{0}$

NA – Not Available
NC – Not Calculated

1 All TAPs for which PEL-TWAs are available are listed. PEL-TWAs are not available for diesel exhaust, chromium VI and PAHs other than naphthalene.

2 CalOSHA (California Occupational Safety and Health Administration). 2000. Table AC-1, Permissible Exposure Limits for Chemical Contaminants. http:\www.dir.ca.gov/title8/5155a.htm.

3 CalOSHA Value not available; value is from American Conference of Governmental Industrial Hygienists (ACGIH), Documentation of the Threshold Limit Values and Biological Exposure Indices, 8th ed., Cincinnati, Ohio, 1998.

Source: Camp Dresser & McKee Inc., 2000.

6.6.2 Incremental Risks for Maximally Exposed Individuals (MEI)

6.6.2.1 Residents (Adults and Children)

Locations for residences with the highest TAP concentrations for each of the three build alternatives are shown in **Figure 13**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2015, Post-Mitigation Conditions. Total estimated cancer risks for adult residents were -18 in one million, -4 in one million, and -11 in one million for Alternatives A, B, and C, respectively (**Table 19**, Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2015 Post-Mitigation Conditions). Total estimated cancer risks for young children living at residences with maximum predicted TAP concentrations were -13 in one million, -3 in one million, and -8 in one million for Alternatives A, B, and C, respectively. Estimated cancer risks are higher for adults than for children, because exposure duration for adults is longer.

Negative incremental cancer risks indicate that implementation of any of the build alternatives after mitigation would result in a decrease in health impacts associated with release of TAPs from LAX compared to the baseline (1996). In contrast, the No Action/No Project Alternative would result in substantial increase in incremental cancer risk compared to baseline conditions. The LAX Master Plan with mitigation would result in a beneficial impact for human health concerns for many areas adjacent to and near LAX.

Estimated HIs for children living at locations with maximum TAP concentrations are -1, 4, and 0.7 for Alternatives A, B, and C, respectively, (**Table 20**, Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2015 Post-Mitigation Conditions). Incremental HIs for maximally exposed adult residents are -0.4, 1, and 0.2 for Alternatives A, B, and C, respectively. HI estimates are higher for children than adults, because they are normalized to body weight, which is lower for children than for adults. HI estimates are slightly higher for Alternative B than for Alternatives A and C.

Importantly, for a large area extending east-northeast from the LAX boundary almost to I-110, incremental hazard indices between 1 and 5 under the No Action/No Project Alternative are completely eliminated under any of the three build alternatives after mitigation. Implementing one of the three build alternatives with mitigation would have a substantial beneficial impact when compared with predicted health impacts from LAX under the No Action/No Project Alternative. For Alternative A, incremental hazard indices are negative indicating that non-cancer health impacts under this alternative would actually be lower than hazards predicted for baseline (1996) conditions.

Acrolein is the only chemical for which the HQ exceeds one. Acrolein contributes more than 97 percent to the total HI estimates for all alternatives.

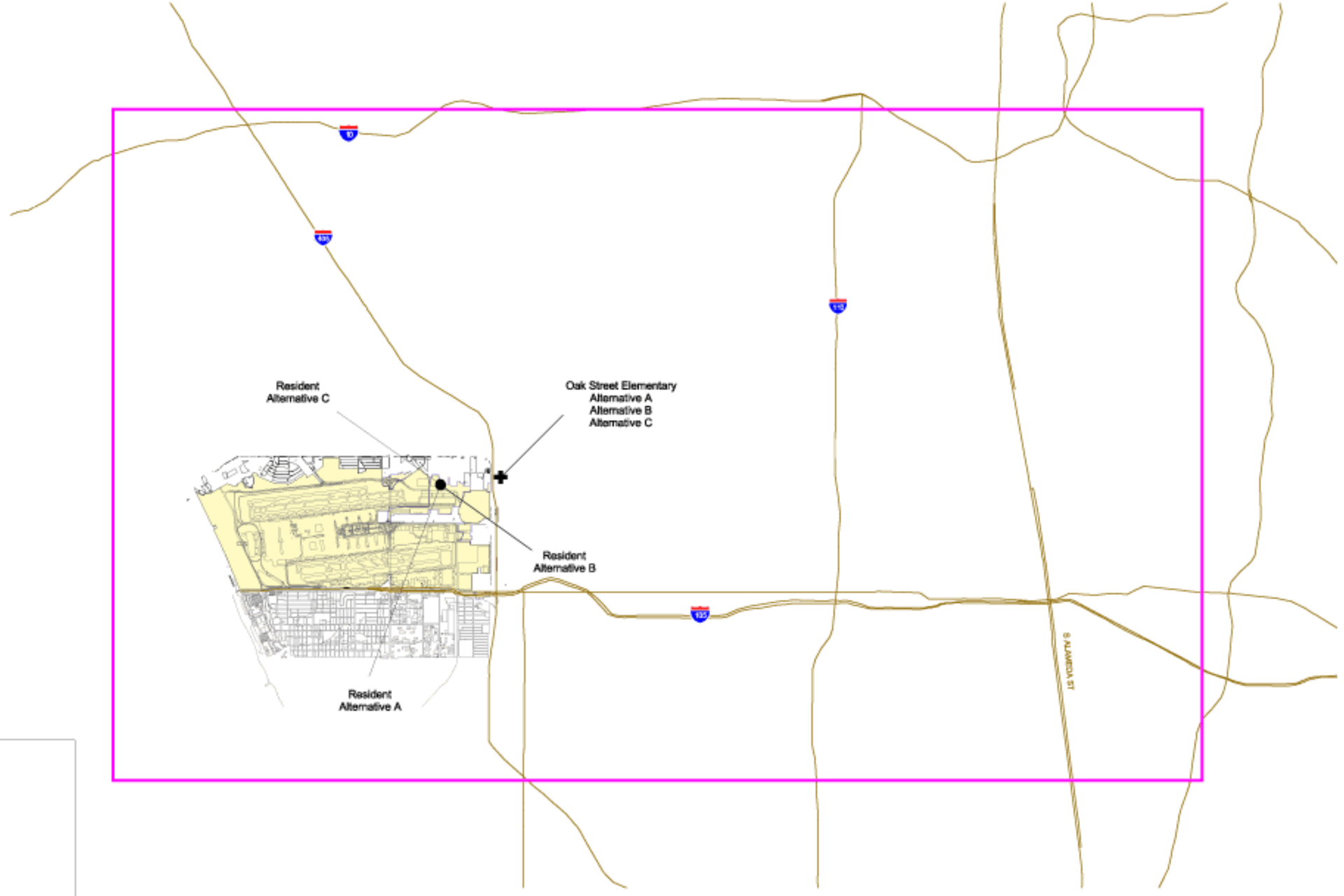
Table 19

Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2015 Post-Mitigation Conditions

	Cancer Risk (per million individuals)								
	Alternative A			Alternative B			Alternative C		
TAP	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident
VOCs									
Acetaldehyde	-0.02	-0.0009	-0.03	0.03	0.0007	0.04	0.001	-0.0004	0.002
Acrolein	NA	NA	NA	NA	NA	NA	NA	NA	NA
Benzene	-0.7	-0.03	-1	-0.5	-0.01	-0.6	-0.7	-0.03	-1
1,3-Butadiene	-1	-0.06	-2	0.6	-0.003	0.8	-0.5	-0.04	-0.7
Formaldehyde	-0.1	-0.008	-0.2	0.2	0.004	0.3	-0.003	-0.004	-0.005
Xylene (total)	NA	NA	NA	NA	NA	NA	NA	NA	NA
PAHs									
PAHs as Benzo(a)pyrene equivalents	-0.0007	-0.00004	-0.001	0.004	0.00009	0.006	0.001	-0.00002	0.002
Naphthalene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Diesel									
Diesel Particulates	-0.06	-0.0006	-0.08	-0.03	0.0005	-0.04	-0.05	-0.0006	-0.07
Dioxins									
TCDD equivalents	-10	-0.6	-15	-3	-0.3	-5	-7	-0.5	-9
Metals									
Arsenic	0.007	0.0003	0.01	0.02	0.0007	0.03	0.01	0.0004	0.02
Beryllium	0.0008	0.00003	0.001	0.003	0.00007	0.004	0.003	0.00002	0.004
Cadmium	0.003	0.002	0.004	0.1	0.006	0.2	0.006	0.002	0.008
Chromium (VI)	0.01	0.0004	0.02	0.04	0.001	0.06	0.02	0.0003	0.03
Manganese	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total	**-13**	**-0.7**	**-18**	**-3**	**-0.3**	**-4**	**-8**	**-0.5**	**-11**

NA – Not Available
Risk estimates rounded to one significant figure.

Source: Camp Dresser & McKee Inc., 2000.



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CDM Camp Dresser & McKee Inc.

Note: Airport Boundary Footprint shown is for Alternative C.

Los Angeles International Airport Master Plan	**Locations for Maximally Exposed Residents and School Children for Horizon Year 2015 Post-Mitigation Conditions**	Figure 13

Table 20

Estimated Incremental Non-Cancer Health Hazards for Maximally Exposed Individuals for 2015 Post-Mitigation Conditions

	Hazard Quotient								
	Alternative A			Alternative B			Alternative C		
TAP	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident	Child Resident	School Child	Adult Resident
VOCs									
Acetaldehyde	-0.009	-0.0004	-0.003	0.01	0.0004	0.004	0.0007	-0.0002	0.0002
Acrolein	-1	-0.1	-0.4	3.5	0.03	1	0.7	-0.07	0.2
Benzene	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,3-Butadiene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Formaldehyde	-0.0004	-0.00002	-0.0001	0.0006	0.00001	0.0002	-0.000009	-0.00001	-0.000003
Xylene (total)	-0.00007	-0.000001	-0.00002	-0.00005	-0.0000002	-0.00002	-0.00007	-0.000001	-0.00002
PAHs									
Naphthalene	-0.005	-0.0002	-0.001	0.03	0.0008	0.009	0.01	0.00006	0.003
Dioxins									
TCDD equivalents	NA	NA	NA	NA	NA	NA	NA	NA	NA
Diesel									
Diesel Particulates	-0.08	-0.004	-0.02	-0.03	-0.002	-0.007	-0.05	-0.004	-0.01
Metals									
Arsenic	0.00002	0.000001	0.000007	0.00007	0.000003	0.00002	0.00004	0.000001	0.00001
Beryllium	0.0002	0.000007	0.00006	0.0008	0.00002	0.0002	0.0008	0.000005	0.0002
Cadmium	0.00004	0.00003	0.00001	0.002	0.00008	0.0005	0.00008	0.00002	0.00002
Chromium (VI)	0.000009	0.0000003	0.000003	0.00003	0.0000008	0.000009	0.00002	0.0000001	0.000005
Manganese	0.03	0.001	0.008	0.08	0.003	0.02	0.05	0.002	0.01
Total Hazard Index	**-1**	**-0.1**	**-0.4**	**4**	**0.03**	**1**	**0.7**	**-0.07**	**0.2**
Total HI for Respiratory Effects	**-1**	**-0.1**	**-0.4**	**4**	**0.03**	**1**	**0.7**	**-0.07**	**0.2**

NA – Not Available

Source: Camp Dresser & McKee Inc., 2000.

6.6.2.2 School Children

Maximum TAP concentrations under Alternatives A, B, and C were predicted to be highest for the Oak Street Elementary School. The locations of this school is shown in **Figure 13**, Locations for Maximally Exposed Residents and School Children for Horizon Year 2015, Post-Mitigation Conditions.

Estimated cancer risks for children attending Oak Street Elementary are -0.7 in one million, -0.3 in one million, and -0.5 in one million for Alternatives A, B, and C, respectively (**Table 19**, Estimated Incremental Cancer Risks for Maximally Exposed Individuals for 2015 Post-Mitigation Conditions). All risk estimates are less than risks associated with baseline conditions.

Negative incremental cancer risks indicate that implementation of any of the build alternatives after mitigation would result in a decrease in health impacts associated with release of TAPs from LAX compared to the baseline (1996). In contrast, the No Action/No Project Alternative would result in substantial increased incremental cancer risk compared to baseline conditions. The LAX Master Plan with mitigation would result in a beneficial impact for human health concerns for many areas adjacent to and near LAX.

Estimated total HIs for chemicals affecting the same target (i.e., the respiratory system) for schoolchildren at the schools with the highest estimated TAP concentrations are -0.1, 0.03, and -0.1 for Alternatives A, B, and C, respectively (**Table 20,** Estimated Non-Cancer Health Hazards for Maximally Exposed Individuals for 2015 Post-Mitigation Conditions).

Negative incremental hazard indices for Alternatives A and C indicate that implementation of these alternatives with mitigation would result in a decrease in health impacts associated with release of TAPs from LAX compared to the baseline (1996). In contrast, the No Action/No Project Alternative would result in increased incremental health hazards compared to baseline conditions. Alternatives A and C with

mitigation would result in a beneficial impact for human health concerns for many areas adjacent to and near LAX.

6.6.3 Risks Described Geographically

For each build alternative and the No Action/No Project Alternative, total cancer risks, and HIs for all respiratory irritants (acetaldehyde, acrolein, formaldehyde, diesel particulates, beryllium, chromium VI, and manganese) were calculated for each grid node in the ISC3 modeling domain. Risks and HIs were then used to generate estimates of risks and hazards on a spatial basis as overlays for maps of LAX and surrounding communities. All risk and hazard estimates used to describe risk geographically were for residents living within the study area. Cancer risk results for post-mitigation conditions in 2015 are presented in **Figure 14**, Geographic Extent of Incremental Cancer Risks for Horizon Year 2015, Post-Mitigation Conditions. This figure depicts ranges of incremental cancer risks from -1 in 100 million to 10 in one million. Negative values indicate that cancer risks would be reduced when compared to risks associated with baseline (1996) conditions; positive values indicate an incremental increase in cancer risk compared to this baseline.

Estimated incremental upper range cancer risk estimates are greatly reduced over 2015 pre-mitigation conditions for all build alternatives (see **Figure 10,** Geographic Extent of Incremental Cancer Risks for Horizon Year 2015, Pre-Mitigation Conditions. For post-mitigation conditions, areas of impact are much smaller for all of the build alternatives than for the No Action/No Project Alternative. Mitigation would have a substantial positive effect in reducing potential for human health impacts east of LAX.

Cancer risks that exceed 10 in one million under the No Action/No Project Alternative are predicted in an area extending east-northeast from the eastern LAX boundary a distance of approximately 24,000 feet (4.5 miles) from the LAX theme building. In contrast, incremental cancer risks in virtually all of this area would be reduced by 1 in one million or more under any of the build alternatives after mitigation. Implementing any of the three build alternatives would convert a predicted substantial adverse impact in the absence of airport expansion into a beneficial impact.

Non-cancer health effects for the No Action/No Project Alternative and Alternative C are depicted in **Figure 11**, Geographic Extent of Incremental Non-Cancer Health Hazards for Horizon Year 2015, Pre-Mitigation Conditions. This figure depicts the extent of non-cancer HIs greater than 5, between 5 and 1, and less than 1.

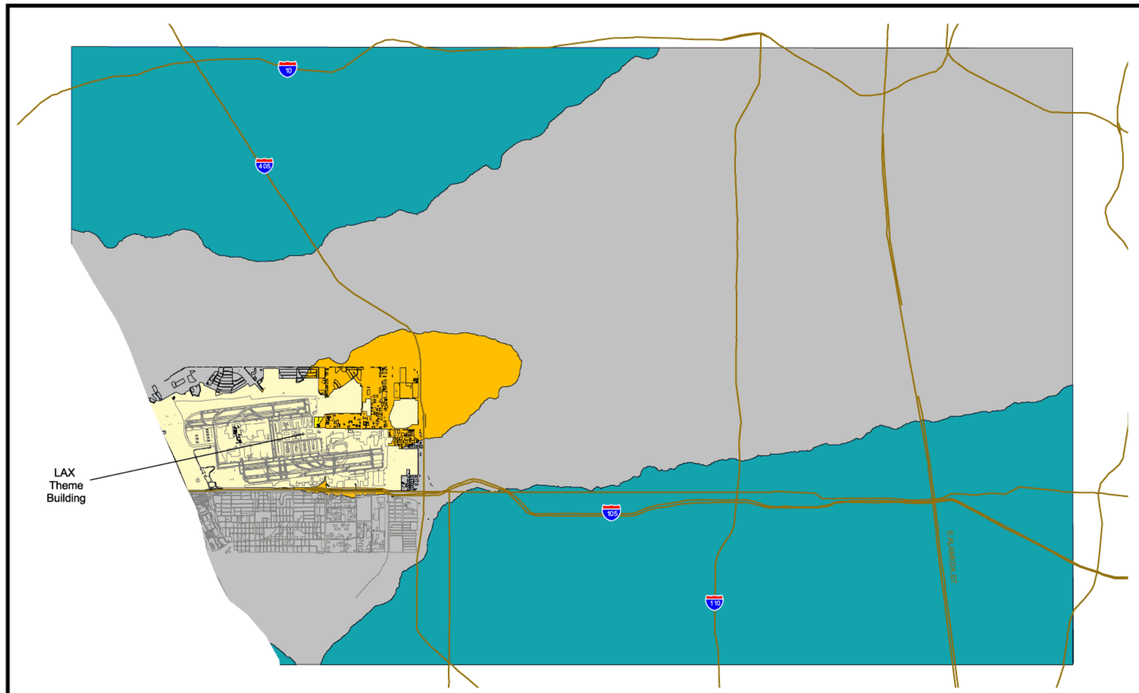
For 2015 post-mitigation conditions, estimated upper range non-cancer HIs for respiratory effects are greatly reduced over those for pre-mitigation conditions for all build alternatives (see **Figure 15**, Geographic Extent of Incremental Non-Cancer Health Hazards for Horizon Year 2015, Post-Mitigation Conditions). Importantly, areas of impact are also much smaller for all of the build alternatives than for the No Action/No Project Alternative. Mitigation has a substantial positive effect in reducing potential for human health impacts east of LAX.

Importantly, for a large area extending east-northeast from the LAX boundary almost to I-110, incremental hazard indices between 1 and 5 under the No Action/No Project Alternative are completely eliminated under any of the build alternatives with mitigation. Implementing any of the three build alternatives with mitigation would have a substantial beneficial impact when compared with predicted health impacts from LAX under the No Action/No Project Alternative. For Alternatives A and C, incremental hazard indices are mostly negative indicating that non-cancer health impacts under these alternatives would actually be lower than hazards predicted for baseline (1996) conditions.

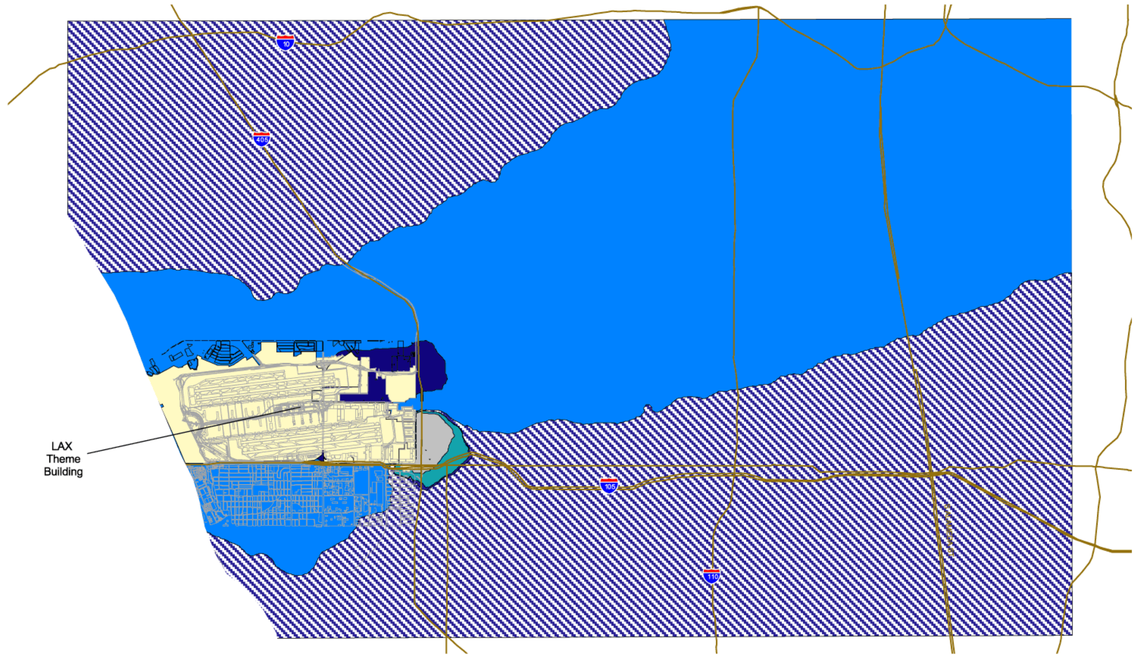
6.7 Cumulative Risks Associated with LAX Operations

Estimated risks for LAX operations are compared to risks associated with other sources to determine the impact of LAX operations on cumulative risks for people living in the South Coast Air Basin. This comparison is facilitated by the recent release of the MATES-II,[39] which provides a general evaluation of cancer risks associated with TAPs from all sources within the South Coast Air Basin.

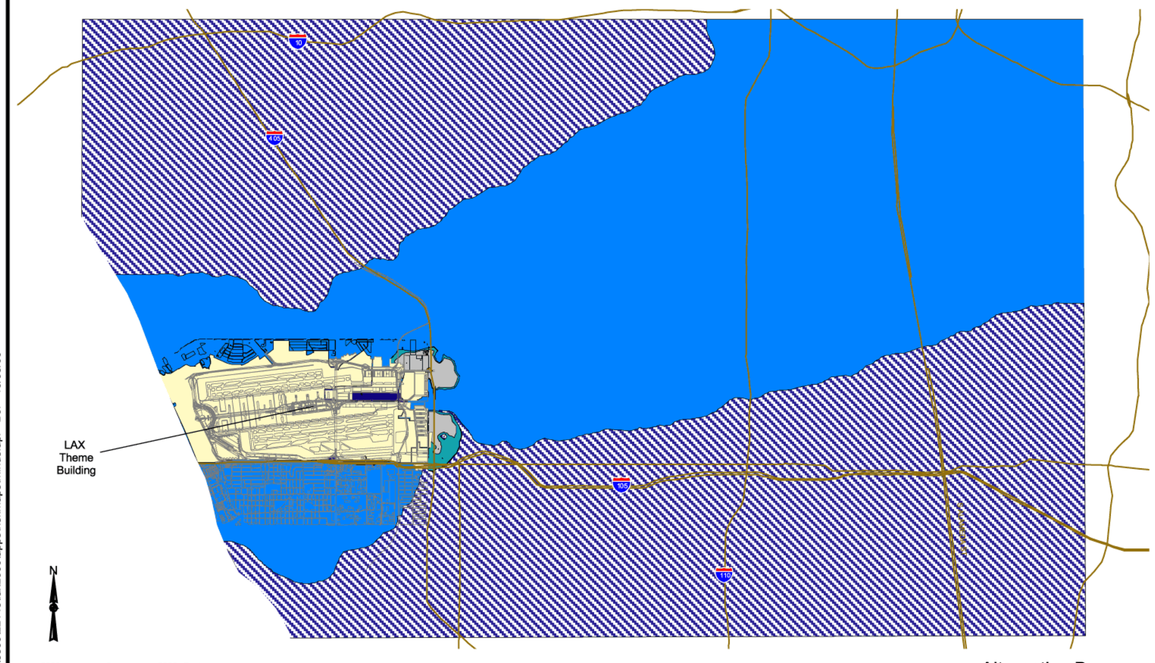
[39] SCAQMD, Multiple Air Toxics Exposure Study in the South Coast Air Basin (MATES-II), November 1999.



No Action/No Project Alternative



Alternative A



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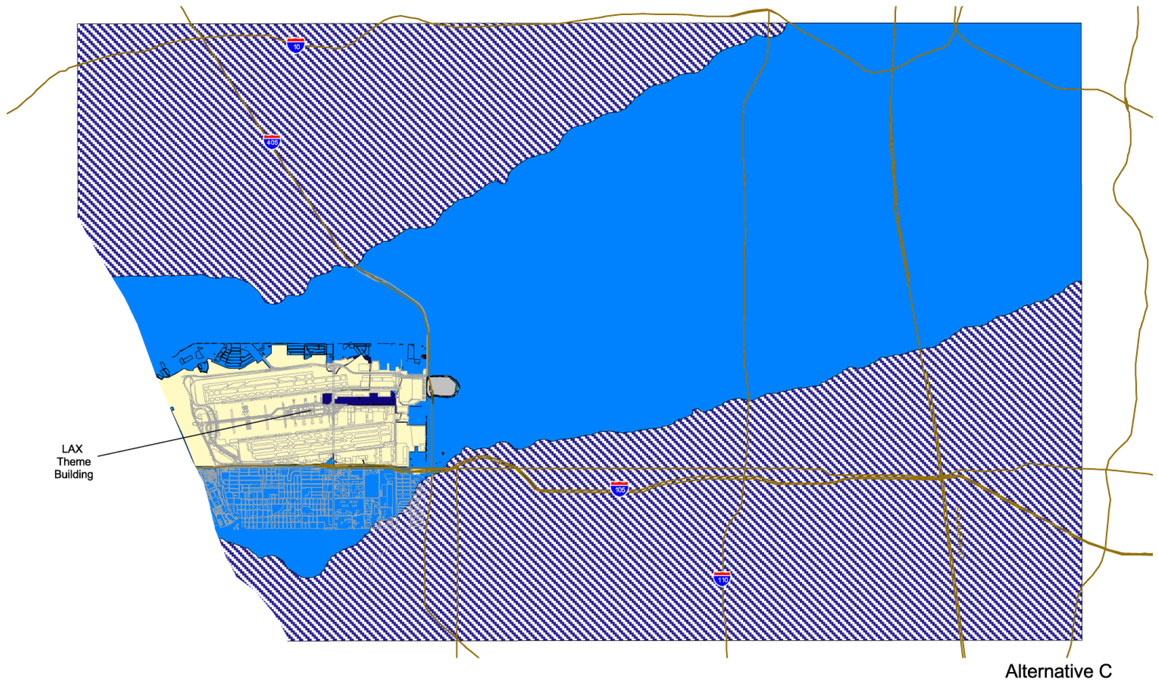

Alternative C

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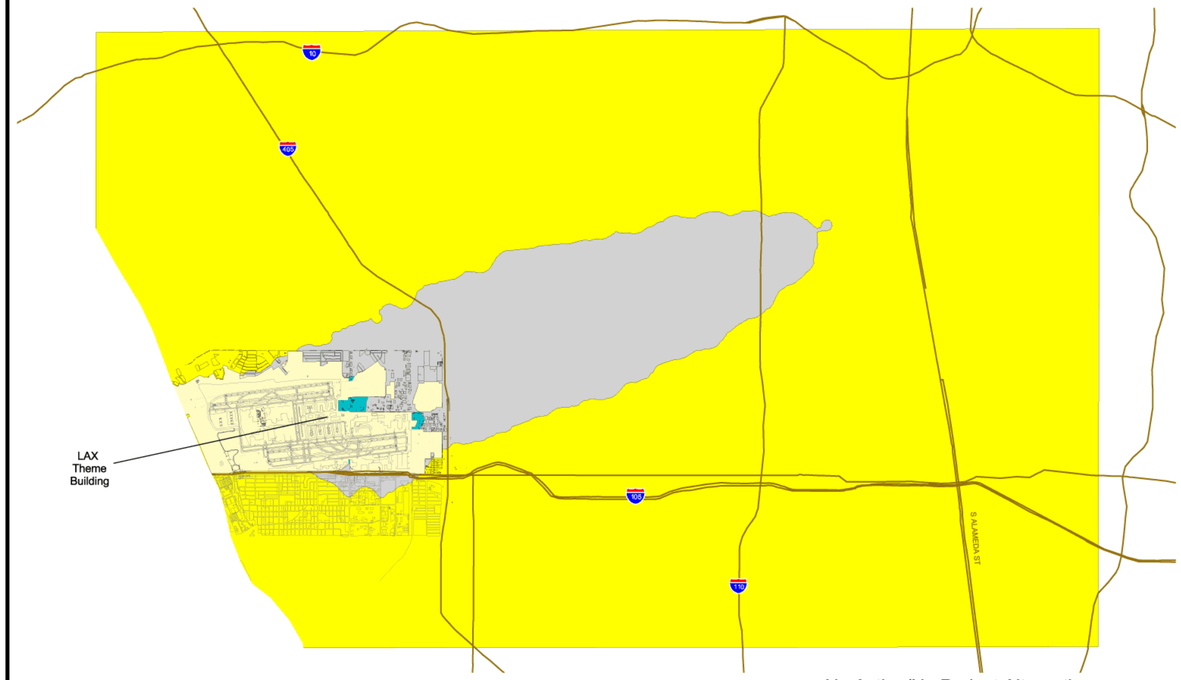
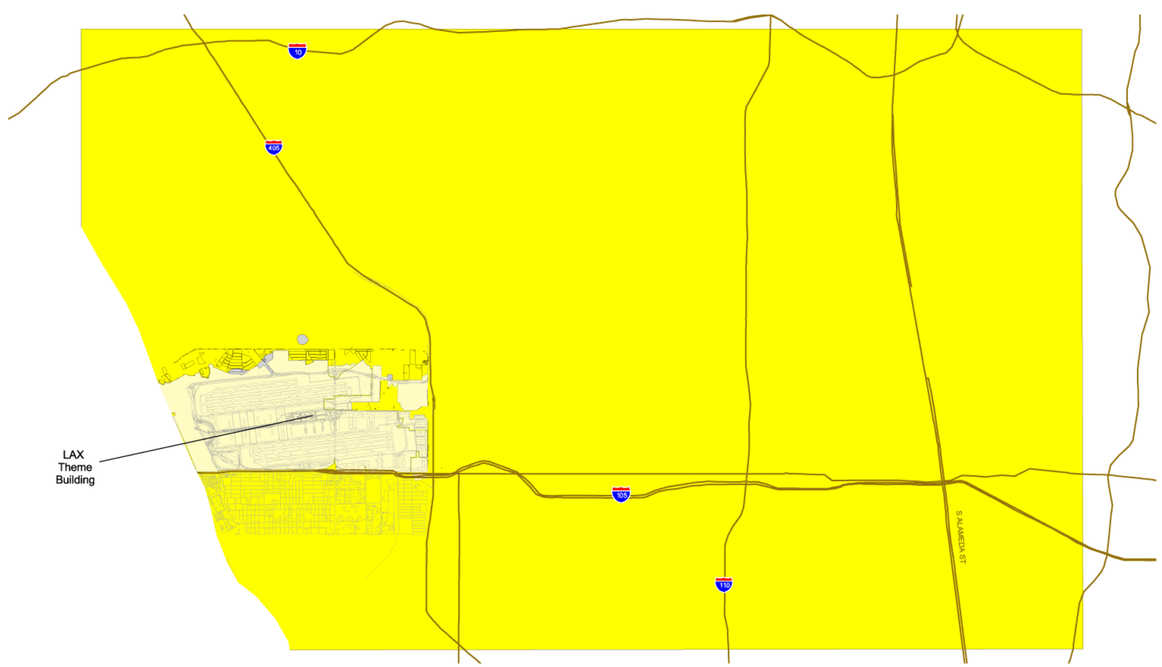
Los Angeles International Airport Master Plan

Geographic Extent of Incremental Cancer Risks for Horizon Year 2015 Post-Mitigation Conditions

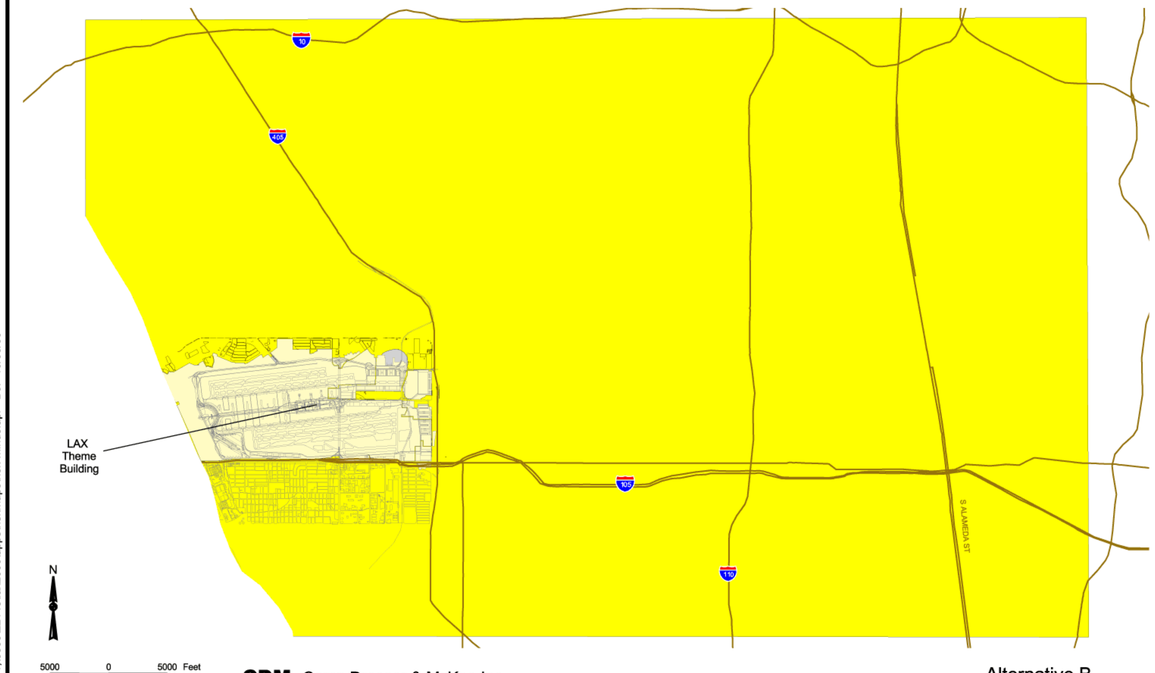
Figure 14



No Action/No Project Alternative



Alternative A



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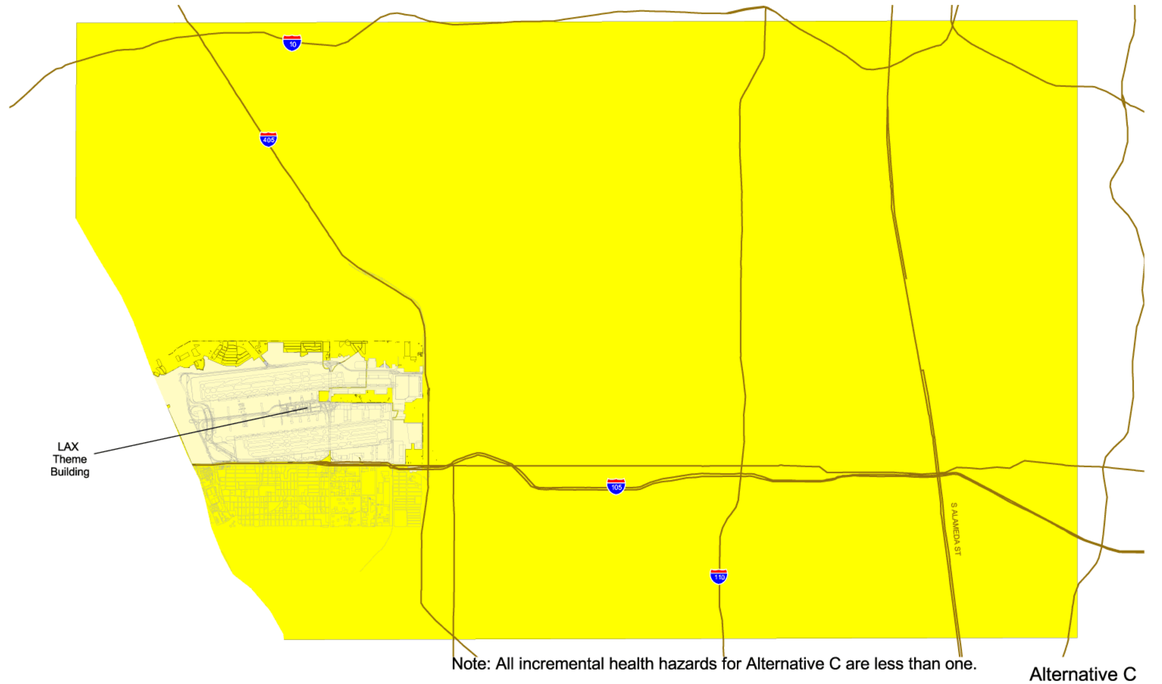
Alternative B



Note: All incremental health hazards for Alternative C are less than one.

Alternative C

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Los Angeles International Airport Master Plan

Geographic Extent of Incremental Non-Cancer Health Hazards for Horizon Year 2015 Post-Mitigation Conditions

Figure 15

Risk estimates from the MATES-II were compared to incremental risk estimates for the No Action/No Project Alternative and the three build alternatives for 2015. 2015 is the year projected for completion of the LAX Master Plan and is thus the first year when full operations are expected after implementation. Since the MATES-II study does not report estimates for non-cancer effects, cumulative impacts on human health are only evaluated for exposure to carcinogenic TAPs.

Estimated incremental risks associated with Alternatives A, B, and C for post-mitigation conditions for Horizon Year 2015 are compared to "background" risks in the South Coast Air Basin in **Figure 16**, Cancer Risks, Inclusive of Environmental Baseline, for the Build Alternatives and the No Action/No Project Alternative, East-Northeast Projection, 2015 Post-Mitigation. Since minimal mitigation is possible under the No Action/No Project Alternative, risks for this alternative under pre-mitigation conditions are presented in the figure for comparison.

Cumulative risks associated with LAX operations were plotted from east of the LAX fence line to the east, east-northeast, north, and south, and are included in Attachment E, *Risks Associated with the Build Alternatives Compared to Background*. The east-north east direction was selected for further evaluation because it indicates the risks in the direction of the prevailing winds. Average background cancer risks identified from the MATES-II study (1,400 in one million) were plotted for comparison. These values were assumed to represent the possible range of background risks at various distances generally east of LAX.

All risk estimates presented are based on adult residential exposure assumptions. Since adult residential exposures produced the highest cancer risk estimates, the comparisons shown represent the greatest cumulative risk estimates for emissions from LAX.

The comparison of average background cancer risks from MATES II with estimated cumulative cancer risks is shown in **Figure 17,** Cancer Risks, Inclusive of Environmental Baseline, for the Build Alternatives and the No Action/No Project Alternative, East-Northeast Projection, 2015 Pre-Mitigation. Estimated risks for the No Action/No Project Alternative near the LAX fence line suggest an increase in cumulative risks of approximately two percent over average background risks. Cumulative risks associated with the No Action/No Project Alternative rapidly decrease with distance from LAX and are only slightly above background at a distance of 40,000 to 50,000 feet from the LAX fence line. Cumulative cancer risks associated with Alternatives B and C suggest an increased cumulative risk of approximately one percent above background near the LAX fence line, but approach background risks at a distance of approximately 15,000 feet from the fenceline. **Figure 17,** Cancer Risks, Inclusive of Environmental Baseline, for the Build Alternatives and the No Action/No Project Alternative, East-Northeast Projection, 2015 Pre-Mitigation, indicates that cumulative risks associated with Alternatives B and C are reduced below estimated background risks at approximately 15,000 feet from the LAX fence line. Cumulative cancer risks associated with Alternative A were estimated to be approximately one percent below background risks in the South Coast Air Basin and to approach background risks at approximately 15,000 to 20,000 feet from the LAX fence line.

These findings suggest that under pre-mitigation conditions, Alternatives B and C could result in an increase of approximately one percent to cumulative cancer risks in the South Coast Air Basin at the LAX fence line. However, measurement of a change of 1 percent is generally impossible with current air sampling and analysis techniques. Thus, impacts on air quality and, hence, cancer risk would not be measurable for the build alternatives. The No Action/No Project Alternative may be associated with an increase in cumulative cancer risks of up to 2 percent for people living immediately adjacent to the east end of the north runways. Cancer risk estimates associated with the build alternatives and the No Action/No Project Alternative decrease rapidly with distance east-northeast from the LAX fence line.

Potential impacts of LAX operations on cumulative cancer risks to communities north and south of the airport would be even less, since these communities are not located in the direction of prevailing winds. Under post-mitigation conditions (**Figure 16**, Cancer Risks, Inclusive of Environmental Baseline, for the Build Alternatives and the No Action/No Project Alternative, East-Northeast Projection, 2015 Post-Mitigation) cumulative cancer risks associated with the No Action/No Project Alternative would increase to approximately three percent above background risks. This expected increase is due to increased vehicle congestion in the absence of implementation of the build alternatives. Estimated cumulative cancer risks associated with Alternatives A, B, and C for residents near the LAX fence line are approximately two percent less than background risks.

Overall, the above analyses indicate that:

- LAX operations would have a small impact on cumulative human cancer risks associated with living in the South Coast Air Basin.

- Cumulative risks associated with LAX are higher for the No Action/No Project Alternative than for any of the build alternatives, both in terms of magnitude and geographic extent.
- Mitigation reduces cancer risks below those predicted for pre-mitigation conditions. That is, mitigation would result in a decrease in cumulative risks for many people living closest to the airport.

7. UNCERTAINTIES

7.1 Uncertainties Associated with Emissions Estimates and Dispersion Modeling

Risk estimates were based on chemical concentration estimates obtained through emissions and dispersion modeling. Emissions estimates are sensitive to the values used to represent the numerous emission source variables (e.g., future aircraft operation assumptions) and to the air toxic emission factor values used for each source. Consequently, estimated emissions values are subject to uncertainties. Different assumptions and values of variables would result in different emissions estimates. The HHRA used well-accepted methods and best available emission factor data to develop estimates of emissions, and estimates and assumptions are reasonable and appropriate. Actual emissions are unlikely to be substantially greater than those used in the analyses.

The dispersion model used in this analysis represents current state of the art in modeling methodology using a well-developed and accepted air dispersion model (ISC3). Results provided offer the best estimates available to predict future ambient concentrations, within the accuracy of the input data. Some uncertainties are, however, associated with dispersion modeling. The model results are sensitive to the emission source parameters and meteorological data inputs. Different assumptions, models and values or variables would result in different concentration estimates. An attempt was made to ensure that modeled concentrations would not underpredict those possible. For example, meteorological data used was taken from a dataset thought to provide generally conservative (higher than average) annual average concentrations.

Additionally, studies of ISC3 model accuracy have consistently confirmed the following conclusions:

- Dispersion models are more reliable for predicting long-term concentrations than for estimating short-term concentrations at specific locations.
- Dispersion models are reasonably reliable in predicting the magnitude of the highest concentrations occurring, without respect to a specific time or location.

USEPA's Guideline on Air Quality Models (Revised) at 40 CFR 51, Appendix W provides additional discussion of modeling uncertainties and sensitivities.[40]

7.2 Evaluation of Sensitive Receptor Populations

Certain subpopulations may be more sensitive or susceptible to negative health impacts caused by environmental contaminants than the population at large. These critical subpopulations were also considered in the exposure assessment and two subpopulations (young children living near LAX and school children near LAX) were selected for quantitative evaluation. Residents are expected to spend more time in affected areas than any other populations, and were therefore included in quantitative evaluations. Day care or nursery school children near LAX represent the same age group as young children living near LAX and are therefore adequately represented by the child resident. School children were selected, because they represent a different age group and may be present at locations with different estimates for TAP concentrations. Evaluation of other populations would either be redundant or quantitative methods for evaluation of these populations are not available (e.g., nursery home residents and people in hospitals).

Risk estimates presented in the HHRA represent a wide range of potential exposures including the highest that can be reasonably expected. Thus, even though risk estimates are not provided for all potentially sensitive receptors in the area, populations not specifically evaluated are still expected to be represented. For example, quantitatively evaluated populations include those with the highest expected exposure

[40] USEPA, Air Quality Modeling Guideline. Code of Federal Regulations, Title 40, Part 51, Appendix W, August 12, 1996

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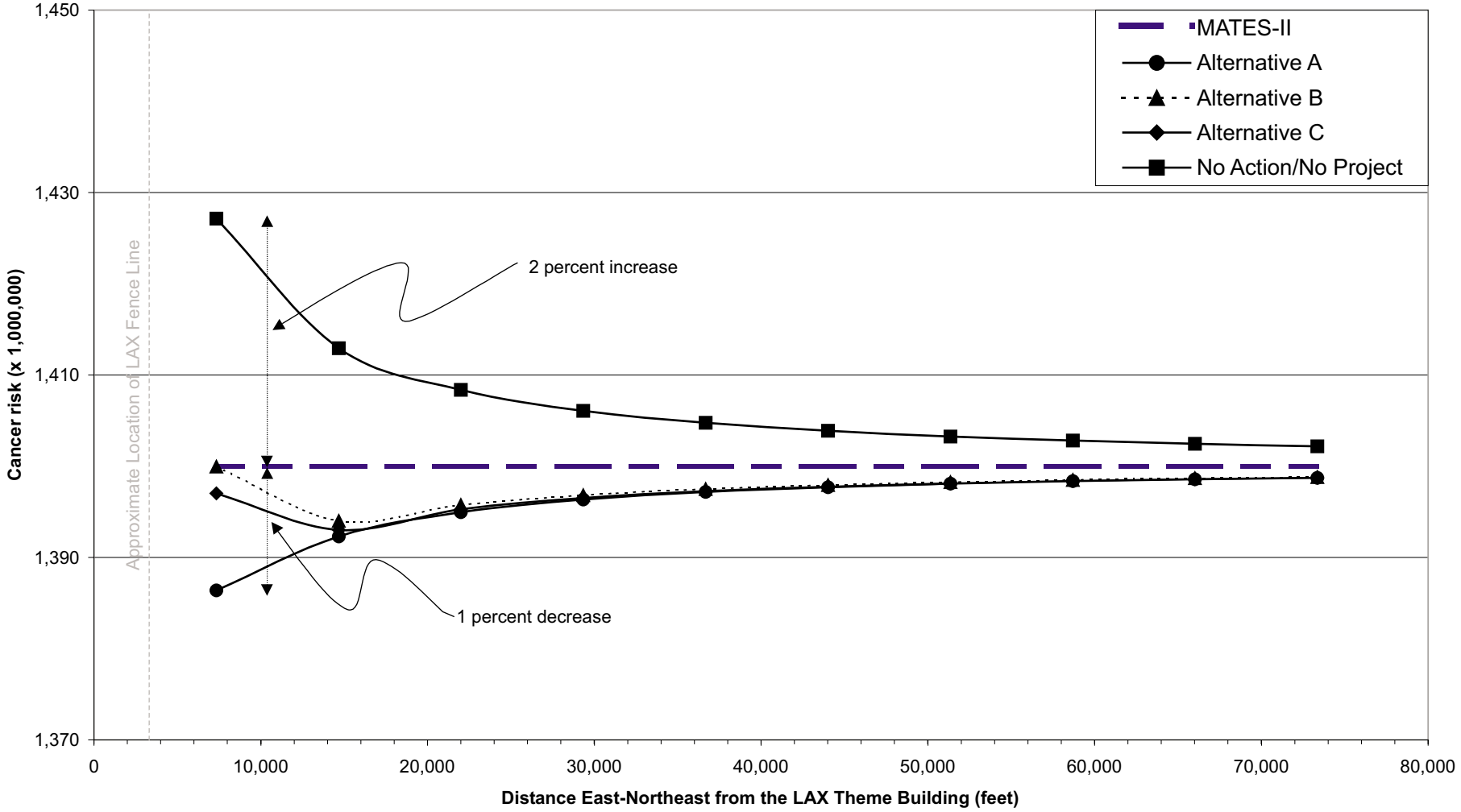

Los Angeles International Airport Master Plan	Cancer Risks, Inclusive of Environmental Baseline, for the Build Alternatives and the No Action/No Project Alternative East-Northeast Projection, 2015 Post-Mitigation	Figure 16

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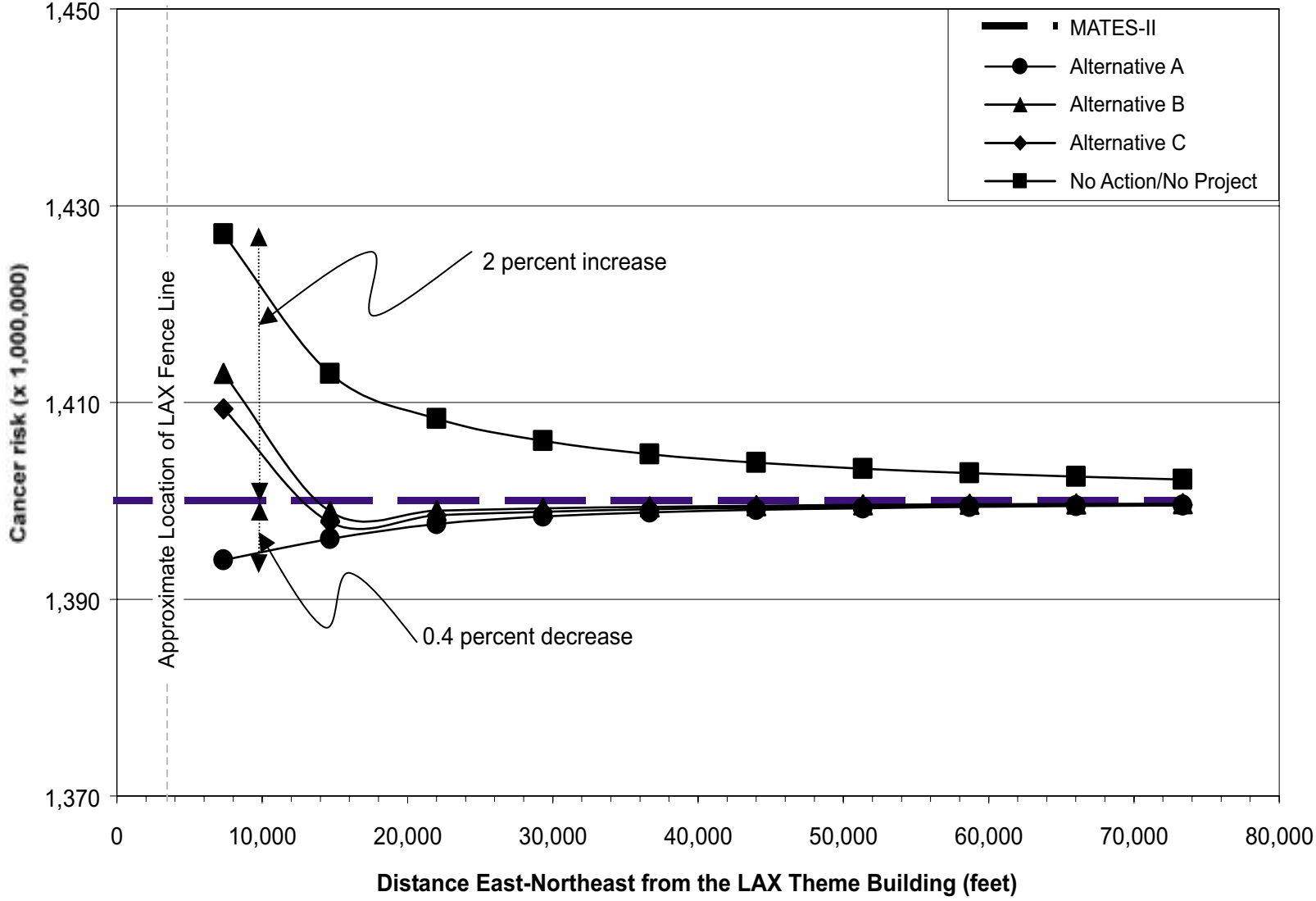

Los Angeles International Airport Master Plan	Cancer Risks, Inclusive of Environmental Baseline, for the Build Alternatives and the No Action/No Project Alternative, East-Northeast Projection, 2015 Pre-Mitigation	Figure 17

durations and exposure frequencies (e.g., residents). Exposures are therefore expected to be less for other populations, even those with higher chemical sensitivities.

7.3 Uncertainties Associated with Toxicity Assessment

A potentially large source of uncertainty is inherent in the derivation of the CalEPA and USEPA toxicity criteria (i.e., oral and inhalation RfDs, and cancer slope factors). In many cases, data used to develop RfDs must be extrapolated from animals to sensitive humans by the application of uncertainty factors to estimated no-observable-adverse-effects-levels (NOAELs) or lowest-observed-adverse-effects-levels (LOAELs). While designed to be protective, in many cases these uncertainty factors overestimate the magnitude of differences that may exist between human and animals, and among humans.

In some cases, however, toxicity criteria may be based on studies that did not detect the most sensitive adverse effects. For example, many past studies have not measured possible toxic effects on the immune system. Moreover, some chemicals may cause subtle effects not easily recognized in animal studies.

Derivation of cancer slope factors often involves linear extrapolation of effects at high doses to potential effects a lower doses commonly seen in environmental exposure settings. Currently, it is not known whether linear extrapolation is appropriate. Probably, the shape of the dose-response curve for carcinogenesis varies with different chemicals and mechanisms of action. Description of any such differences in quantitative terms is problematic at this time because of difficulties in observing effects at very low exposure levels. In addressing uncertainties, USEPA recognizes that risks calculated at very low levels of exposure could be overestimated by the linear extrapolation process and may even be zero.

Uncertainties in the CalEPA slope factor for diesel particulates is notable because diesel exposure accounts for a large fraction of total cancer risks estimated. A recent examination of animal studies for diesel particulates concludes that "loading" of the lungs with high amounts of particles is necessary for the carcinogenic action of diesel exhaust. At levels of exposure at which the lung is not overloaded with particles, no observable increase in cancer occurs. If this mechanism can be extrapolated to people, cancer risks in the HHRA may be dramatically overestimated.

CalEPA has recognized the possible importance of the above mechanism (and other suggested mechanisms) in diesel carcinogenesis, but has determined that available data are not sufficiently clear to establish this or any other mechanism as the underlying factor in diesel-induced cancer. This determination by CalEPA is subject to challenge by reasonable scientists studying the same database. Uncertainties in the diesel slope factor as applied in this HHRA are large and interpretation of the results of the assessment should consider these uncertainties.

Finally, RELs proposed by CalEPA are still under review and re-evaluation. Use of these criteria for the screening described in Section 3, *Summary of Selection of TAPs of Concern*, is reasonable because:

- No quantitative risk estimates were derived.
- The design of the toxicity screening would allow RELs to include additional TAPs, but would not allow exclusion of TAPs.

Uncertainties in proposed RELs are, however, too great to allow use in quantification of risks in the final HHRA. Instead, the impact of use of RELs as proposed on the findings and conclusions of the HHRA is discussed below as part of the uncertainties analysis. Some of the above factors are discussed in more detail below.

7.3.1 Lack of Quantitative Evaluation for Particulates in Jet Exhaust

Diesel exhaust is expected to be emitted in large quantities from LAX under the three build alternatives and the No Action/No Project Alternative and is therefore quantitatively evaluated in the HHRA. Only diesel exhaust from ground sources (e.g., trucks and buses) is included in these evaluations. Aircraft emissions were not included because there is insufficient information regarding the nature and toxicity of total petroleum hydrocarbon (TPH) emissions associated with aircraft and toxicity criteria for these emissions are not available. Toxicity criteria are available for diesel exhaust in general, however, extrapolation of these criteria to TPH emitted from aircraft was not considered scientifically justifiable.

Aircraft use a lighter fuel and a substantially different combustion process than do diesel engines and have therefore very different emissions.

Toxicological research indicates that the component of diesel exhaust responsible for most toxicological effects is PM[41] and USEPA's RfC for diesel exhaust is based entirely upon PM. Diesel engines produce particulate matter in large amounts. Burning of jet fuel in engines using modern turbine technology creates much less particulate matter than is created during diesel fuel combustion. Due to different fuels and combustion processes in jet engines and diesel engines, particulate emissions from both types of engines are also expected to differ chemically, physically, and therefore, toxicologically. These issues are discussed in more detail in Section 3.6, *Evaluation of Diesel Exhaust as a TAP of Concern*.

Due to expected toxicological differences, extrapolation of PM emissions from diesel exhaust to jet exhaust is not considered appropriate or scientifically justifiable. This was recognized in a January 2000 CARB Advisory Committee draft report on commercial airport activities.[42] This report stated that it may not be appropriate to use the CalEPA Unit Risk Factor for diesel PM in (3.0×10^{-4} ug/m^3) when assessing toxic impacts associated with particulate emissions from aircraft.

The lack of quantitative evaluation of TPH emissions from aircraft results in some uncertainty in the risk estimates presented in the HHRA. However, because ground sources are expected to emit large quantities of PM, and aircraft engines are expected to emit relatively little, risks associated with TPH emissions from LAX are still expected to be adequately characterized.

7.3.2 Uncertainties for 1,3-Butadiene and Acrolein

Two volatile TAPs of concern dominate non-diesel associated risk estimates for LAX emissions. 1,3-butadiene and acrolein are "risk drivers" for cancer and non-cancer effects, not subject to the large uncertainties in the CalEPA diesel toxicity assessment. Recently, a new health assessment for 1,3-butadiene has been published by USEPA, and Toxicological Excellence in Risk Assessment (TERA) recently published a toxicological review of acrolein.[43] Both these recent reviews were evaluated, and new toxicological insights incorporated into toxicological profiles (Attachment C, *Toxicological Profiles*). No new information was uncovered that would indicate that toxicological criteria for either TAP would be likely to change substantially in the near future. Thus, cancer risks and non-cancer hazards for both TAPs are consistent with the most current toxicological literature.

7.3.3 Uncertainties Associated with Proposed California RELs

RELs are non-cancer toxicity criteria that are being considered but have not yet been adopted by the CalEPA. CalEPA RELs were considered in the selection of TAPs of concern but not in the characterization of health effects associated with the No Action/No Project Alternative or implementation of the three build alternatives. This section discusses potential impacts from adoption of the RELs for each TAP of concern for which the proposed REL is substantially different from the USEPA criteria used in the HHRA.

1,3-Butadiene

Currently, no USEPA inhalation RfC or oral RfD is available. Non-cancer health effects for 1,3-butadiene were therefore not evaluated. The proposed CalEPA REL for 1,3-butadiene is 2.3×10^{-3} mg/kg-day. If the REL is adopted, total estimated non-cancer health effects would increase slightly. This increase would, however, not contribute substantially to overall non-cancer health effects estimates. The proposed REL and the estimated emission rate for 1,3-butadiene are similar to those for acetaldehyde. Acetaldehyde contributes only a fraction of a percentile to non-cancer risks estimated in the HHRA. A substantial contribution from adoption of the REL for 1,3-butadiene is, therefore, not expected.

Benzene

No RfD or RfC for benzene has been adopted by the USEPA. Non-cancer health effects for benzene were therefore not estimated in the HHRA. A REL of 1.7×10^{-2} mg/kg-day has been proposed by CalEPA. If benzene was quantitatively evaluated using the proposed REL, there could theoretically be some

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41 USEPA National Center for the Environmental Assessment, Online Integrated Risk Information Database (IRIS), 1999.

42 CARB, Advisory Committee Draft Report on Commercial Airport Activities, January 2000.

43 USEPA National Center for the Environmental Assessment, Integrated Risk Information System (IRIS) Online Database, 1999.

contribution to overall non-cancer health effects. Because the REL for benzene is relatively low compared to the RfCs or RfDs for other chemicals, especially acrolein, and emissions are not dominated by benzene, this contribution is expected to be low.

Formaldehyde

A REL of 5.7×10^{-4} mg/kg-day is proposed for formaldehyde. This value is almost three orders of magnitude lower (more stringent) than the USEPA oral RfD of 2×10^{-1} mg/kg-day used in the HHRA. Because currently formaldehyde contributes very little to overall non-cancer health effects estimates and these estimates are strongly dominated by acrolein, substantial contribution to overall non-cancer effects is also not expected if the proposed REL for formaldehyde is adopted. For example, the total HI for a child resident at the location with the highest estimated TAP concentration is 13 for Alternative A with mitigation in 2015. The contribution from formaldehyde is only 3×10^{-3}. Adoption of the REL would thus not result in a substantial increase of the total HI.

Xylene

The proposed REL for xylene (5.7×10^{-2} mg/kg-day) is approximately 1.5 orders of magnitude lower than the oral RfD used to evaluate xylene in the HHRA (2 mg/kg-day). The oral RfD was used, because an inhalation RfC for xylene is not currently available. Because xylene contributes relatively little to overall non-cancer health effects associated with emissions from LAX, adoption of the REL is not expected to substantially impact the results of the HHRA. For example, even for sensitive populations at the locations with the highest estimated TAP concentrations, estimated non-cancer effects from xylene are only approximately 100 in one million or less. Thus, even if the proposed REL was adopted, estimated non-cancer effects (HQs) from xylene would still be well below one.

Chromium VI

The proposed REL for chromium VI (2.3×10^{-7} mg/kg-day) is approximately two orders of magnitude lower than the USEPA Inhalation RfD for chromium VI (2.86×10^{-5} mg/kg-day). Chromium VI was not estimated to contribute substantially to overall risks associated with LAX. Estimated HQs for residents and school children at locations with the highest estimated TAP concentrations were at least four orders of magnitude below one. This suggests that adoption of the REL for chromium VI would not have a substantial impact on the results of the HHRA.

7.4 Uncertainties in Background Risk Estimates (MATES-II)

It is important to note that the risks from MATES-II were calculated based on monitoring data collected from April 1998 through March 1999. Modeling during the MATES-II study was used only to fully characterize basin risks – not to project what future concentrations and risks would be or to evaluate possible cumulative impacts from other proposed projects near the airport. As such, comparisons between 2015 projected risks with the MATES-II results must be interpreted in recognition of the different time periods being represented. One may surmise that basin-wide cancer risks would likely increase in time with the inevitable increase in mobile sources along with population growth. MATES-II might underpredict background concentrations of TAPs in the South Coast Air Basin for year 2015. If background is underpredicted, cumulative impacts of the build alternatives may be overestimated.

However, according to CARB data, carcinogenic risks due to many TAPs have decreased 44 to 63 percent since 1990, primarily due to decreased air concentrations of benzene, 1,3-butadiene, and hexavalent chromium, but also in part to decreases air concentrations in carbon tetrachloride, methylene chloride, perchloroethene, trichloroethene, lead, and nickel. If continuing progress is made toward reductions in TAP emissions in the South Coast Air Basin, MATES-II could overpredict potential background risks for horizon year 2015. If this is true, however, the traffic component of the air dispersion modeling for LAX emissions is likely to be too large also. Progress toward decreasing TAP emissions in the South Coast Air Basin must focus on mobile sources, which are the major contributors. Reductions in mobile source emissions would affect emissions from both airport and non-airport related traffic. Overall, the effect of general reductions in mobile source emissions could increase the relative contribution of LAX to basin-wide risks, but any such increase may be tempered by effects of general reductions on LAX-related traffic.

Unfortunately, trends are not available for diesel particulates because these compounds were not previously monitored. Since diesel particulates have been found to contribute up to 70 percent of the

carcinogenic risks in the South Coast Air Basin, whether estimated risks (such as those calculated in the MATES-II) would increase or decrease by 2015 is difficult to project. Again, and importantly, any general decrease in diesel emissions would also reduce diesel emissions in LAX-related traffic. Since diesel emissions were also a major contributor to LAX-related cancer risks, changing background as a result of better control of diesel emissions may not greatly affect fractional LAX contribution to basin-wide cancer risks.

7.5 Uncertainties in Mitigation Impacts

As discussed in Section 4.6, *Air Quality*, of the Draft EIS/EIR, an extensive list of potential mitigation options were considered in response to comments received during the environmental-scoping process or listed in CARB guidance.[44] These mitigation options must be approved by LAWA or another implementing agency and are therefore considered preliminary at this time. Some of these measures may not be implementable for a variety of technical reasons or may be preempted by the federal government. Additional mitigation options will be developed in response to public and agency comments on the Draft EIS/EIR. The final Mitigation Measures will be developed jointly with LAWA, FAA, USEPA, CARB, SCAQMD, and other implementing agencies.

The preliminary mitigation options currently under consideration are expected to result in substantial reductions in releases of TAPs during LAX operations for the build alternatives. These reductions may be either under- or overestimated by assumptions in the effectiveness of Mitigation Measures. In particular, one-engine taxi will be encouraged to reduce emissions of TAPs from jet engines during taxi and queue. These aircraft modes cause the bulk of impacts of TAP emissions on air quality. Since aircraft represent the largest source of TAPs other than diesel particulates, beneficial impacts of the potential mitigation options may be particularly sensitive to assumptions made for the percentage of time that aircraft would use a single engine for taxi and queue. The assumption for this percentage of time is still being considered. The possible range of assumptions for the effectiveness of Mitigation Measures could cause estimated incremental cancer risks due to exposure to TAPs other than diesel particulates to increase by a factor of two or less. Since TAPs other than diesel particulates contribute perhaps 30 percent to total cancer risks, incremental cancer risks after mitigation could be underestimated by about 60 percent. Such a change in incremental risk estimates would not change the conclusions of this HHRA.

For non-cancer health risks, about 80 percent of the hazard index for respiratory irritants, which reflects essentially all (~ 99 percent) non-cancer health hazard, is due to acrolein in jet engine exhaust. If release estimates for this TAP are underestimated by a factor of 2, hazard indices after mitigation will be underestimated by about 160 percent. Such a change would not alter the conclusions of the HHRA for Alternatives A and C. However, increasing the MEI incremental hazard index, post mitigation, for Alternative B in 2015 by a factor of 1.6 would cause the incremental HI for respiratory effects to exceed the threshold of significance.

[44] California Air Resources Board, Research Division, Air Pollution Mitigation Measures for Airports and Associated Activity-Final Report, 1994.

Attachment A
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Attachment B
Screening Level Human Health Risk Assessment

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1. INTRODUCTION

1.1 Overview

Los Angeles World Airports (LAWA) has proposed expansion of the existing Los Angeles International Airport (LAX) in response to increasing demand for air travel and freight services in the region. Three separate airport development alternatives are currently under consideration as part of the LAX Master Plan.

Activity levels are anticipated to increase at LAX, regardless of whether or not the Master Plan is implemented. However, with the improvements, a higher level of activity will be accommodated in the long term. As a consequence of a greater level of activity with the Master Plan projects, the potential exists that higher toxic air pollutant levels will result. California statutes and regulations, including the Air Toxics "Hot Spots" Information and Assessment Act of 1987, California Health & Safety Code Section 44300 et seq. (AB2588), South Coast Air Quality Management District (SCAQMD) Rules 1401, 1402, and 212, and the Environmental Impact Report (EIR) requirement of the California Environmental Quality Act (CEQA) require an evaluation of the potential impacts of significant new developments on surrounding communities. SCAQMD has specifically required that a quantitative assessment of potential human health impacts of toxic air pollutants (TAPs) be performed for the proposed LAX Master Plan, and that this human health risk assessment (HHRA) follows California Environmental Protection Agency (CalEPA) and U.S. Environmental Protection Agency (USEPA) guidance for assessing human health risks from exposure to air toxics.

This report is a preliminary screening analysis for potential impacts of future LAX emissions on the area surrounding the airport, and is developed in partial fulfillment of SCAQMD requirements. The objective of screening is to focus the final HHRA on those TAPs, receptors, exposure pathways, and impact areas of potential concern for the EIR process.

1.2 Scope of the Screening Level Human Health Risk Assessment

HHRAs can be performed at differing levels of detail, ranging from simple "screening" level assessments to complex formal evaluations. Often, screening level assessments precede more complex analyses as a means of identifying important issues and allowing more efficient use of resources. For example, a screening level assessment may determine that groundwater will not be affected at a site, thus allowing analyses for the final HHRA to focus on other media (e.g., air). This document is a screening level assessment designed to help focus the final HHRA on TAPs likely to cause the greatest impacts. The screening process involves the following tasks:

- Identification of emissions sources for TAPs at LAX and quantification of TAP emissions (Section 2, *Selection of Preliminary TAPs of Concern*)
- Selection of TAPs of concern for the HHRA (Section 2, *Selection of Preliminary TAPs of Concern*)
- Screening level air dispersion modeling for initial determination of areas of impact (Section 3, *Screening Level Air Dispersion Modeling for Toxic Air Pollutants*)
- Analysis of exposure pathways of concern for TAPs emitted during LAX operations (Section 4, *Preliminary Exposure Assessment*)
- Determination of areas of potential impact through screening level air dispersion modeling (Section 4, *Preliminary Exposure Assessment*)
- Characterization of potential for human health impacts for individual TAPs of concern (Section 5, *Toxicity Assessment*)

Each of these tasks is described briefly below and in more detail in indicated sections of the report. References for this report are provided in Attachment B5, *Bibliography*.

1.3 Approach to Screening Level Assessment for LAX Master Plan

In a screening assessment, the evaluation of potential TAPs is dependent on several key factors. First, TAPs that pose the largest potential threats to human receptors must be accurately identified. Second, pathways likely to result in the most toxicologically relevant exposures must be identified. Third, areas near LAX where emissions could result in the greatest potential health impacts must be determined. These factors are addressed in several LAX-specific analyses as described below.

1.3.1 Sources of Toxic Air Pollutants

Large numbers of potential TAP emission sources are associated with current LAX conditions and with each alternative. Such sources include increases in aircraft operations and air traffic, ground service vehicles, stationary sources (including central utility plants, parking facilities, and smaller power generating sources), and automotive traffic. Each of these potential sources is associated with emissions of a variety of chemicals, many of which appear on one or more lists of TAPs identified by California or federal agencies as "of concern." Because of the number of potential TAP emission sources and the toxic nature of some TAPs, the LAX Master Plan falls under the jurisdiction of the three California statutes and regulatory programs described earlier, all of which list TAPs of concern.

1.3.2 Selection of TAPs of Concern for Use in the HHRA

TAPs of concern are selected via a multi-step screening process designed to eliminate TAPs less likely to be responsible for significant impacts. The list of TAPs to be considered in assessment of the LAX Master Plan is first narrowed by comparing TAPs on regulatory lists with lists of TAPs known to be released during LAX operations (determined through emissions inventories, literature searches, and projections for the future) and removing TAPs not included in the regulatory lists from further consideration. Next, TAPs that are likely to cause the greatest impacts based on estimated concentrations in emissions and toxicity are identified by estimating an impact factor for each chemical, based on the chemical's estimated annual emissions and known toxicity. For carcinogenic TAPs, impact factors for each TAP are determined by multiplying annual emissions by an established cancer slope factor (state or federal, inhalation or oral exposure). For non-carcinogenic TAPs, impact factors are determined by dividing emissions for each TAP by established reference doses (state or federal, inhalation or oral exposure). In all cases, California toxicity criteria take precedence over federal criteria, and inhalation criteria take precedence over oral criteria. The percentage each chemical contributes to overall potential impact is then calculated by dividing the estimated impact factor for individual chemicals by the sum of all impact factors. The analysis is conducted separately for carcinogens and non-carcinogens. TAPs that are likely to cause the greatest impacts are selected for further assessment if they contribute at least 0.1 percent to total impacts based on recommendations made in USEPA's Risk Assessment Guidance for Superfund.[1]

1.3.3 Analysis of Exposure Pathways and Identification of Exposure Areas

Critical steps in the assessment of potential human health risks from TAPs released during airport operations are the characterization of exposure pathways and identification of locations for exposed receptors (people living, working, and or recreating near LAX). This screening level HHRA used preliminary air dispersion modeling to characterize exposure, to define an initial study area, and to identify locations of human receptors for the final HHRA.

1.3.3.1 Exposure Pathways

Exposures to TAPs released from LAX might obviously include direct inhalation of TAPs from air. However, some TAPs could deposit onto soils and other exposure pathways could also be important. After deposition, for example, children might ingest chemicals in soil through hand-to-mouth activity during outdoor play, or residents who have gardens could ingest chemicals taken up from soil into plants. For the HHRA, inhalation of TAPs is considered a potentially important exposure pathway, and exposures and risks from inhalation of TAPs is quantified. Therefore, no screening of this pathway is required.

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[1] USEPA, Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual Interim Final, December 1989.

Other potential exposure pathways are analyzed in a two-step screening process. In the first step, screening level air dispersion modeling was used to determine potential TAP concentrations in air on or near LAX, and these concentrations were used to estimate deposition of TAPs onto soils over time. In the second screening step, concentrations of TAPs estimated in soil were compared to the range of background concentrations of these chemicals to determine the relative impacts of deposition from air. This analysis indicated that impacts to soils from deposition of TAPs from airport operations would be negligible. Therefore, secondary pathways involving TAPs in soil were not further evaluated.

1.3.3.2 Areas of Impact

Identification of areas of potential impact from LAX operations also used the results of the screening level air dispersion modeling in a two-step process. This process was based on the assumption that releases of TAPs from LAX would only be important where they contributed a substantial fraction to the total amount of TAPs found in the South Coast Air Basin. For example, if the amount of benzene in air in the Basin at some distance from LAX was 0.2 micrograms per cubic meter ($\mu g/m^3$), and the expected contribution from LAX operations was 0.002 $\mu g/m^3$ at some location near LAX, one would determine that LAX impacts were negligible compared to the South Coast Air Basin. Likewise, if benzene contributions at another location near LAX were 0.1 $\mu g/m^3$, one would conclude that LAX impacts could be important.

The two-step process for determining the study area for the HHRA therefore required first estimating "background" concentrations of TAPs in the South Coast Air Basin at locations distant from LAX, then using the screening level air dispersion modeling to determine where LAX impacts might be important compared to this background. Since background concentrations were not available for all TAPs of concern, representative TAPs, benzene and toluene, were used for the analysis. Both of these chemicals are found in exhaust from gasoline engines and, therefore, are common air pollutants in the South Coast Air Basin.

Initially, air dispersion modeling results were used to generate isopleths (lines connecting locations of equal TAP concentrations in air) over a map of LAX and surrounding communities. Isopleths were generated for concentrations equal to half the estimated background concentration to provide a preliminary indication of the extent LAX impacts. An isopleth representing one-half the background concentration would encircle an area where LAX contributions could be expected to be near to or greater than background.

These initial isopleths for benzene and toluene suggested that LAX impacts at or near the background for the South Coast Air Basin would be confined to small areas on-airport. Since concentrations of TAPs would decrease greatly with distance from LAX, off-site impacts at or near the estimated background might not occur. However, only two TAPs of concern for LAX were addressed in the screening analysis, and South Coast Air Basin background concentrations were taken from recent measurements at the SCAQMD monitoring station in downtown Los Angeles. The latter location may not be representative of background for communities closer to the airport. Thus, even though Los Angeles impacts could be small, a study area was defined, using professional judgment, to include a relatively large area around LAX for further investigation.

1.3.4 Toxicity Characterization for TAPs of Concern

Human health risks are determined by both the amount of exposure and the inherent toxicity of TAPs. Since toxicity is a property of a chemical, site-specific toxicity evaluation is not necessary. In fact, both USEPA and CalEPA have derived toxicity factors that express the toxicity of different chemicals in quantitative terms. These factors are used in the final HHRA to develop risk estimates.

Toxicity factors are derived separately for carcinogens and non-carcinogens. For carcinogenic chemicals, cancer slope factors (CSFs) are used as measures of the potential for chemicals to cause cancer. For non-carcinogens, reference doses (RfDs) are developed as "safe" thresholds for exposure. A RfD is intended as a daily exposure that will not result in adverse effects even in sensitive individuals.

Since toxicity factors are established for common TAPs of concern by regulatory agencies, the toxicity assessment for the HHRA consists of (1) identifying which regulatory toxicity factors are most appropriate, and (2) examining the most recent toxicity information to ensure that established factors reflect the most current understanding of toxicity. Toxicity factors were selected for use in the HHRA based on the following hierarchy:

- CalEPA toxicity factors based on inhalation exposure
- USEPA toxicity factors based on inhalation exposure

- CalEPA toxicity factors based on oral exposure
- USEPA toxicity factors based on oral exposure

Toxicity factors developed by CalEPA are given precedence over those developed by USEPA. Further, only inhalation was identified as an important exposure pathway for TAPs released from LAX. Thus, preference is given to toxicity factors developed from data on inhalation. However, if no toxicity factors based on inhalation are available, toxicity factors based on oral exposure are used.

Toxicity factors identified for use in the HHRA were evaluated through review of most recent toxicity information to ensure that important new information was incorporated into the HHRA. The evaluation of toxicity for TAPs of concern is included in a series of toxicity profiles.

1.4 Structure of Screening Level Assessment

The LAX toxic air pollutant screening evaluation is divided into the following sections:

- Identification of Emission Factors and Selection of Toxic Air Pollutants of Concern (Section 2, *Selection of Preliminary TAPs of Concern*)
- A description of the TAP air dispersion modeling approach used to predict TAP concentrations at different locations in the area potentially affected by LAX TAP releases (Section 3, *Screening Level Air Dispersion Modeling for Toxic Air Pollutants*)
- Exposure Assessment (Section 4, *Preliminary Exposure Assessment*)
- Toxicity Assessment (Section 5, *Toxicity Assessment*)
- Summary and Conclusions for the Final HHRA (Section 6, *Summary and Conclusions*)

2. SELECTION OF PRELIMINARY TAPS OF CONCERN

The first portion of the HHRA identifies preliminary TAPs of concern to be included in the final HHRA. Chemicals were selected based on identification as TAPs in federal and state regulations, current or future presence in emissions at LAX, magnitude of possible emissions, and toxicity. TAPs of concern for LAX are those chemicals expected to be released in sufficient amounts to contribute substantially to overall impacts from airport operations. TAP listings in regulations were used to help guide this identification process, and particular attention was paid to federal and state concerns for toxic substances released to the atmosphere.

The Federal Aviation Administration (FAA), in a letter of December 10, 1997,[2] directed the use of methods employed at other airports to address issues related to TAPs as a template for this document. These methods incorporate the body of literature on TAPs associated with airports and aircraft. Further, the inventory of LAX emissions presented in the Technical Report 1*, Land Use* was incorporated into this HHRA. This emissions inventory is used to generate a baseline for extrapolation to horizon year 2015.

TAPs of concern were selected for the No Action/No Project Alternative and one build alternative (Alternative B Year 2015). TAP selection for both scenarios is based on emission estimates for the year 2015, the time when all construction associated with the LAX Master Plan is expected to be completed. Thus, construction emissions were not considered in the selection of TAPs of concern.

TAPs for inclusion in the HHRA were selected in the following five steps:

- Potential sources of TAPs from an inventory of airport operations were identified.
- TAPs associated with LAX operations were identified.
- TAPs listed in state and federal guidance were compared with LAX-related TAPs.
- Emissions were estimated for individual TAPs.
- Toxicity screening was used to identify TAPs of concern.

[2] Federal Aviation Administration and the United States Air Force, Air Quality Procedures for Civilian Airports & Air Force Bases, FAA Office of Environment and Energy (AEE-120), 1997.

The approach is illustrated graphically in **Figure B1**, Process for Identifying TAPs of Concern, and is described in more detail in the following sections.

TAPs of concern for LAX include chemicals expected to be present in emissions from LAX, identified as TAPs in state and/or federal guidance, and estimated to contribute at least 0.1 percent to overall impacts associated with LAX operations.

2.1 Identification of Potential Sources of TAPs (Step 1)

The first step in the selection of TAPs of concern involved identification and characterization of emission sources at the airport. Sources were identified during extensive surveys of airport facilities, automotive traffic, air traffic, and typical airport operations. An inventory of potential sources of air pollutants at LAX was conducted in 1997 using the following information:

- Recent surveys of stationary sources at LAX (see Technical Report 1, *Land Use*)
- SCAQMD databases of stationary sources.
- Previous LAX surveys.

As expected, important sources include exhaust from aircraft and ground vehicles, as well as a variety of other sources related to maintenance operations, airport utilities, and fuel tank farms. The inventory was sufficiently detailed to include individual sources such as individual internal combustion engines for emergency power. Attempts were made to identify and annotate all potential TAP sources associated with the airport. Where all individual sources could not be determined (e.g., automobile traffic), assumptions were made such that contributions were more likely to be over- than underestimated. For each source, the inventory compiled data on factors such as size of the source, frequency of use, and fuel consumption. The complete inventory is provided as an attachment to a separate Technical Memorandum titled LAX Master Plan Emissions Inventory Submittal.[3]

2.2 Identification of TAPs Associated with LAX Operations (Step 2)

After emissions sources were identified, the second step in the TAP selection process was characterization of chemicals released from each source. Chemicals that may be released to air during LAX operations include a variety of inorganic chemicals, and volatile and semivolatile organic compounds. As a result, emissions may include chemicals expected to remain in vapor phase (e.g., benzene) and chemicals released as particulates (e.g., polycyclic aromatic hydrocarbons [PAH] and metals). For a facility as large as LAX, quantification of every constituent released from every potential emission source during every use or operation is nearly impossible. However, many tools are available that provide information regarding the types and quantities of chemicals released, emission rates, etc. for a number of types of equipment and operations (e.g., surface coating). These information sources, some of which are listed below, were consulted along with a conservative tally of potential emissions sources (e.g., number/type of aircraft entering/exiting the airport per day, number of generators, and fuel tanks) to produce reasonable estimates of emissions during airport operations.

- California Air Toxics Emission Factors (CATEF) Database[4]
- Factor Information Retrieval (FIRE) System Database[5]
- Volatile Organic Compounds/Particulate Matter (VOC/PM) Speciation Data System (SPECIATE) Database[6]

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3 CDM, Criteria and Toxic Air Pollutant Emission Inventories, Preliminary Draft, March 1998.

4 California Air Resources Board, California Air Toxics Emission Factors Database User's Manual, Version 1.2, October 1993.

5 USEPA Office of Air Quality Planning and Standards, Factor Information Retrieval (FIRE) System: User's Manual, September 1993.

6 USEPA Office of Air Quality Planning and Standards, Volatile Organic Compound (VOC)/Particulate Matter (PM) Speciation Data System (SPECIATE) User's Manual, Version 1.5, February 1993.

- Crosswalk/Air Toxic Emission Factor (XATEF) Database[7]
- USEPA Memorandum, Re: Source Identification and Base Year 1990 Emission[8]
- Inventory Guidance for Mobile Source HAPs on the USEPA Office of Air Quality Planning Services (OAQPS) List of 40 Priority HAPs[9]
- Motor Vehicle-Related Air Toxics Study[10]
- FAA's Aircraft Engine Emissions Database (FAEED) Version 2.1[11]
- FAA/U.S. Air Force (USAF) Emissions and Dispersion Modeling System (EDMS) Version 3.02[12]
- Compilation of Air Pollutant Emission Factors (AP-42)[13]
- EMFAC7 Mobile Emissions Model, Version 7F and 7G[14]
- TANKS Tank Emissions Estimation Model, Version 3.0[15]
- Air Pollution Mitigation Measures for Airports and Associated Activity[16]
- Air Quality Procedures for Civilian Airports and Air Force Bases[17]
- CEQA Air Quality Handbook[18]

TAPs that may be released from emissions sources at LAX are listed in **Table 1**, TAPs Potentially Released from LAX Emission Sources.

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7 USEPA Office of Air Quality Planning and Standards, Crosswalk/Air Toxic Emission Factor (XATEF) Database Management System User's Manual, Version 2.0, EPA-450/B-92-011, October 1992.

8 USEPA Office of Mobile Sources, Memorandum from Rich Cook to Anne Pope, Re: Source Identification and Base Year 1990 Emission Inventory Guidance for Mobile Source HAPs on the OAQPS List of 40 Priority HAPs, June 11, 1997.

9 USEPA, Source Identification and Base Year 1990 Emission Inventory Guidance for Mobile Source HAPs on the OAQPS List of 40 Priority HAPs, 1997.

10 USEPA Office of Mobile Sources, Emission Planning and Strategies Division, Motor Vehicle-Related Air Toxics Study, Report Number EPA 420-R-93-005, 1993.

11 Federal Aviation Administration Office of Environment and Energy (AEE-120) and the United States Air Force Armstrong Laboratory Tyndall Air Force Base, Emissions and Dispersion Modeling System (EDMS) Reference Manual, FAA-AEE-97-01, 1997.

12 Federal Aviation Administration and the United States Air Force Armstrong Laboratory, Tyndall Air Force Base and FAA Office of Environment and Energy (AEE-120), Air Quality Procedures for Civilian Airports & Air Force Bases, 1997.

13 USEPA Office of Air Quality Planning and Standards, Compilation of Air Pollution Emission Factors. Volume I: Stationary Point and Area Sources (AP-42, 5th Edition and Supplements), 1997.

14 California Air Resources Board and California Department of Transportation, Methodology for Estimating Emissions from On-Road Motor Vehicles B Volume II: EMFAC7G, November 1996.

15 USEPA Office of Air Quality Planning and Standards, User's Guide to Tanks. Storage Tank Emissions Calculation Software, Version 3.1, 1997.

16 California Air Resources Board Research Division, Air Pollution Mitigation Measures for Airports and Associated Activity, CARB A132-168, 1994.

17 Federal Aviation Administration and the United States Air Force Armstrong Laboratory, Tyndall Air Force Base and FAA Office of Environment and Energy (AEE-120), Air Quality Procedures for Civilian Airports & Air Force Bases, 1997.

18 SCAQMD, CEQA Air Quality Handbook, 1993.

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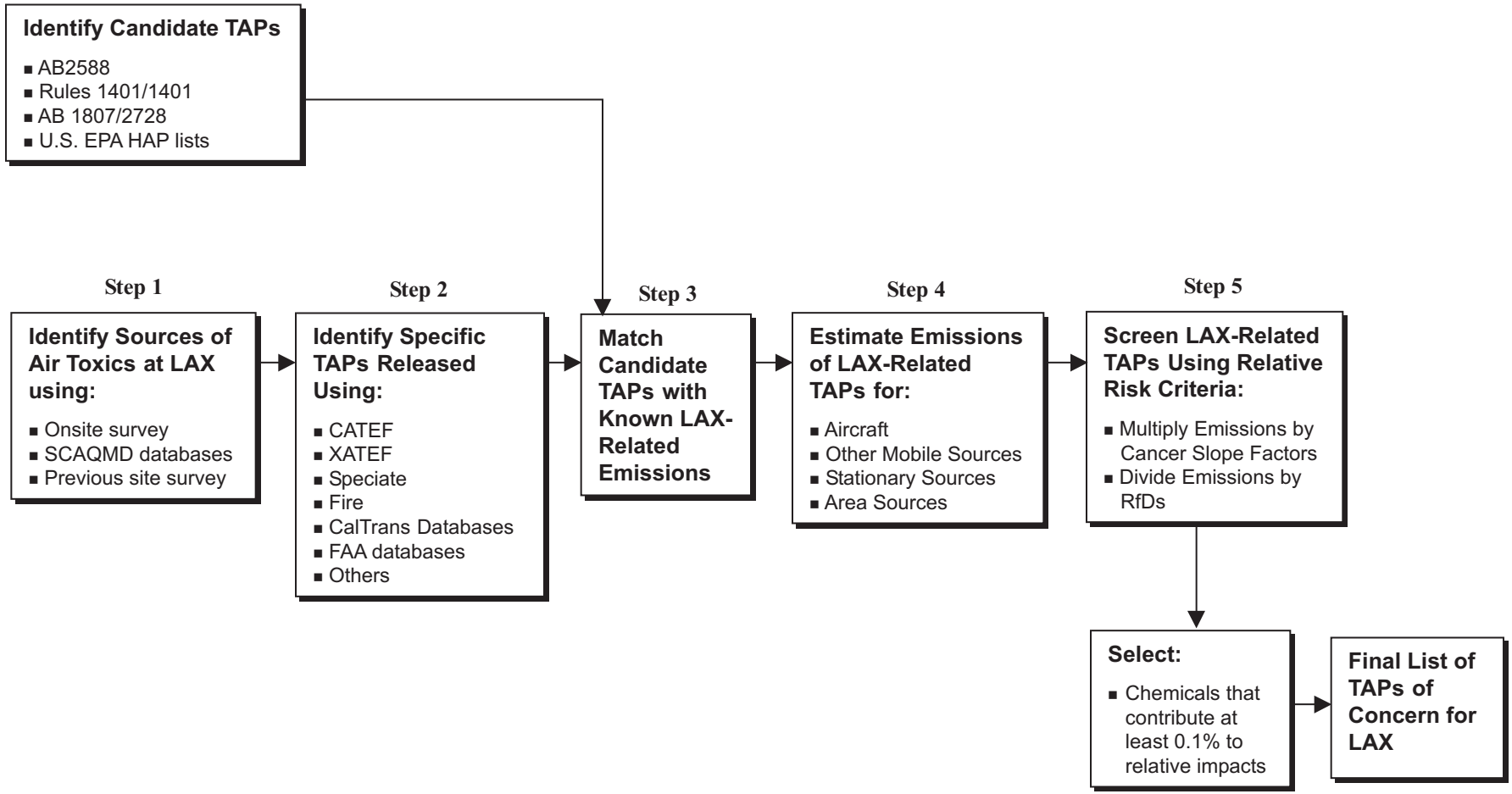

Prepared by: Camp Dresser & McKee
Draft Date: 6/19/00
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Los Angeles International Airport Master Plan	Process for Identifying TAPs of Concern	Figure B1

Table 1

TAPs Potentially Released from LAX Emission Sources

Acenaphthene	Furan (heptachloro total)
Acenaphthylene	Furan (hexachloro total)
Acetaldehyde	Furan (octachloro)
Acrolein	Furan (pentachloro total)
Ammonia	Furan (tetrachloro total)
Anthracene	Hexane
Antimony	Hydrogen chloride
Arsenic	Hydrogen fluoride
Benzaldehyde	Indeno(1,2,3-cd)pyrene
Benzene	Lead
Benzo(a)anthracene	Manganese
Benzo(a)pyrene	Mercury
Benzo(b)fluoranthene	Methanol
Benzo(g,h,i)perylene	Methyl ethyl ketone
Benzo(k)fluoranthene	Methylene Chloride
Beryllium	Naphthalene
Bromine	N-Butanol
Butadiene, 1-3	Nickel
Cadmium	Perchloroethylene
Chromium (Total)	Phenanthrene
Chromium (VI)	Phosphorus
Chrysene	Propylene
Cobalt	Propylene oxide
Copper	Pyrene
Dibenz(a,h)anthracene	Secondary Butanol
Dioxin (heptachloro total)	Selenium
Dioxin (hexachloro total)	Styrene
Dioxin (octachloro)	TCDD, 2,3,7,8 Equivalents
Dioxin (pentachloro total)	Toluene
Dioxin (tetrachloro total)	Trichloroethane, 1,1,1
Ethyl Alcohol	Trichloroethylene
Ethylbenzene	Xylene (total)
Ethylene Glycol Ethers	Xylene, m- or p-
Fluoranthene	Xylene, o-
Fluorene	Zinc
Formaldehyde	

Source: Source: Camp Dresser & McKee Inc., 1998.

2.3 Comparison of TAPs listed in State and Federal Guidance with LAX-Related TAPs (Step 3)

In the third step, TAPs that may be released at LAX were compared to TAPs listed in state and federal regulations. The purpose of this screening step was to identify those TAPs that may be present at LAX and are also considered potential health threats for air releases by regulatory agencies. Emission estimates and subsequent evaluations were focused on such chemicals.

Three state lists and one federal list of TAPs were consulted to identify TAPs of concern for LAX. State listings included: SCAQMD Rules 1401 and 1402, AB2588 and AB1807/2728. These regulatory lists contain most of the common toxic chemicals that are found in air emissions from industrial and other sources. Lists of chemicals to be considered under AB2588, AB1807/2728, and SCAQMD Rules 1401/1402 are included in Attachment B1, *List of Chemicals to be Considered*.

The Clean Air Act (CAA) is also used as guidance for the identification of TAPs. One hundred and eighty-eight pollutants listed in Section 112 of the CAA are considered Hazardous Air Pollutants (HAPs). HAPs are also listed in Attachment B1, *List of Chemicals to be Considered*. Under Section 112(k) of the CAA as amended in 1990, USEPA was required to identify at least 30 HAPs that present the greatest threat to public in the largest number of urban areas. Forty HAPs were identified, based on toxicity, ambient

monitoring, and emissions inventory data. These 40 compounds are known as 112(k) HAPs. A list of toxic air compounds derived from the 112(k) HAPs that apply specifically to aircraft and airport operations is found in USEPA's guidance for Clean Air Act Section 112(k) Emissions Inventory Aircraft.[19] These compounds are listed separately in Attachment B1, *List of Chemicals to be Considered*.

Table 1, TAPs Potentially Released from LAX Emission Sources, lists chemicals that may be released to air during LAX operations. Almost all of these chemicals are also found on state and/or federal lists of TAPs. Because so few chemicals not found on lists of TAPs were identified for LAX, a decision was made to include all chemicals that might be released during airport operations in further analysis. Thus, the list of chemicals in **Table 1**, TAPs Potentially Released from LAX Emissions Sources, was not shortened by elimination of chemicals not found on regulatory lists.

2.4 Estimation of Emissions for Individual TAPs (Step 4)

In Step 4 of the screening process, emissions from sources at LAX were estimated for individual TAPs. Future emissions were estimated using existing data sources and growth projections in the LAX Master Plan Project Description. Emissions from all sources were calculated using emissions factors in data bases obtained from FAA, USEPA, CalEPA, and Caltrans.

Emission estimates for TAPs at LAX were generated in two phases. In the first phase (Phase I), completed in March 1998, emissions were estimated using data collected during a previous survey. In the second phase (Phase II), completed in November 1998, emission estimates for the No Action/No Project Alternative were revised based on inspections at LAX and interviews with LAX tenants identified by LAWA. In essence, Phase II is a more refined version of Phase I estimates. Use of both emissions estimate methodologies may provide a range of possible impacts instead of necessitating reliance upon a single set of values.

In Phase I, emissions were estimated for the No Action/No Project Alternative using the following approach:

- SCAQMD permitted and non-permitted emission sources were identified and inventoried.
- Emission factors and operational data were identified from databases and peer-reviewed literature.
- Chemical species in exhausts or other forms of air emission were determined for each source.
- Emissions factors and operational parameters were used to estimate annual emissions.

The resulting annual emissions factors were then used in the toxicity screening process (Section 2.5, *Toxicity Screening*) to identify a preliminary list of TAPs of concern. This screening identified a short list of chemicals from **Table 1**, TAPs Potentially Released from LAX Emissions Sources that might be important in assessing impacts of airport operations. The analysis demonstrated that emissions of TAPs from the airport are dominated by TAPs in jet exhaust.

After the Phase I screening, additional information on emissions for some chemicals was developed, and projections for future emissions, based on revised proposed airport operations, became available. To ensure that these changes would not invalidate the original toxicity screening analysis, a second phase of screening was performed.

In Phase II, new estimates for releases of some TAPs in jet exhaust, mainly metals, were developed after further review of available information. In addition, projections for future emissions from LAX, based on Alternative B, were generated from the descriptions of airport operations expected for this alternative. Revised emission estimates affected mainly TAPs released in jet exhaust. Since these TAPs are the most important in terms of potential impact, changes in emission estimates could alter the result of toxicity screening. Thus, Phase I toxicity screening was repeated using Phase II emission estimates to ensure that the list of TAPs of concern remained appropriate.

[19] USEPA, Compilation of Air Pollution Emission Factors. Volume I: Stationary Point and Area Sources (AP-42, 5th Edition and Supplements), September 1997.

2.4.1 Phase I Emission Estimates

During Phase I, emissions were estimated for potential stationary, area and mobile sources at LAX. Phase I emission estimates for the No Action/No Project Alternative are provided in **Table 2**, Emissions Summary, Phase I/II**.**

Table 2

Emissions Summary Phase I/II

	Phase I[1]			Phase II[1]					
	No Action/No Project Emissions Estimation			Revised No Action/No Project 2015 Emissions Estimates			Alternative B 2015 Emissions Estimates		
Source Category	Aircraft Totals (kg/yr)	Non-aircraft Totals (kg/yr)	Total Operating (kg/year)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr) [2]	Aircraft + Non-aircraft Emissions (kg/yr)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr) [2]	Aircraft + Non-aircraft Emissions (kg/yr)
Acenaphthene	0.00	1.92	1.92	7.04	1.92	8.96	9.51	1.92	11.42
Acenaphthylene	0.01	8.26	8.26	10	8.26	19	14	8	22
Acetaldehyde	53,956	1,574	55,530	14,873	1,574	16,447	18,022	1,574	19,596
Acrolein	26,323	235	26,559	6,909	235	7,144	8,399	235	8,634
Anthracene	0.51	0.96	1.47	62	0.96	63	84	0.96	85
Arsenic	1,208	1	1,210	10	1.14	11	13	1.14	14
Benzaldehyde	0.00	5.94	5.95	2,360	5.94	2,366	2,845	5.94	2,851
Benzene	22,840	20,412	43,251	10,348	20,412	30,760	12,547	20,412	32,959
Benzo(a)anthracene	0.30	1.08	1.38	5.32	1.08	6.39	7.17	1.08	8.24
Benzo(a)pyrene	0.18	0.48	0.66	1.24	0.48	1.71	1.67	0.48	2.15
Benzo(b)fluoranthene	0.03	0.59	0.62	4.12	0.59	4.71	5.56	0.59	6.15
Benzo(g,h,i)perylene	0.04	0.89	0.94	2.19	0.89	3.08	2.96	0.89	3.85
Benzo(k)fluoranthene	0.03	0.41	0.44	4.12	0.41	4.53	5.56	0.41	5.97
Beryllium	0.00	0.00	0.00	2.56	0.00	2.56	3.48	0.00	3.48
Butadiene, 1-3	20,876	2,567	23,443	8,926	2,567	11,494	10,857	2,567	13,424
Cadmium	114	8.03	122	15	8.03	23	21	8.03	29
Chromium VI (all sources)	0.00	0.00	0.00	0.51	0.00	0.51	0.69	0.00	0.69
Chromium (total)		0.00		20	52	72	27	52	79
Chrysene	0.19	0.87	1.06	11	0.87	12	15	0.87	15
Copper	0.06	46	46	47	46	93	64	46	110
Dibenz(a,h)anthracene	0.00	0.24	0.24	3.14	0.24	3.39	4.24	0.24	4.49
Ethylbenzene	0.06	69	69	1,995	69	2,064	2,425	69	2,494
Fluoranthene	1.47	1.56	3.03	192	1.56	194	259	1.56	261
Fluorene	0.00	3.03	3.03	0.00	3.03	3.03	0.00	3.03	3.03
Formaldehyde	174,648	3,969	178,617	48,316	3,969	52,285	58,579	3,969	62,548
Hexane	0.22	96	96	6,493	96	6,589	7,906	96	8,002
Indeno(1,2,3-cd)pyrene	0.00	0.24	0.24	2.46	0.24	2.71	3.32	0.24	3.57
Lead	1,253	1,687	2,941	29	1,687	1,716	39	1,687	1,726
Manganese	0.00	120	120	485	120	605	659	120	780
Mercury	0.00	0.62	0.62	0.13	0.62	0.74	0.17	0.62	0.79
Naphthalene	418	608	1,026	11,668	608	12,276	15,703	608	16,311
Nickel	114	29	143	2,298	29	2,328	3,124	29	3,153
Phenanthrene	6.44	11	17	628	11	639	845	11	856
Propylene	50,747	8,119	58,866	29,036	8,119	37,156	35,313	8,119	43,432
Pyrene	1.65	1.44	3.09	160	1.44	162	216	1.44	217
Selenium	114	0.00	114	0.40	0.00	0.40	0.54	0.00	0.55
Styrene	4,574	1,052	5,626	2,975	1,052	4,027	3,616	1,052	4,668
Toluene	5,101	33,883	38,984	14,511	33,883	48,394	17,663	33,883	51,547
Xylene (total)	4,709	17,645	22,354	10,679	17,645	28,324	12,988	17,645	30,633
Xylene, m- or p-	2,857	0.00	2,857	7,791	0.00	7,791	9,474	0.00	9,474
Xylene, o-	1,872	0.00	1,872	2,895	0.00	2,895	3,521	0.00	3,521
Zinc	1,254	649	1,903	2,534	649	3,183	3,444	649	4,093

[1] Figures are as reported; risk estimates based on emissions estimates are rounded to two significant figures
[2] Non-aircraft emissions for Phase II estimates were calculated as part of Phase I.

Source: Camp Dresser & McKee Inc., 1998.

2.4.1.1 Stationary Sources

Several stationary operations were considered possible current and future sources of TAPs at LAX. These included power/HVAC plants, fuel storage tanks, surface coating facilities, solvent degreasing operations, deicing/anti-icing operations, and fire training operations. Each of these potential sources is discussed separately below.

Power/HVAC Plants

TAPs emissions from on-airport power plants and HVAC plants were calculated based on fuel type, consumption rate, and pollutant emission factors. Fuel consumption rates and air pollution control information were based on on-airport surveys completed in 1997, and on future year forecasts of fuel usage and SCAQMD control requirements. Volatile organic chemical (VOC) and particulate matter (PM) emission factors were obtained primarily from the Compilation of Air Pollutant Emission Factors (AP-42), or FIRE database. Air toxic emissions were developed using data in CATEF, FIRE, SPECIATE, and XATEF.

Fuel Storage Tanks

VOC and TAPs emissions from existing and future fuel storage tanks were calculated using USEPA's TANKS (version 3.0) emission estimation program, and data from FIRE, SPECIATE, and XATEF. Storage tank type (floating or fixed roof), fuel type, fuel throughput, and tank-specific characteristics (color, breather vent settings, etc.) were determined from the existing conditions survey (see Technical Report 1, *Land Use*). Data used to represent the relocated off-site fuel farm were provided by the LAX Master Plan Project Description. The off-site fuel farm is part of Alternative B. Climatic data contained in the TANKS database were used to calculate the evaporative emissions. For any new tanks, it was assumed that SCAQMD Rules and Regulations and Best Available Control Technology (BACT) Guidelines would be followed; emission estimates from these new tanks were based on adherence to these rules and guidelines. For example, new aboveground storage tanks in California must be equipped with internal floating roofs if used to store volatile chemicals. Accordingly, emissions from new aboveground storage tanks were assumed to be equal to those of aboveground tanks equipped with internal floating roofs.

Surface Coating Facilities

Volatile hydrocarbons (VOC or HC) are emitted into the atmosphere during surface coating operations through evaporation of the paint vehicle, thinner, or solvent used in the application of coatings. Volatile hydrocarbon emissions can be calculated using methods recommended in the Air Quality Procedures for Civilian Airports and Air Force Bases, supplemented by the requirements of the SCAQMD Rules and Regulations, and BACT Guidelines. Toxic air emissions were determined using CATEF, FIRE, SPECIATE, and XATEF.

Types and quantities of coatings used at on-airport facilities, as well as the effectiveness of air pollution controls associated with coating operations and devices were derived from the 1997 existing conditions survey (see Technical Report 1, *Land Use*). Where possible, VOC contents of coatings were obtained from product-specific MSDS sheets and facility use records. In some cases, default values from Air Quality Procedures for Civilian Airports and Air Force Bases were used if MSDS sheets or facility records were not available. For any new storage or operations, it was assumed that SCAQMD Rules and Regulations and BACT Guidelines were adhered to.

Solvent Degreasers

The use of organic solvents, such as chlorinated hydrocarbons, petroleum distillates, ketones, and alcohols, for degreasing and other maintenance operations results in evaporation and release of volatile chemicals to air. Estimated emissions for these volatiles are based on the assumption that solvent not recaptured and disposed as waste liquid will be released into the atmosphere. Emissions from solvent degreasing were calculated using the methods recommended in Air Quality Procedures for Civilian Airports and Air Force Bases. Air toxic emissions were determined using CATEF, FIRE, SPECIATE, and XATEF. Estimated quantities of solvent were based on typical usages determined during the existing conditions survey. Where water-based or other inorganic degreasers were used, evaporation of VOCs was assumed not to occur.

Deicing/Anti-Icing Operation

Deicing and anti-icing operations include the application of deicing/anti-icing fluids (usually water mixed with propylene glycol or ethylene glycol) to the aircraft, runways and taxiways. Deicing of on-airport roadways is usually performed with a salt or salt/sand mixture. Due to the mild winter climate in Southern California, deicing/anti-icing operations were assumed to be non-existent. Therefore, emissions of VOC and hydrocarbons from these operations, and emissions of PM from wind erosion of sand/salt piles, were not considered.

Training Fires

TAPs from the burning of training fires include PM and VOC emissions. Emissions depend upon the type of fuel burned and the duration of the burn (quantity of fuel burned). Emissions from training fires were calculated using methods recommended in Air Quality Procedures for Civilian Airports and Air Force Bases. Air toxic emissions were determined using CATEF, FIRE, SPECIATE, and XATEF. The quantity of fuel burned was obtained from the aircraft rescue and fire fighting department at LAX. The results of the emissions speciation and the resource used for each source is provided in as an attachment to a separate memorandum titled LAX Master Plan Emissions Inventory Submittal.[20]

2.4.1.2 Area Sources

Area sources are defined as clusters of small stationary sources that cannot be reasonably modeled on the basis of individual sources and are, instead, considered larger single sources of emissions. Two examples of area sources associated with LAX include parking structures and emergency generators. Parking structures can be modeled as a single stationary area source by using data on the numbers and types of vehicles that use the facility on a daily basis. Emergency generators and general power units (GPU), auxiliary power units (APU), and air conditioning (AC) units operate at numerous locations around the terminals as needed for support of aircraft. These sources can also be conveniently modeled as one or more area sources based on their size and frequency of use. Methodologies used in the calculation of emissions from parking structures and emergency generators/GPUs/APUs/AC units are described in the following sections.

Emergency Generators/GPUs/APUs/AC Units

TAPs emissions from Emergency Generators/GPUs/APUs/AC units were calculated using USEPA-approved methodology[21, 22] based on capacity or engine power ratings, usage rate, and pollutant emissions indices (based on power output and fuel type). Reported air pollution control equipment was documented and appropriate emission factors identified to reflect these controls.

Emissions from emergency generators were calculated using methods recommended in Air Quality Procedures for Civilian Airports and Air Force Bases. The capacity of the emergency generators, typical operating hours, and pollution controls were based on data from the existing conditions survey in the Technical Report 1, *Land Use.* Uncontrolled VOC and PM emission factors were obtained from the USEPA Compilation of Air Pollutant Emission Factors. Control efficiencies were applied to those units with control devices/technologies. TAP emissions were estimated using CATEF, FIRE, SPECIATE, and XATEF.

Parking Facilities

Methods similar to those for evaluating on-road emissions for private and commercial traffic (described below) were used to characterize emissions from parking facilities. The California Vehicle Emission Inventory Model (MVEI7G) was used with site-specific data on the numbers and types of vehicles using the parking facilities on a daily basis. However, unlike the analysis for on-road vehicles, resting evaporation of automobile fuels is included in emission estimates.

TAPs emissions were calculated for each parking lot or garage. Emissions from multi-level garages were estimated by calculating each level individually and then summing the emissions from the individual levels. Assumptions were made for idle time on each level, average distance traveled within the parking facility,

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[20] CDM, Criteria and Toxic Air Pollutant Emission Inventories, Preliminary Draft, March 1998.

[21] Federal Aviation Administration & the United States Air Force, Air Quality Procedures for Civilian Airports & Air Force Bases, FAA Office of Environment and Energy, (AEE-120), 1997.

[22] USEPA, Procedures for Emission Inventory Preparation, Volume IV: Mobile Sources, EPA-450/4-81-026d (Revised), 1992b.

and vehicle mix within the facility. Number and type of vehicles were determined by CDM using traffic surveys information by Leigh Fisher Associates[23] and JKH.[24]

2.4.1.3 Mobile Sources

Mobile sources considered in the preparation of this HHRA include:

- Aircraft (commercial and private)
- Ground support equipment (GSE)
- On-road vehicles (on- and off-airport)

Aircraft

In Phase I, aircraft TAP emissions were estimated using methods obtained from USEPA[25] and the International Civil Aviation Organization (ICAO).[26] Emissions produced during five aircraft operational modes were evaluated, including:

- Takeoff
- Taxi
- Queue
- Climb out
- Approach

Wherever possible, information supplied by LAWA was used to provide LAX-specific times in each operational mode. In cases where LAX-specific times were not available, emissions were based on ICAO default times. Further, taxi/in and taxi/out times were combined into one total taxi time. Total taxi time also included aircraft queue time, since emissions during queue and taxi are very similar. Addition of taxi times is appropriate since most taxi and queue events occur in the same areas of the runway; aircraft taxi for very short distances on runways after landing before turning onto taxi ways.

Rotary wing aircraft do not operate under the normal definition of takeoff, and climb out times for these aircraft types include takeoff. Takeoff time for rotary wing aircraft is therefore set to zero.

Because of the relatively long runways at LAX, the use of reverse thrust is generally not necessary and was assumed to have a negligible impact on air quality. Reverse thrust impacts were not included in emissions calculations.

Using aircraft engine emission factors from EDMS, FAEED, AP-42, and other sources, VOC and PM emissions were calculated for each aircraft and engine type. TAP emissions were estimated using VOC and PM emission estimates and combined with speciation data from SPECIATE, FIRE, and XATEF, and USEPA Guidance on Mobile Source HAPs.

Ground Support and Auxiliary Power

VOC and PM emissions from GSE and APU were calculated using procedures obtained from the FAA, USEPA, and ICAO.[27, 28, 29] Assignments of GSE and APUs to aircraft and appropriate usage times were based on owner/operator provided data, wherever such data were available. If unit-specific data were not available, default values from EDMS were used to estimate usage. TAP emission factors were obtained from CATEF, FIRE, SPECIATE, XATEF, and USEPA Guidance on Mobile Source HAPs.[30] Emissions

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23 Leigh Fisher Associates, On-Airport Existing Transportation Conditions Memorandum, Draft Final, January 1996.

24 JKH, LAX Master Plan 2005 Concepts 1,2 &3 Landside Results, Memorandum, 1997.

25 USEPA, Procedures for Emission Inventory Preparation, Volume IV: Mobile Sources, EPA-450/4-81-026d (Revised), 1992.

26 ICAO, AICAO Engine Exhaust Emissions Data Bank, First Edition B, 1995.

27 Federal Aviation Administration & the United States Air Force, Air Quality Procedures for Civilian Airports & Air Force Bases, FAA Office of Environment and Energy (AEE-120), 1997.

28 ICAO, AICAO Engine Exhaust Emissions Data Bank, First Edition B, 1995.

29 USEPA, Procedures for Emission Inventory Preparation, Volume IV: Mobile Sources, EPA-450/4-81-026d (Revised), 1992.

30 USEPA, Compilation of Air Pollution Emission Factors. Volume I: Stationary Point and Area Sources (AP-42, 5th Edition and supplements), 1997.

were based on equipment fuel type and brake horsepower. Electric powered GSE was assumed to produce no toxic air pollutant emissions.

On-Road Vehicles

VOC and PM emissions from on-road vehicles and ground access vehicles were calculated using California mandated methodology.[31] Ground access vehicles include privately owned vehicles, government-owned vehicles, rental cars, shuttles, buses, taxicabs, and trucks. Due to varying emissions characteristics, CalEPA divides these vehicles into 10 categories:

- LDA - Light duty autos (non-catalyst, catalyst, and diesel), typical passenger car, but does not include vans or sport utility vehicles
- LDT - Light duty trucks (non-catalyst, catalyst, and diesel) with a gross vehicle weight (GVW) of 6,000 pounds or less
- MDT - Medium duty trucks (non-catalyst and catalyst) with a GVW between 6,001 and 8,500 pounds
- LHDT - Light-heavy diesel trucks with a GVW between 8,501 and 14,000 pounds
- LHGT - Light-heavy duty gasoline trucks (catalyst and non-catalyst) with a GVW between 8,501 and 14,000 pounds
- MHGT - Medium-heavy gasoline trucks (non-catalyst and catalyst) with a GVW between 14,001 and 33,000 pounds
- MHDT - Medium-heavy diesel trucks with a GVW between 14,001 and 33,000 pounds
- HHDT - Heavy diesel trucks with a GVW of greater than 33,000
- UBD - Urban transit buses (diesel) and intra-city transit buses, does not include inter-city transit buses (e.g., Greyhound) or school buses
- MCY - Motorcycles (non-catalyst)

VOC and PM emissions from on-road vehicles were calculated using the California vehicle emission inventory model,[32] MVEI7G, and site-specific conditions. Vehicle trip distances and average travel speeds were based on specific roadway segments. Resting emissions were not included. California mandated default values were used where appropriate. TAP emissions were determined using factors from CATEF, FIRE, SPECIATE, USEPA Guidance on Mobile Source HAPs, and the Motor Vehicle-Related Air Toxics Study.[33]

2.4.2 Phase II Emission Estimates

During Phase II, revised and expanded emission estimates were generated for two alternatives, No Action/No Master Plan Alternative, and Alternative B, for horizon year 2015. The purpose of Phase II was to determine if any additional TAPs should be included in the analysis based on more accurate emission estimates and on estimates for one of the Master Plan alternatives.

Based on the emission estimates derived from the LAX inventory, aircraft emissions account for about 97 percent of total overall emissions and also contribute the most to individual TAP emissions. For example, aircraft emissions comprise more than 99 percent of total acrolein emissions at LAX (**Table 3**, Aircraft Contribution to Total LAX Emissions, Phase I/II Emissions). As discussed in Section 2.5, *Toxicity Screening (Step 5),* emissions of acrolein may be associated with the greatest potential non-cancer health effects at LAX. With the exception of benzene, for which approximately 52.7 percent of total estimated emissions are not from aircraft, aircraft emissions account for the bulk of total emissions for all chemicals selected as TAPs of concern. Since aircraft emissions dominate potential impacts from LAX by a wide margin, it was reasonable to focus Phase II screening analyses on these emissions.

Implementation of the LAX Master Plan would result in increased number of aircraft and flights at LAX, and, therefore, increased aircraft emissions. Aircraft emissions would, therefore, also be expected to

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31 California Air Resources Board and California Department of Transportation, Methodology for Estimating Emissions from On-Road Motor Vehicles B Volume II: EMFAC7G, November 1996.

32 California Air Resources Board and California Department of Transportation, Methodology for Estimating Emissions from On-Road Motor Vehicles B Volume II: EMFAC7G, November 1996.

33 USEPA, Motor Vehicle-Related Air Toxics Study, April 1993.

Table 3

Aircraft Contribution to Total LAX Emissions Phase I/II Emissions

	Phase I[1]				Phase II[1]							
	No Action/No Project Alternative Emissions Estimation				Revised No Action/ No Project Alternative 2015 Emissions Estimates				Alternative 2015 Emissions estimates			
Source Category	Aircraft Totals (kg/yr)	Non-aircraft Totals (kg/yr)	Total Operating (kg/year)	Percent from Aircraft (%)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr) [2]	Total Operating (kg/yr)	Percent from Aircraft (%)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr) [2]	Total Operating (kg/yr)	Percent from Aircraft (%)
Acenaphthene	0.00	1.92	1.92	0.00%	7.04	1.92	8.96	78.59%	9.51	1.92	11	83.21%
Acenaphthylene	0.00	8.26	8.26	0.00%	10	8.26	19	55.83%	14	8.26	22	63.04%
Acetaldehyde	53,956	1,574	55,530	97.17%	14,873	1,574	16,447	90.43%	18,022	1,574	19,596	91.97%
Acrolein	26,323	235	26,559	99.11%	6,909	235	7,144	96.70%	8,399	235	8,634	97.27%
Anthracene	0.51	0.96	1.47	34.79%	62	0.96	63	98.48%	83.57	0.96	84.53	98.86%
Arsenic	1,208	1.14	1,210	99.91%	9.51	1.14	11	89.31%	13	1.14	14	91.91%
Benzaldehyde	0.00	5.94	5.95	0.05%	2,360	5.94	2,366	99.75%	2,845	5.94	2,851	99.79%
Benzene	22,840	20,412	43,251	52.81%	10,348	20,412	30,760	33.64%	12,547	20,412	32,959	38.07%
Benzo(a)anthracene	0.30	1.08	1.38	21.93%	5.32	1.08	6.39	83.18%	7.17	1.08	8.24	86.96%
Benzo(a)pyrene	0.18	0.48	0.66	27.41%	1.24	0.48	1.71	72.19%	1.67	0.48	2.15	77.82%
Benzo(b)fluoranthene	0.03	0.59	0.62	4.72%	4.12	0.59	4.71	87.39%	5.56	0.59	6.15	90.34%
Benzo(g,h,i)perylene	0.04	0.89	0.94	4.46%	2.19	0.89	3.08	71.01%	2.96	0.89	3.85	76.79%
Benzo(k)fluoranthene	0.03	0.41	0.44	6.54%	4.12	0.41	4.53	90.89%	5.56	0.41	5.97	93.09%
Beryllium	0.00	0.00	0.00	0.00%	2.56	0.00	2.56	99.83%	3.48	0.00	3.48	99.87%
Butadiene, 1-3	20,876	2,567	23,443	89.05%	8,926	2,567	11,494	77.66%	10,857	2,567	13,424	80.87%
Cadmium	114	8.03	122	93.42%	15	8.03	23	65.60%	21	8.03	29	72.16%
Chromium Hexavalent (all sources)	0.00	0.00	0.00	0.00%	0.51	0.00	0.51	99.83%	0.69	0.00	0.69	99.87%
Chromium (total)	1,057	52	1,109	95.34%	20	52	72	27.86%	27	51.71	79	34.42%
Chrysene	0.19	0.87	1.06	17.65%	11	0.87	12	92.52%	15	0.87	15	94.34%
Copper	0.06	46	46	0.14%	47	46	93	50.40%	63.89	46	110	58.00%
Dibenz(a,h)anthracene	0.00	0.24	0.24	0.07%	3.14	0.24	3.39	92.80%	4.24	0.24	4.49	94.56%
Ethylbenzene	0.06	69	69.00	0.09%	1,995	68.95	2,064	96.66%	2,425	69	2,494	97.24%
Fluoranthene	1.47	1.56	3.03	48.56%	192	1.56	194	99.20%	259	1.56	261	99.40%
Fluorene	0.00	3.03	3.03	0.00%	0.00	3.03	3.03	0.00%	0.00	3.03	3.03	0.00%
Formaldehyde	174,648	3,969	178,617	97.78%	48,316	3,969	52,285	92.41%	58,579	3,969	62,548	93.65%
Hexane	0.22	96	96	0.23%	6,493	96.16	6,589	98.54%	7,906	96	8,002	98.80%
Indeno(1,2,3-cd)pyrene	0.00	0.24	0.24	0.05%	2.46	0.24	2.71	91.02%	3.32	0.24	3.57	93.19%
Lead	1,253	1,687	2,941	42.62%	29	1,687	1,716	1.67%	39	1,687	1,726	2.25%
Manganese	0.00	120	120	0.00%	485	120	605	80.14%	659	120	780	84.58%
Mercury	0.00	0.62	0.62	0.21%	0.13	0.62	0.74	17.14%	0.17	0.62	0.79	21.97%
Naphthalene	418	608	1,026	40.74%	11,668	608	12,276	95.05%	15,703	608	16,311	96.27%
Nickel	114	29	143	79.68%	2,298	29	2,328	98.75%	3,124	29	3,153	99.08%
Phenanthrene	6.44	11	17	36.97%	628	11	639	98.28%	845	11	856	98.72%
Propylene	50,747	8,119	58,866	86.21%	29,036	8,119	37,156	78.15%	35,313	8,119	43,432	81.31%
Pyrene	1.65	1.44	3.09	53.51%	160	1.44	162	99.11%	216	1.44	217	99.34%
Selenium	114	0.00	114	100.00%	0.40	0.00	0.40	99.83%	0.54	0.00	0.55	99.88%
Styrene	4,574	1,052	5,626	81.30%	2,975	1,052	4,027	73.87%	3,616	1,052	4,668	77.46%

Table 3

Aircraft Contribution to Total LAX Emissions Phase I/II Emissions

	Phase I[1]				Phase II[1]							
	No Action/No Project Alternative Emissions Estimation				Revised No Action/ No Project Alternative 2015 Emissions Estimates				Alternative 2015 Emissions estimates			
Source Category	Aircraft Totals (kg/yr)	Non-aircraft Totals (kg/yr)	Total Operating (kg/year)	Percent from Aircraft (%)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr) [2]	Total Operating (kg/yr)	Percent from Aircraft (%)	Aircraft Totals (kg/yr)	Non-aircraft Emissions (kg/yr) [2]	Total Operating (kg/yr)	Percent from Aircraft (%)
Toluene	5,101	33,883	38,984	13.08%	14,511	33,883	48,394	29.98%	17,663	33,883	51,547	34.27%
Xylene (total)	4,709	17,645	22,354	21.07%	10,679	17,645	28,324	37.70%	12,988	17,645	30,633	42.40%
Xylene, m- or p-	2,857	0.00	2,857	100.00%	7,791	0.00	7,791	100.00%	9,474	0.00	9,474	100.00%
Xylene, o-	1,872	0.00	1,872	100.00%	2,895	0.00	2,895	100.00%	3,521	0.00	3,521	100.00%
Zinc	1,254	649	1,903	65.91%	2,534	649	3,183	79.62%	3,444	649	4,093	84.15%

[1] Figures are as reported; risk estimates based on emissions estimates are rounded to two significant figures
[2] Non-aircraft emissions for Phase II estimates were calculated as part of Phase I

Source: Camp Dresser & McKee Inc., 1998.

dominate in the future. Because the number and type of flights and aircraft are unlikely to differ greatly among alternatives, the use of aircraft emission estimates from the Alternative B Year 2015 scenario are considered to be representative of emissions likely to occur under Alternatives A and C for the purpose of selection of TAPs of concern.

Phase I and II emission estimates for aircraft and estimates for total airport emissions are presented in **Table 3**, Aircraft Contribution to Total LAX Emissions, Phase I/II Emissions. Total airport emissions for the Phase II analysis were estimated by adding Phase I non-aircraft emissions to Phase II aircraft emissions. Comparing the Phase II emission estimates with those from Phase I indicates that, for most TAPs, Phase II estimates are similar to those from Phase I. Phase II estimates predict higher concentrations of PAHs, but somewhat lower concentrations for other chemicals, (e.g., 1,3-butadiene, acetaldehyde, benzene, and acrolein). Results for metals vary. For some metals (e.g., arsenic), higher concentrations were predicted in Phase I. For others (e.g., nickel) higher concentrations were predicted in Phase II.

The revised (Phase II) emission estimates were used to further evaluate TAPs of concern in Step 5.

2.5 Toxicity Screening (Step 5)

In the last step of the TAPs identification process, toxicity screening was conducted to focus the HHRA on those chemicals that might represent a health risk for receptors living in the vicinity of the airport. The toxicity screening methodology used here involves combining established regulations with toxicity criteria with conservatively estimated emissions data to estimate relative overall impacts for TAPs. Both original (Phase I) and revised (Phase II) emission estimates were used in this evaluation, and the results from both screening iterations were compared to provide a range of potential impacts. Total airport emission estimates were used in developing toxicity-based impacts.

Quantitative toxicity screening cannot be conducted for chemicals for which toxicity criteria are not available. Potential impacts from such chemicals are qualitatively evaluated in Section 2.5.1, *Evaluation of Chemicals without Toxicity Criteria.* Quantitative toxicity screening is addressed in Section 2.5.2, *Estimation of Relative Impact for Chemicals with Toxicity Criteria.*

2.5.1 Evaluation of Chemicals without Toxicity Criteria

For several chemicals identified in the LAX emissions inventory, no toxicity criteria are available. These chemicals are, with one exception, not selected for further consideration. Chemicals without toxicity criteria include bromine, n-butanol, s-butanol, ethyl alcohol, hydrogen fluoride, lead, and phosphorus (**Table 4**, TAPs without Toxicity Criteria). Of these chemicals, bromine, n-butanol, s-butanol, ethyl alcohol, and phosphorus are not recognized as toxic at low concentrations, and only small quantities of these chemicals may be released compared to more toxic chemicals. For example, the estimated quantity of ethyl alcohol, that might be emitted (616 kilograms per year [kg/yr]) (**Table 4**, TAPs without Toxicity Criteria) is approximately 3/100th that of 1,3-butadiene (18,733 kg/yr) and 4/1,000th that of formaldehyde (145,648 kg/yr). Based on current emission estimates, the probability that bromine, n-butanol, s-butanol, ethyl alcohol and phosphorus would contribute substantially to site-related exposures and risks is extremely low and risks related to LAX emissions will not be underestimated as a result of eliminating these chemicals from the quantitative assessment. More toxic chemicals for which no toxicity criteria are available include hydrogen fluoride (HF) and lead. HF can be very dangerous, but only at relatively high concentrations. Concentrations that could theoretically be present in ambient air at LAX are orders of magnitude lower than those associated with adverse effects from acute exposures. Long-term exposure to low levels of HF is generally not associated with adverse effects. None of the available toxicological information suggests that long-term exposure to HF at very low concentrations will result in significant human health impacts. In support of this conclusion, only about 18 kilogram (kg) of HF might be released each year. If HF were emitted for 12 hours each day during the year, only about 1 milligram (mg) of HF per second would be released. Such a small quantity would represent negligible concentrations in air.

Table 4

TAPs without Toxicity Criteria

Chemical	Total Emissions (kg/yr)[1]
Bromine	1.1
n-Butanol	26.5
s-Butanol	0.9
Ethyl alcohol	616
Hydrogen Fluoride	17.8
Lead	2,941
Phosphorus	63.9

[1] Phase I emission estimates.

Source: Camp Dresser & McKee Inc., 1998.

Lead, which is a significant community health concern in many areas, may be released in significant quantities (2,941 kg/yr) from LAX. Lead is, therefore, retained as a TAP of potential concern. Potential risks associated with lead exposure may be evaluated through comparison of maximum modeled concentrations of lead with the National Ambient Air Quality Standard (1.5 micrograms per cubic liter [$\mu g/L^3$]).

2.5.2 Estimation of Relative Impact for Chemicals with Toxicity Criteria

In this section, relative impacts for individual TAPs are estimated and chemicals expected to contribute very little to overall human health impacts associated with LAX emissions are eliminated. Only chemicals associated with at least 0.1 percent of total relative impacts were retained as TAPs of concern based on USEPA guidance.[34] Relative impacts for TAPs were determined using both Phase I and Phase II emission estimates in separate analyses.

Relative impact factors for individual TAPs were estimated by multiplying annual emission rates by CSFs for chemicals that are carcinogens, or by dividing the annual emission rate by RfDs, for chemicals assessed as non-carcinogens. Individual impact factors for carcinogenic and non-carcinogenic TAPs were summed separately, providing the overall impacts. Division of individual impact factors by the overall impact value provides the fraction of total impacts contributed by each individual TAP. CSFs and RfDs derived by USEPA[35] and CalEPA[36] for evaluation of risks to human health were used in this analysis (see Section 5, *Toxicity Assessment*).

Impact factors are not expressions of health risk. Instead, they are an expression of the relative importance of TAPs released from LAX based on human toxicity. For example, a potent carcinogen released in relatively large amounts presents a greater potential threat than a weak carcinogen released in relatively small amounts. However, one cannot determine if neither or both chemicals present a real health threat without evaluating exposure to populations living, working, or recreating near LAX. Thus, impact factors are appropriate for choosing TAPs of concern, but the analysis of risk requires more detailed analysis as presented in the final HHRA.

For systemic toxicants (those which cause non-carcinogenic effects), two relative impacts estimates were generated. First, relative impacts were estimated as described above, using both USEPA and CalEPA toxicity criteria. Second, relative impacts were estimated using only USEPA criteria. CalEPA toxicity criteria for non-carcinogens were derived from chemical-specific reference exposure levels (RELs) proposed in 1998 to protect the general public from long-term exposure to hazardous substances released to the environment. Since it is not currently known whether the proposed RELs will be retained

[34] USEPA, Risk Assessment Guidance for Superfund, Volume 1, Human Health evaluation Manual (Part A)", EPA/540-1-89/002, 1989.

[35] USEPA, Risk Assessment Guidance for Superfund, Volume 1, Human Health evaluation Manual (Part A)", EPA/540-1-89/002, 1989.

[36] CALEPA, Noncancer Chronic Reference Exposure Levels (RELs), 1997.

by CalEPA, relative impacts from TAPs were estimated with and without consideration of CalEPA RELs. LAX TAPs retained only on the basis of RELs were identified as TAPs of concern on a probationary basis pending the release of final recommendations by CalEPA.

Toxicity screening results are discussed below and are summarized in **Tables 5** through **9**. Toxicity screening results for the No Action/No Project Alternative are presented in Section 2.5.2.1, *No Action/No Project Alternative,* and results for the Alternative B year 2015 scenario are presented in Section 2.5.2.2, *Alternative B Year 2015*.

2.5.2.1 No Action/No Project Alternative

Toxicity screening results for the No Action/No Project Alternative are presented in **Table 5**, Toxicity Screening, No Action/No Project Alternative, Phase I/II Carcinogen Emissions, for carcinogens; and **Table 6**, Toxicity Screening, No Action/No Project Alternative, Non-Carcinogens – CalEPA RELs Not Included, Phase I/II Emissions, and **Table 7**, Toxicity Screening, No Action/No Project Alternative, Non-Carcinogens – CalEPA RELs Included, Phase I/II Emissions, for non-carcinogens. These results are discussed below.

Toxicity Screening for Carcinogens

Estimates based on emissions predicted during Phase I suggest that for carcinogens eight TAPs would likely contribute over 99.9 percent of potential risks. These eight chemicals consist of both VOCs (1,3-butadiene, benzene, formaldehyde, and acetaldehyde), semivolatiles (2,3,7,8-tetrachlorodibenzo(p)dioxin equivalents (TCDD), and metals (arsenic, cadmium, and nickel). Arsenic and 1,3-butadiene are predicted to dominate total impacts, accounting for approximately 36.3 and 36.1 percent of total impacts, respectively.

Toxicity screening results using Phase II estimates are similar to those using Phase I estimates, except that two additional chemicals, hexavalent chromium and beryllium, also were estimated to contribute 0.1 percent or more to overall impacts. These chemicals are also retained as TAPs of concern. TCDD was not predicted to contribute more than 0.1 percent during Phase II. It should be noted, however, that the revised aircraft emission estimates for Phase II did not include estimates for TCDD. Potential impacts for TCDD are therefore only from non-aircraft sources. For this reason, TCDD is retained as a TAP of concern based on the Phase I estimates.

Results based on Phase II estimates indicate that nine TAPs of concern would contribute over 99.9 percent to total expected impacts from LAX. Other carcinogens are not expected to contribute significantly to overall risks from LAX emissions and were not retained as TAPs of concern. Chemicals contributing less than 0.1 percent to total impacts were eliminated from further consideration and are shown in **Tables 5** through **9**. Carcinogenic PAHs were not eliminated based on this screening criterion since they have been the subject of public concern and, therefore, warrant additional evaluation. Further screening conducted for PAHs is described in Section 2.5.3, *Screening of TAPs of Concern for Deposition onto Soils.*

Toxicity Screening for Non-carcinogens (Not Considering RELs)

For systemic toxicants screened against existing (USEPA) RfDs, one chemical, acrolein, would contribute over 98.6 percent of the cumulative relative impacts of all TAPs evaluated (**Table 6**, Toxicity Screening, No Action/No Project Alternative, Non-Carcinogens – CalEPA RELs Not Included, Phase I/II Emissions). The chemicals with the second and third greatest estimated relative impacts, benzene and acetaldehyde, would contribute only 0.54 and 0.46 percent relative impact, respectively. While acrolein clearly dominates from a screening standpoint, toxicologically significant emissions of other TAPs might also occur. For this reason, relative impacts of all TAPs except acrolein were also separately assessed. Of these chemicals, eight additional non-carcinogens--(benzene (39.5 percent), acetaldehyde (33.8 percent), manganese (13.1 percent), arsenic (6.3 percent), cadmium (3.3 percent), naphthalene (1.9 percent), formaldehyde (1.4 percent), and toluene (0.5 percent)--would each account for over 0.1 percent of the remaining relative impacts. Combined, these chemicals comprise over 99.8 percent of total remaining impacts. Thus, a total of eight TAPs would account for essentially all non-cancer risks from emissions from LAX in the absence of the proposed CalEPA RELs.

Table 5

Toxicity Screening No Action/No Project Alternative Phase I/II Carcinogen Emissions

Phase I Emissions[1]				Phase II Emissions[1]			
Source Category	Total Operating (kg/year)	Emission Rate Slope Factor	Percent Relative Impact	Source Category	Total Operating (kg/yr)	Emission Rate Slope Factor	Percent Relative Impact
Arsenic	1,210	14,031	36.31%	Butadiene, 1,3	11,494	6,839	48.39%
Butadiene, 1,3	23,443	13,949	36.10%	Benzene	30,760	3,138	22.20%
Benzene	43,251	4,412	11.42%	Nickel	2,328	2,118	14.99%
Formaldehyde	178,617	3,751	9.71%	Formaldehyde	52,285	1,098	7.77%
Cadmium	122	1,794	4.64%	Cadmium	23	343	2.43%
Acetaldehyde	55,530	525	1.36%	Chromium VI	0.51	268	1.89%
Nickel	143	130	0.34%	Acetaldehyde	16,447	155	1.10%
TCDD, 2,3,7,8 Equivalents	0.00	41.87	0.11%	Arsenic	11	124	0.87%
Benzo(a)pyrene	0.66	2.56	0.01%	Beryllium	2.56	18	0.13%
Perchloroethylene	81	1.67	0.00%	Dibenz(a,h)anthracene	3.39	14	0.10%
Methylene Chloride	422	1.48	0.00%	Benzo(a)pyrene	1.71	6.68	0.05%
Dibenz(a,h)anthracene	0.24	1.03	0.00%	Benzo(a)anthracene	6.39	2.49	0.02%
Benzo(a)anthracene	1.38	0.54	0.00%	Benzo(b)fluoranthene	4.71	1.84	0.01%
Chromium VI (all sources)	0.00	0.46	0.00%	Benzo(k)fluoranthene	4.53	1.77	0.01%
Benzo(b)fluoranthene	0.62	0.24	0.00%	Perchloroethylene	81	1.67	0.01%
Propylene Oxide	18	0.23	0.00%	Methylene Chloride	422	1.48	0.01%
Benzo(k)fluoranthene	0.44	0.17	0.00%	Indeno(1,2,3-cd)pyrene	2.71	1.06	0.01%
Indeno(1,2,3-cd)pyrene	0.24	0.09	0.00%	Chrysene	12	0.45	0.00%
Chrysene	1.06	0.04	0.00%	TCDD, 2,3,7,8 Equivalents	0.00	0.28	0.00%
Beryllium	0.00	0.03	0.00%	Propylene Oxide	18	0.23	0.00%
Acenaphthene	1.92	0.00	0.00%	Acenaphthene	8.96	0.00	0.00%
Acenaphthylene	8.26	0.00	0.00%	Acenaphthylene	18.69	0.00	0.00%
Acrolein	26,559	0.00	0.00%	Acrolein	7,144	0.00	0.00%
Anthracene	1.47	0.00	0.00%	Anthracene	63.06	0.00	0.00%
Benzaldehyde	5.95	0.00	0.00%	Benzaldehyde	2,366	0.00	0.00%
Benzo(g,h,i)perylene	0.94	0.00	0.00%	Benzo(g,h,i)perylene	3.08	0.00	0.00%
Copper	46	0.00	0.00%	Copper	93.27	0.00	0.00%
Ethylbenzene	69	0.00	0.00%	Ethylbenzene	2,064	0.00	0.00%
Fluoranthene	3.03	0.00	0.00%	Fluoranthene	194	0.00	0.00%
Fluorene	3.03	0.00	0.00%	Fluorene	3.03	0.00	0.00%
Hexane	96	0.00	0.00%	Hexane	6,589	0.00	0.00%
Lead	2,941	0.00	0.00%	Lead	1,716	0.00	0.00%
Manganese	120	0.00	0.00%	Manganese	605	0.00	0.00%
Mercury	0.62	0.00	0.00%	Mercury	0.74	0.00	0.00%
Naphthalene	1,026	0.00	0.00%	Naphthalene	12,276	0.00	0.00%
Phenanthrene	17	0.00	0.00%	Phenanthrene	639	0.00	0.00%
Pyrene	3.09	0.00	0.00%	Propylene	37,156	0.00	0.00%
Propylene	58,866	0.00	0.00%	Pyrene	162	0.00	0.00%
Selenium	114	0.00	0.00%	Selenium	0.40	0.00	0.00%
Trichloroethane, 1,1,1	185	0.00	0.00%	Trichloroethane, 1,1,1	185	0.00	0.00%
Styrene	5,626	0.00	0.00%	Styrene	4,027	0.00	0.00%
Toluene	38,984	0.00	0.00%	Toluene	48,394	0.00	0.00%
Xylene (total)	22,354	0.00	0.00%	Xylene (total)	28,324	0.00	0.00%
Zinc	1,903	0.00	0.00%	Zinc	3,183	0.00	0.00%

[1] Figures are as calculation; final risk estimates were rounded to two significant figures.

Source: Camp Dresser & McKee Inc., 1998.

Table 6

Toxicity Screening No Action/No Project Alternative Non-Carcinogens – Cal EPA RELS Not Included Phase I/II Emissions

	Phase I Emissions[1]					Phase II Emissions[1]			
Source Category	Total Operating (kg/year)	Emission/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)	Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)
Acrolein	26,559	4,651,281,585	98.64%	NA	Acrolein	7,144	1,251,170,384	93.78%	NA
Benzene	43,251	25,293,212	0.54%	39.53%	Manganese	605	42,330,744	3.17%	51.00%
Acetaldehyde	55,530	21,606,912	0.46%	33.77%	Benzene	30,760	17,988,277	1.35%	21.67%
Manganese	120	8,395,556	0.18%	13.12%	Naphthalene	12,276	14,323,877	1.07%	17.26%
Arsenic	1,210	4,031,896	0.09%	6.30%	Acetaldehyde	16,447	6,399,447	0.48%	7.71%
Cadmium	122	2,136,821	0.05%	3.34%	Beryllium	2.56	449,458	0.03%	0.54%
Naphthalene	1,026	1,200,000	0.03%	1.88%	Toluene	48,394	424,512	0.03%	0.51%
Formaldehyde	178,617	893,086	0.02%	1.40%	Cadmium	23	408,687	0.03%	0.49%
Toluene	38,984	341,968	0.01%	0.53%	Formaldehyde	52,285	261,425	0.02%	0.31%
Selenium	114	22,801	0.00%	0.04%	Nickel	2,328	116,377	0.01%	0.14%
Styrene	5,626	19,672	0.00%	0.03%	Hexane	6,589	115,400	0.01%	0.14%
Xylene (total)	22,354	11,177	0.00%	0.02%	Arsenic	11	35,509	0.00%	0.04%
Mercury	0.62	7,214	0.00%	0.01%	Benzaldehyde	2,366	23,657	0.00%	0.03%
Nickel	143	7,153	0.00%	0.01%	Phenanthrene	639	21,288	0.00%	0.03%
Zinc	1,903	6,343	0.00%	0.01%	Chromium VI	0.51	17,816	0.00%	0.02%
Propylene Oxide	17.80	2,080	0.00%	0.00%	Chromium	72	14,336	0.00%	0.02%
Hexane	96.38	1,688	0.00%	0.00%	Xylene (total)	28,324	14,162	0.00%	0.02%
Chromium VI (all sources)	1,260	1,260	0.00%	0.00%	Styrene	4,027	14,082	0.00%	0.02%
Copper	46	1,158	0.00%	0.00%	Zinc	3,183	10,609	0.00%	0.01%
Beryllium	0.00	770	0.00%	0.00%	Mercury	0.74	8,688	0.00%	0.01%
Trichloroethane, 1,1,1	185	647	0.00%	0.00%	Ethylbenzene	2,064	7,215	0.00%	0.01%
Phenanthrene	17	581	0.00%	0.00%	Pyrene	162	5,384	0.00%	0.01%
Perchloroethylene	81	575	0.00%	0.00%	Fluoranthene	194	4,849	0.00%	0.01%
Methylene Chloride	422	492	0.00%	0.00%	Copper	93	2,332	0.00%	0.00%
Acenaphthylene	8.26	275	0.00%	0.00%	Propylene Oxide	18	2,080	0.00%	0.00%
Ethylbenzene	69	241	0.00%	0.00%	Trichloroethane, 1,1,1	185	647	0.00%	0.00%
Pyrene	3.09	103	0.00%	0.00%	Acenaphthylene	19	623	0.00%	0.00%
Fluoranthene	3.03	76	0.00%	0.00%	Perchloroethylene	81	575	0.00%	0.00%
Fluorene	3.03	76	0.00%	0.00%	Methylene Chloride	422	492	0.00%	0.00%
Benzaldehyde	5.95	59	0.00%	0.00%	Chrysene	12	388	0.00%	0.00%
Benzo(a)anthracene	1.38	46	0.00%	0.00%	Benzo(a)anthracene	6.39	213	0.00%	0.00%
Chrysene	1.06	35	0.00%	0.00%	Anthracene	63	210	0.00%	0.00%
Acenaphthene	1.92	32	0.00%	0.00%	Benzo(b)fluoranthene	4.71	157	0.00%	0.00%
Benzo(g,h,i)perylene	0.94	31	0.00%	0.00%	Benzo(k)fluoranthene	4.53	151	0.00%	0.00%
Chromium Hexavalent (all sources)	0.00	31	0.00%	0.00%	Acenaphthene	8.96	149	0.00%	0.00%
Benzo(a)pyrene	0.66	22	0.00%	0.00%	Dibenz(a,h)anthracene	3.39	113	0.00%	0.00%
Benzo(b)fluoranthene	0.62	21	0.00%	0.00%	Benzo(g,h,i)perylene	3.08	103	0.00%	0.00%

Table 6

Toxicity Screening No Action/No Project Alternative Non-Carcinogens – Cal EPA RELS Not Included Phase I/II Emissions

	Phase I Emissions[1]					Phase II Emissions[1]			
Source Category	Total Operating (kg/year)	Emission/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)	Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)
Benzo(k)fluoranthene	0.44	15	0.00%	0.00%	Indeno(1,2,3-cd)pyrene	2.71	90	0.00%	0.00%
Dibenz(a,h)anthracene	0.24	8.14	0.00%	0.00%	Selenium	0.40	79	0.00%	0.00%
Indeno(1,2,3-cd)pyrene	0.24	8.11	0.00%	0.00%	Fluorene	3.03	76	0.00%	0.00%
Anthracene	1.47	4.92	0.00%	0.00%	Benzo(a)pyrene	1.71	57	0.00%	0.00%
Butadiene, 1,3	23,443	0.00	0.00%	0.00%	Butadiene, 1,3	11,494	0.00	0.00%	0.00%
Lead	2,941	0.00	0.00%	0.00%	Propylene	37,156	0.00	0.00%	0.00%
TCDD 2, 3, 7, 8 Equivalents	0.00	0.00	0.00%	0.00%	TCDD , 2,3,7,8 Equivalents	0.00	0.00	0.00%	0.00%
Propylene	58,866	0.00	0.00%	0.00%	Lead	1,716	0.00	0.00%	0.00%

NA = Not Applicable

[1] Figures are as calculation; final risk estimates were rounded to two significant figures.

Source: Camp Dresser & McKee Inc., 1998.

Table 7

Toxicity Screening No Action/No Project Alternative Non-Carcinogens – Cal EPA RELs Included Phase I/II Emissions

	Phase I Emissions[1]					Phase II Emissions[1]			
Source Category	Total Operating (kg/year)	Emission/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)	Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)
Acrolein	26,559	4,651,281,585	89.06%	NA	Acrolein	7,144	1,251,170,384	77.41%	NA
Formaldehyde	178,617	312,814,555	5.99%	54.77%	Nickel	2,328	162,765,332	10.07%	44.57%
Arsenic	1,210	141,139,880	2.70%	24.71%	Formaldehyde	52,285	91,567,306	5.67%	25.08%
Cadmium	122	42,661,715	0.82%	7.47%	Manganese	605	42,330,744	2.62%	11.59%
Acetaldehyde	55,530	21,606,912	0.41%	3.78%	Copper	93.27	16,334,849	1.01%	4.47%
Butadiene, 1,3	23,443	10,192,661	0.20%	1.78%	Zinc	3,183	12,383,676	0.77%	3.39%
Nickel	143	10,004,566	0.19%	1.75%	Beryllium	2.56	8,957,722	0.55%	2.45%
Manganese	120	8,395,556	0.16%	1.47%	Cadmium	23	8,159,444	0.50%	2.23%
Copper	46	8,113,988	0.16%	1.42%	Acetaldehyde	16,447	6,399,447	0.40%	1.75%
Zinc	1,903	7,404,473	0.14%	1.30%	1,3-Butadiene	11,494	5,019,032	0.31%	1.37%
Selenium	114	4,978,326	0.10%	0.87%	Naphthalene	12,276	4,776,483	0.30%	1.31%
Benzene	43,251	2,529,321	0.05%	0.44%	Chromium VI	0.51	2,225,102	0.14%	0.61%
Naphthalene	1,026	399,107	0.01%	0.07%	Benzene	30,760	1,798,828	0.11%	0.49%
Xylene (total)	22,354	391,489	0.01%	0.07%	Arsenic	10.65	1,243,019	0.08%	0.34%
Toluene	38,984	341,968	0.01%	0.06%	Xylene (total)	28,324	496,041	0.03%	0.14%
Propylene	58,866	68,688	0.00%	0.01%	Toluene	48,394	424,512	0.03%	0.12%
TCDD Equivalents	0.00	28,636	0.00%	0.01%	Hexane	6,589	115,400	0.01%	0.03%
Styrene	5,626	19,672	0.00%	0.00%	Propylene	37,156	43,356	0.00%	0.01%
Beryllium	0.00	15,347	0.00%	0.00%	Benzaldehyde	2,366	23,657	0.00%	0.01%
Mercury	0.62	7,214	0.00%	0.00%	Phenanthrene	639	21,288	0.00%	0.01%
Perchloroethylene	81	7,061	0.00%	0.00%	Selenium	0.40	17,286	0.00%	0.00%
Methylene Chloride	422	4,924	0.00%	0.00%	Chromium	71.68	14,336	0.00%	0.00%
Chromium VI (all sources)	0.00	3,812	0.00%	0.00%	Styrene	4,027	14,082	0.00%	0.00%
Propylene Oxide	18	2,080	0.00%	0.00%	Mercury	0.74	8,688	0.00%	0.00%
Hexane	96	1,688	0.00%	0.00%	Ethylbenzene	2,064	7,215	0.00%	0.00%
Trichloroethane, 1,1,1	185	1,620	0.00%	0.00%	Perchloroethylene	80.50	7,061	0.00%	0.00%
Chromium (Total) (all sources)	1,260	1,260	0.00%	0.00%	Pyrene	162	5,384	0.00%	0.00%
Phenanthrene	17	581	0.00%	0.00%	Methylene Chloride	422	4,924	0.00%	0.00%
Acenaphthylene	8.26	275	0.00%	0.00%	Fluoranthene	194	4,849	0.00%	0.00%
Ethylbenzene	69	241	0.00%	0.00%	Propylene Oxide	18	2,080	0.00%	0.00%
Pyrene	3.09	103	0.00%	0.00%	1,1,1-Trichloroethane	185	1,620	0.00%	0.00%
Fluoranthene	3.03	76	0.00%	0.00%	Acenaphthylene	19	623	0.00%	0.00%
Fluorene	3.03	76	0.00%	0.00%	Chrysene	12	388	0.00%	0.00%
Benzaldehyde	5.95	59	0.00%	0.00%	Benzo(a)anthracene	6.39	213	0.00%	0.00%
Benzo(a)anthracene	1.38	46	0.00%	0.00%	Anthracene	63	210	0.00%	0.00%
Chrysene	1.06	35	0.00%	0.00%	TCDD Equivalents	0.00	193	0.00%	0.00%
Acenaphthene	1.92	32	0.00%	0.00%	Benzo(b)fluoranthene	4.71	157	0.00%	0.00%
Benzo(g,h,i)perylene	0.94	31	0.00%	0.00%	Benzo(k)fluoranthene	4.53	151	0.00%	0.00%

Table 7

Toxicity Screening No Action/No Project Alternative Non-Carcinogens – Cal EPA RELs Included Phase I/II Emissions

	Phase I Emissions[1]					Phase II Emissions[1]			
Source Category	Total Operating (kg/year)	Emission/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)	Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)
Benzo(a)pyrene	0.66	22	0.00%	0.00%	Acenaphthene	8.96	149	0.00%	0.00%
Benzo(b)fluoranthene	0.62	21	0.00%	0.00%	Dibenz(a,h)anthracene	3.39	113	0.00%	0.00%
Benzo(k)fluoranthene	0.44	15	0.00%	0.00%	Benzo(g,h,i)perylene	3.08	103	0.00%	0.00%
Dibenz(a,h)anthracene	0.24	8.14	0.00%	0.00%	Indeno(1,2,3-cd)pyrene	2.71	90	0.00%	0.00%
Indeno(1,2,3-cd)pyrene	0.24	8.11	0.00%	0.00%	Fluorene	3.03	76	0.00%	0.00%
Anthracene	1.47	4.92	0.00%	0.00%	Benzo(a)pyrene	1.71	57	0.00%	0.00%

[1] Figures are as calculation; final risk estimates were rounded to two significant figures.

NA = Not Applicable

Source: Camp Dresser & McKee Inc., 1998.

Table 8

Toxicity Screening Alternative B Carcinogen Phase II Emissions

Source Category	Phase II Emissions[1] Total Emissions (kg/yr)	Slope Factor Emissions	Percent Relative Impact
Butadiene, 1,3	13,424	7,987.21	47.73%
Benzene	32,959	3,361.83	20.09%
Nickel	3,153	2,869.55	17.15%
Formaldehyde	62,548	1,313.51	7.85%
Cadmium	29	423.89	2.53%
Chromium VI	0.69	363.38	2.17%
Acetaldehyde	19,596	185.19	1.11%
Arsenic	14	163.22	0.98%
Beryllium	3.48	24.37	0.15%
Dibenz(a,h)anthracene	4.49	18.85	0.11%
Benzo(a)pyrene	2.15	8.37	0.05%
Benzo(a)anthracene	8.24	3.21	0.02%
Benzo(b)fluoranthene	6.15	2.40	0.01%
Benzo(k)fluoranthene	5.97	2.33	0.01%
Perchloroethylene	81	1.66	0.01%
Methylene Chloride	422	1.48	0.01%
Indeno(1,2,3-cd)pyrene	3.57	1.39	0.01%
Chrysene	15	0.60	0.00%
TCDD, 2,3,7,8 Equivalents	0.00	0.28	0.00%
Propylene Oxide	18	0.23	0.00%
Trichloroethane, 1,1,1	185	0.00	0.00%
Acenaphthene	11	0.00	0.00%
Acenaphthylene	22	0.00	0.00%
Acrolein	8,634	0.00	0.00%
Anthracene	84.53	0.00	0.00%
Benzaldehyde	2,851	0.00	0.00%
Benzo(g,h,i)perylene	3.85	0.00	0.00%
Copper	110	0.00	0.00%
Ethylbenzene	2,494	0.00	0.00%
Fluoranthene	261	0.00	0.00%
Fluorene	3.03	0.00	0.00%
Hexane	8,002	0.00	0.00%
Manganese	780	0.00	0.00%
Mercury	0.79	0.00	0.00%
Naphthalene	16,311	0.00	0.00%
Phenanthrene	856	0.00	0.00%
Propylene	43,432	0.00	0.00%
Pyrene	217	0.00	0.00%
Selenium	0.54	0.00	0.00%
Styrene	4,668	0.00	0.00%
Toluene	51,547	0.00	0.00%
Xylene (total)	30,633	0.00	0.00%
Zinc	4,093	0.00	0.00%

PRI = Percent Relative Impact

Note: Non-carcinogenic TAPs, which by definition do not have slope factors, are included in this table in order to present emissions.

[1] Figures are as calculated; risk estimates based on available emissions were rounded to two significant figures.

Source: Camp Dresser & McKee Inc., 1998.

Table 9

Toxicity Screening Alternative B Non-Carcinogen Phase II Emissions

	Phase II Emissions – Cal EPA RELs Not Included[1]					Phase II Emissions – Cal EPA RELs Included[1]			
Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)	Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)
Acrolein	8,634	1,512,152,483	93.63%	NA	Acrolein	8,634	1,512,152,483	76.30%	NA
Manganese	780	54,519,544	3.38%	53.00%	Nickel	3,153	220,514,219	11.13%	46.94%
Benzene	32,959	19,274,316	1.19%	18.74%	Formaldehyde	62,548	109,541,097	5.53%	23.32%
Naphthalene	16,311	19,032,970	1.18%	18.50%	Manganese	780	54,519,544	2.75%	11.61%
Acetaldehyde	19,596	7,625,053	0.47%	7.41%	Copper	110	19,292,528	0.97%	4.11%
Beryllium	3.48	610,653	0.04%	0.59%	Zinc	4,093	15,926,170	0.80%	3.39%
Cadmium	29	505,005	0.03%	0.49%	Beryllium	3.48	12,170,357	0.61%	2.59%
Toluene	51,547	452,163	0.03%	0.44%	Cadmium	29	10,082,437	0.51%	2.15%
Formaldehyde	62,548	312,740	0.02%	0.30%	Acetaldehyde	19,596	7,625,053	0.38%	1.62%
Nickel	3,153	157,668	0.01%	0.15%	Naphthalene	16,311	6,346,792	0.32%	1.35%
Hexane	8,002	140,140	0.01%	0.14%	Butadiene, 1,3	13,424	5,836,468	0.29%	1.24%
Arsenic	14	46,903	0.00%	0.05%	Chromium VI	0.69	3,022,470	0.15%	0.64%
Phenanthrene	856	28,526	0.00%	0.03%	Benzene	32,959	1,927,432	0.10%	0.41%
Benzaldehyde	2,851	28,510	0.00%	0.03%	Arsenic	14	1,641,877	0.08%	0.35%
Chromium VI	0.69	24,201	0.00%	0.02%	Xylene (total)	30,633	536,476	0.03%	0.11%
Styrene	4,668	16,323	0.00%	0.02%	Toluene	51,547	452,163	0.02%	0.10%
Chromium	79	15,771	0.00%	0.02%	Hexane	8,002	140,140	0.01%	0.03%
Xylene (total)	30,633	15,316	0.00%	0.01%	Propylene	43,432	50,679	0.00%	0.01%
Zinc	4,093	13,643	0.00%	0.01%	Phenanthrene	856	28,526	0.00%	0.01%
Mercury	0.79	9,225	0.00%	0.01%	Benzaldehyde	2,851	28,510	0.00%	0.01%
Ethylbenzene	2,494	8,719	0.00%	0.01%	Selenium	0.54	23,819	0.00%	0.01%
Pyrene	217	7,233	0.00%	0.01%	Styrene	4,668	16,323	0.00%	0.00%
Fluoranthene	261	6,514	0.00%	0.01%	Chromium	79	15,771	0.00%	0.00%
Copper	110	2,754	0.00%	0.00%	Mercury	0.79	9,225	0.00%	0.00%
Propylene Oxide	18	2,077	0.00%	0.00%	Ethylbenzene	2,494	8,719	0.00%	0.00%
Acenaphthylene	22	744	0.00%	0.00%	Pyrene	217	7,233	0.00%	0.00%
Trichloroethane, 1,1,1	185	647	0.00%	0.00%	Perchloroethylene	81	7,061	0.00%	0.00%
Perchloroethylene	81	575	0.00%	0.00%	Fluoranthene	261	6,514	0.00%	0.00%
Chrysene	15	513	0.00%	0.00%	Methylene Chloride	422	4,924	0.00%	0.00%
Methylene Chloride	422	492	0.00%	0.00%	Propylene Oxide	18	2,077	0.00%	0.00%
Anthracene	85	282	0.00%	0.00%	Acenaphthylene	22	744	0.00%	0.00%
Benzo(a)anthracene	8.24	275	0.00%	0.00%	Trichloroethane, 1,1,1	185	647	0.00%	0.00%
Benzo(b)fluoranthene	6.15	205	0.00%	0.00%	Chrysene	15	513	0.00%	0.00%
Benzo(k)fluoranthene	5.97	199	0.00%	0.00%	Anthracene	85	282	0.00%	0.00%
Acenaphthene	11	190	0.00%	0.00%	Benzo(a)anthracene	8.24	275	0.00%	0.00%
Dibenz(a,h)anthracene	4.49	150	0.00%	0.00%	Benzo(b)fluoranthene	6.15	205	0.00%	0.00%
Benzo(g,h,i)perylene	3.85	128	0.00%	0.00%	Benzo(k)fluoranthene	5.97	199	0.00%	0.00%
Indeno(1,2,3-cd)pyrene	3.57	119	0.00%	0.00%	TCDD equivalents	0.00	193	0.00%	0.00%

Table 9

Toxicity Screening Alternative B Non-Carcinogen Phase II Emissions

	Phase II Emissions – Cal EPA RELs Not Included[1]					Phase II Emissions – Cal EPA RELs Included[1]			
Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)	Source Category	Total Operating (kg/yr)	Emissions/ RFD	Percent Relative Impact (w/ acrolein)	Percent Relative Impact (w/o acrolein)
Selenium	0.54	109	0.00%	0.00%	Acenaphthene	11	190	0.00%	0.00%
Fluorene	3.03	76	0.00%	0.00%	Dibenz(a,h)anthracene	4.49	150	0.00%	0.00%
Benzo(a)pyrene	2.15	72	0.00%	0.00%	Benzo(g,h,i)perylene	3.85	128	0.00%	0.00%
Butadiene, 1,3	13,424	0	0.00%	0.00%	Indeno(1,2,3-cd)pyrene	3.57	119	0.00%	0.00%
Propylene	43,432	0	0.00%	0.00%	Fluorene	3.03	76	0.00%	0.00%
Lead	1,726	0	0.00%	0.00%	Benzo(a)pyrene	2.15	72	0.00%	0.00%
TCDD equivalents	0.00	0	0.00%	0.00%	Lead	1,726	0	0.00%	0.00%
Xylene, m- or p-	9,474	0	0.00%	0.00%	Xylene, m- or p-	9,474	0	0.00%	0.00%
Xylene, o-	3,521	0	0.00%	0.00%	Xylene, o-	3,521	0	0.00%	0.00%

NA = Not Applicable

[1] Figures are as calculated; risk estimates based on available emissions were rounded to two significant figures.

Source: Camp Dresser McKee & Inc., 1998.

When Phase II emission estimates were used, acrolein again accounted for the majority (93.8 percent) of estimated impacts. When the impacts of acrolein are removed, manganese would account for 51.0 percent of the remaining impacts, followed by benzene (21.7 percent), naphthalene (17.3 percent), acetaldehyde (7.7 percent), beryllium (0.5 percent), toluene (0.5 percent), cadmium (0.5 percent), formaldehyde (0.3 percent), nickel (0.1 percent), and hexane (0.1 percent). Hexane and nickel were not identified in screening results based on Phase I emissions, but were retained as TAPs of concern based on the revised (Phase II) emission estimates.

Toxicity Screening for Non-carcinogens (With RELs)

Toxicity screening results for non-carcinogenic effects based on RELs are presented in **Table 7**, Toxicity Screening, No Action/No Project Alternative, Non-Carcinogens – CalEPA RELs Included, Phase I/II Emissions. When Phase I emission estimates of non-carcinogens were screened against proposed CalEPA RELs, acrolein accounted for approximately 89.1 percent of total relative impacts, followed by formaldehyde (6.0 percent of total impacts), arsenic (2.7 percent), cadmium (0.8 percent), acetaldehyde (0.4 percent), 1,3-butadiene (0.2 percent), nickel (0.2 percent), manganese (0.2 percent), copper (0.2 percent), zinc (0.1 percent), and selenium (0.1 percent). Not considering acrolein, TAPs accounting for at least 0.1 percent of total impacts the same as those selected when acrolein is included, except that benzene (0.4 percent) is also included.

Toxicity screening with RELs using Phase II emission estimates were similar to those using Phase I estimates, except that toluene, xylene, beryllium, and hexavalent chromium were added as TAPs of concern. These chemicals were estimated to contribute less than 0.1 percent to overall impacts using the Phase I emission estimates, but were expected to contribute more than 0.1 percent based on Phase II emission estimates, when impacts are estimated without including acrolein.

Chemicals not selected for further evaluation are not expected to contribute substantially to total impacts from LAX operations, individually or in combination. The sum of the estimated relative impacts of all non-carcinogenic TAPs not selected for further evaluation is less than 0.01 percent (**Table 7**, Toxicity Screening, No Action/No Project Alternative, Non-Carcinogens – CalEPA RELs Included, Phase I/II Emissions.

Toxicity Screening for Lead

Toxicity criteria, cancer slope factors, and/or reference doses are not available for lead. This TAP is usually assessed using models to predict blood lead concentrations in exposed individuals. These concentrations are then compared to blood lead levels suspected of causing subtle neurological damage. To screen for lead, instead of assessing relative toxicity, maximum on- and off-site concentrations of lead predicted with the screening level air dispersion modeling were compared to the current USEPA ambient air quality standard (AAQS) of 1.5 :g/m^3. Even maximum predicted on-airport annual concentrations of lead (1.2 :g/m^3) were below this standard. At the LAX fence line and at the closest resident, maximum predicted annual lead concentrations in air were 0.2 and 0.05 :g/m^3, respectively. Recalling that these maximum concentrations are overestimated because of the conservative nature of the screening level air dispersion modeling, lead concentrations in air are highly unlikely to exceed the current ambient standard. Lack of impact of LAX operations on lead concentrations in air as defined by the AAQS indicate that lead is not a TAP of concern for the LAX Master Plan.

2.5.2.2 Alternative B Year 2015

The purpose of screening emissions for Alternative B year 2015 is to identify additional TAPs of concern that may be present in significant quantities in emissions for Alternative B year 2015, but not in emissions for the No Action/No Project Alternative.

Toxicity screening results for TAPs associated with emission estimates for Alternative B year 2015 is presented in **Tables 8** and **9**. **Table 8**, Toxicity Screening, Alternative B, Carcinogen Phase II Emissions, presents screening based on carcinogens; **Table 9**, Toxicity Screening, Alternative B, Non-carcinogen Phase II Emissions, presents screening based on non-carcinogenic effects from TAPs. Results indicate that no additional TAPs of concern will need to be evaluated based on Alternative B year 2015. Since lead is not an important TAP in jet exhaust, further evaluation of lead as a TAPs of concern for release after implementation of Alternative B was not necessary.

TAPs of concern for emissions from LAX, identified in screening steps 1 through 5 are identified in **Table 10**, Toxic Air Pollutants of Concern for LAX.

2.5.3 Screening of TAPs of Concern for Deposition onto Soils

This section evaluates TAPs of concern for soil. TAPs released from LAX could deposit onto soils and other surfaces, and residents, workers, and other receptors might then be exposed to these chemicals via incidental ingestion of soil, and dermal contact with contaminated soil. These pathways are further discussed in Section 4, *Preliminary Exposure Assessment*. This section evaluates the potential for chemicals to accumulate in soil.

Table 10

Toxic Air Pollutants of Concern for LAX

Substance[1]	CAS Number	Chemical Class
Acetaldehyde	5-07-0	Volatile organic
Acrolein	107-02-8	Volatile organic
Arsenic	7440-38-2	Metalloid
Benz(a)anthracene	556-55-3	Carcinogenic PAH
Benzene	71-43-2	Volatile organic
Benzo(b)fluoranthene	205-99-2	Carcinogenic PAH
Benzo(k)fluoranthene	207-08-9	Carcinogenic PAH
Benzo(a)pyrene	50-32-8	Carcinogenic PAH
Beryllium[2]	7440-41-7	Metal
Butadiene, 1,3	106-99-0	Volatile organic
Cadmium	7440-43-9	Metal
Chromium (total) (evaluated as Cr(VI))	1606508301	Metal
Chrysene	218-01-9	Carcinogenic PAH
Copper[3]	7440-50-8	Metal
Dibenz(a,h)anthracene	53-70-3	Carcinogenic PAH
Formaldehyde	50-00-0	Volatile organic
Hexane [4]	110-54-3	Aliphatic Hydrocarbon
Indeno(1,2,3-cd)pyrene	193-39-5	Carcinogenic PAH
Lead	7439-92-1	Metal
Manganese	7439-96-5	Metal
Naphthalene[4]	91-20-3	PAH
Nickel	7440-02-0	Metal
Selenium[3]	7782-40-2	Metal
Toluene	108-88-3	Volatile organic
Xylene[3]	1330-20-7	Volatile organic
Zinc[3]	7440-66-6	Metal
TCDD, 2,3,7,8 Equivalents	1746-01-6	Chlorinated Dioxins and Furans

1 Carcinogenic PAHs were retained as TAPs of concern, even though they were not identified in the toxicity screening. This group of chemicals was retained due to public concern with PAHs.
2 Greater than 0.1 percent relative impact under No Action/No Project Alternative Year 2015 emissions and only if CalEPA RELs adopted.
3 Selected only if proposed CalEPA RELs are adopted.
4 Greater than 0.1 percent relative impact under No Action/No Project Alternative Year 2015 emissions scenario.

Source:: Camp Dresser & McKee Inc., 1998.

Screening of TAPs of concern for indirect exposure pathways consisted of the following steps:

- Volatile chemicals were eliminated.
- Concentrations for TAPs in soil were estimated from results of air dispersion and deposition modeling.
- Estimated TAP concentrations were compared to background concentrations.

These steps are discussed in the following sections.

2.5.3.1 Elimination of Volatile TAPs

Not all TAPs of concern would deposit and accumulate in soils or other media. Most volatile organic compounds, for example, would not be expected to deposit in significant quantities onto soils and other media since, based on properties such as vapor pressure, they are more likely to remain in this atmosphere. Further, any volatile chemical deposited in association with particles or in precipitation would

rapidly re-volatilize. For volatile chemicals, impacts from pathways other than inhalation can thus be assumed to be negligible.

Volatile organic compounds are generally defined as chemicals having Henry's law constants greater than 0.00001 atmosphere-cubic meters per mole (atm-m^3/mol), and a molecular weight of less than 200 grams per mole (g/mole).[37] Applying these criteria to the list of TAPs of concern for LAX, the following organic TAPs are defined as volatile.

- Acetaldehyde
- Acrolein
- Benzene
- 1, 3-butadiene
- Trichloroethylene
- Formaldehyde

These chemicals were not assessed for indirect exposure pathways following deposition onto soils.

All other TAPs of concern for LAX were further evaluated as potential TAPs of concern for soil, including:

- Arsenic
- Cadmium
- Chromium (hexavalent)
- Lead
- Manganese
- Nickel
- Polycyclic aromatic hydrocarbons (carcinogenic PAHs only)

2.5.3.2 Estimation of TAP Concentrations in Soil

For TAPs of concern that would be expected to deposit onto soils, estimates of deposition were made by multiplying modeled annual average air concentrations (in units of micrograms per cubic meter [µg/m^3]) by a generic deposition velocity of 0.0018 meters per second (m/s) (see Section 3*, Screening Level Air Dispersion Modeling for Toxic Air Pollutants*, for a description of model methodology. Deposition velocity provides an estimate of the rate at which an atmospheric TAP may settle onto soil, where it may accumulate). This multiplication results in deposition rates in units of micrograms per square meter per second (µg/m^2/s). For use in estimation of concentrations of TAPs in soil, these values were multiplied by 86,400 (the number of seconds in one day) to convert µg/m^2/s to micrograms per square meter per day (µg/m^2/d).

Methods used to estimate concentrations of TAPs in soil over time from air particulate deposition were taken from Air Toxics Hot Spot Program Risk Assessment Guidelines.[38] The model used to estimate soil concentration of chemicals of potential concern (COPCs) is:

$$C_s = Dep \times X/(K_s \times SD \times BD \times T_t)$$

Where:

	C_s	=	Average soil concentration over the evaluation period (µg/kg)
	Dep	=	Deposition on affected soil area per day (µg/m^2/d)
	Ks	=	Soil elimination constant (chemical specific, 1/day)
	SD	=	Soil mixing depth (0.01 meter)
	BD	=	Soil bulk density (1,000 kg/m^3)
	Tt	=	Total days of exposure (36,500 days)
	X	=	$\{[EXP(-K_s \times T_t) - EXP(-Ks \times To)]/Ks\} + T_t$

[37] USEPA, Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part b. Development of Risk-Based Preliminary Remediation Goals, 1991.

[38] California Air Pollution Control Officers Association, California Environmental Protection Agency, and California Air Resources Board, Air Toxics Hot Spots Program, Revised 1992, Risk Assessment Guidelines, October 1993.

Deposition of TAPs to soil, Dep, expressed as µg deposited/m^2/day, was obtained from air deposition modeling.

The soil elimination constant, K_s, was calculated:

$$K_s = 0.693/t_{½}$$

Where: 0.693 =natural log of 2

$t_{1/2}$ =chemical specific soil half-life (days)

Using the results of dispersion modeling for the No Action/No Project Alternative 2015 (Section 3*, Screening-Level Air Dispersion Modeling for Toxic Air Pollutants)*, and applying the above methods, possible soil concentrations for PAH and metals were estimated (**Table 11**, Comparisons of Estimated Soil Concentrations with Background Concentrations for Soil. Calculations of estimated soil concentrations due to deposition of LAX TAPs of concern are provided in Attachment B2, *Calculation of Estimated Soil Concentrations from Particle Deposition*.

Table 11

Comparison of Estimated Soil Concentration with Background Concentrations for Soil

Element	Estimated[1] (mg/kg)	Background[2] (Geomean)	GSD [2]	Standardized Value [4]	Percentile [5]
On-Site					
Arsenic	2.6	5.5	1.98	-0.37	35.3
Cadmium	0.25				
Chromium (as Cr(VI))	0.06	41	2.19	-2.97	0.2
Lead	2.7	17	1.8	-1.02	15.4
Manganese	0.002	380	1.8	-6.9	0.0
Nickel	0.25	15	2.1	-1.96	2.5
Off-Site					
Arsenic	0.49	5.5	1.98	-1.2	10.9
Cadmium	0.046				
Chromium, total	0.49	41	2.19	-3.7	2.1
Chromium (as Cr(VI))	0.01	41	2.19	-2.0	0.01
Lead	0.5	17	1.8	-1.96	2.5
Manganese	0.0008	380	1.8	-7.3	0.0
Nickel	0.046	15	2.1	-2.8	0.3
On-Site	**Estimated (µg/kg)**			**Background (µg/kg)**	
PAH					
Benzo(a)anthracene	11.5			900 Benzo(a) pyrene Equivalents	
Benzo(a)pyrene	1.3				
Benzo(b)fluoranthene	7.4				
Benzo(k)fluoranthene	8.1				
Chrysene	4.2				
Dibenzo(a,h,)anthracene	1.8				
Indeno(1,2,3-cd)pyrene	1.3				

1 Attachment B2 (Calculation of Estimated Soil Concentrations from Particle Deposition.

2 Shacklette and Boerngen, Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States, US Geological Survey Professional Paper 1270, 1984.

3 GSD - Geometric Standard Deviation. The GSD is a measure of variance from the geometric mean within a distribution.

4 Standardized value.

5 A percentile is a measure that indicates the magnitude of an individual value within a population. For example, an arsenic concentration that occupies the 33rd percentile is lower than 67 percent of the other values in the distribution.

Source: Camp Dresser & McKee Inc., 1998.

2.5.3.3 Comparison with Background Concentrations

Estimated soil concentrations were compared to background concentrations to evaluate relative overall impacts from emissions to soil at and near LAX (**Table 11**, Comparisons of Estimated Soil Concentrations with Background Concentrations for Soil). Background concentrations are geometric mean

concentrations along with geometric standard deviations for the western United States, including California.[39] Using these values to define a distribution of background levels, the percentiles for the estimated contributions from LAX emissions were calculated. That is, using standard statistical methods based on a normal distribution for the natural logs of background data, the position of the LAX contribution on the distribution of background concentrations was calculated. The ranges of concentrations reported by Shacklette and Boerngen span a wide range and reflect many different geologies and soil types. By using the whole range of background concentrations in comparisons, background concentrations in the LAX area will be included. Thus, if the percentile estimated is low, the LAX contribution falls at the low end of background and contribution is small relative to naturally occurring concentrations even if background at LAX falls in the low end of the reported range. Where percentiles are higher, the contribution of LAX could be important for consideration in the final HHRA.

Estimated percentiles for TAPs of concern are presented in **Table 11**, Comparisons of Estimated Soil Concentrations with Background Concentrations for Soil. For on-site soils, all except two of the estimated percentiles are less than 1, indicating that the estimated contribution from LAX would be less than 98 percent of all background estimates in the western United States.

Background concentrations were reported in Shacklette and Boerngen for all LAX-related metals except cadmium.[40] However, the estimated cadmium concentration resulting from LAX operations (0.39 mg/kg) is two orders of magnitude lower than the USEPA Region III risk-based concentration for cadmium in residential soil (39 mg/kg).[41] This observation suggests that cadmium from LAX is unlikely to present a significant health risk based on soil exposures.

For arsenic, the percentile falls at about the 35th percentile. The LAX contribution for this metalloid would be less than 65 percent of all background estimates in the Western United States. The estimate for impacts of arsenic is based on Phase I emission rates. Refined estimates of arsenic released in Phase II suggest that arsenic emissions would be much less than originally estimated (11 kg/yr versus 1,200 kg/yr). Possible impacts of arsenic are, therefore, greatly exaggerated in this analysis.

Analysis of soil deposition suggests that estimated contributions from LAX emissions would make no measurable difference in expected background concentrations of metals.

Onsite estimates of deposition of LAX-related TAPs are very conservative. The assumption was made that all metals deposited would stay in the soil indefinitely. On-site, most surfaces are asphalt or concrete and TAPs deposited would be easily removed by rain, wind, and efforts made by the airport to keep areas clean. Concentrations of metals in dusts at the airport would be likely to reflect air concentrations at a given time, not accumulation over many decades. Thus, estimates of metal deposition are likely to significantly overestimate possible impacts of LAX-related TAPs.

The conservative nature of the calculations and the low or negligible impacts predicted by these conservative calculations argue that quantitative analysis of exposures and risks for pathways associated with contaminants in soil are unnecessary. Therefore, no further quantitative analysis of soil/dust related exposure pathways for metals will be included in the final HHRA. However, the above arguments concerning arsenic are re-evaluated in the final HHRA.

For off-site locations, as represented by the locations with the highest air concentrations at the current LAX fence line, percentiles are so low that essentially all background estimates for the Western United States would exceed the estimated contribution from LAX. At all off-site locations, deposition of metals onto soils would be negligible.

Deposition of PAHs onto soil would also be very small (**Table 11**, Comparisons of Estimated Soil Concentrations with Background Concentrations for Soil). The highest concentration of any PAH estimated for soil/dust is 11.5 microgram per kilogram (µg/kg) for benzo(a)anthracene. This concentration is below detection limits and perhaps one order of magnitude less than urban background concentrations for PAHs in the Los Angeles area (900 µg/kg as benzo(a)pyrene equivalents).[42]

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[39] Shacklette, H. and J. Boerngen, Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States, U.S. Geological Survey Professional Paper 1270, 1984.

[40] Shacklette, H. and J. Boerngen, Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States, U.S. Geological Survey Paper 1270, 1984.

[41] USEPA, Risk Assessment Guidance for Superfund, Volume 1, Human Health evaluation Manual (Part A)", EPA/540-1-89/002, OERR, 1989.

[42] CDM, PAHs in Los Angeles Background, 1997.

Even at the on-site location where this estimate was generated, the LAX contribution to PAH in soil/dust would be negligible. Therefore, risk estimates for pathways involving contaminated soil are not necessary in the final HHRA.

Possible impacts of dioxins (as TCDD equivalents) to soils were not assessed by estimating impacts to soils because reliable estimates of dioxins in "background" urban locations were not available. However, some data have been collected on TCDD deposition in urban areas in the U.S. and Europe. These measurements suggest that 2 to 6 mg of TCCD equivalents may be deposited per square meter of soil per year in urban settings.[43] These values translate into deposition rates of 5 to 16 x 10^{-6} µg/m^2/d. Air dispersion modeling suggests that deposition rates for dioxins might be many orders of magnitude less, on the order of 6 x 10^{-8} µg/m^2/d and 9 x 10^{-9} µg/m^2/d on-site and at the fence line, respectively. These very low deposition rates indicate that deposition of dioxins onto soils would be highly unlikely to result in measurable increases in concentrations of dioxins in soil. Therefore, soil-associated pathways will not be evaluated for dioxins in the final HHRA.

2.5.3.4 Deposition of TAPs to Surface Water and Sediment

Deposition of TAPs to surface water and sediment was not quantitatively evaluated. The minimal impacts of deposition of TAPs on background concentrations in soils indicates that the potential for impacts to streams is also minimal. The greatest concentrations of TAPs in sediments due to either direct deposition or runoff from adjacent soils would be unlikely to exceed the maximum concentrations estimated for deposition onto soils. Since these former impacts to soil concentrations are too small to be of concern, impacts to TAP concentrations in sediment would also be small.

Concentrations of TAPs in surface water are also expected to be negligible since TAPs of concern for deposition (metals, PAHs, and dioxins) are not highly soluble, and these TAPs would not be expected to be present in the dissolved phase. Further, both dissolved and suspended particulates in surface water would be rapidly carried away from LAX by stream flow or ocean currents. Since only minute amounts of TAPs might be deposited per unit time, no conditions are foreseen where concentrations of TAPs in water would build up to an unacceptable level.

Potential human health impacts from deposition of TAPs to surface water and sediment are anticipated to be negligible. Such health impacts are not further evaluated.

2.6 Uncertainties

Selection of TAPs of concern for LAX results in a relatively short list of chemicals that are expected to contribute over 99 percent of any risks to on-site or off-site receptors. Uncertainties in this selection process, and their possible impact on estimated risks, are discussed below for two key components, the toxicity screening and the analysis of deposition of TAPs onto soil.

2.6.1 Toxicity Screening

Toxicity screening is intended to focus the HHRA on TAPs that would define the impact of the LAX Master Plan, and aid in evaluating any mitigation measures that might be necessary. The process used estimated relative impact for each TAP, but provided no indication of absolute impacts on receptors on or near the airport. Thus, impacts by chemicals such as 1,3-butadiene could be negligible despite its prominence in the screening. Likewise, chemicals not selected as TAPs of concern could, in theory, have a significant absolute impact even though this impact would probably be much less than that for evaluated TAPs. If the latter were true, the assessment of TAP-related risks might somewhat understate possible impacts from implementation of the LAX Master Plan.

Understatement of possible risks linked to the relative nature of the toxicity screening will, however, have no impact on the goals and objectives of the risk analysis. If a chemical that makes a minor contribution to risks is judged to be a significant risk, then other chemicals that make a major contribution will imply even greater risks. Further, any mitigation measures that might be applied to reduce threats implied by TAPs of concern would also reduce emissions, and associated risks, for other chemicals not addressed quantitatively. Although there are many sources of TAPs at LAX, only a few fundamentally different types of sources are present (e.g., turbines, internal combustion engines, maintenance facilities, tank farm).

[43] USEPA, Health Assessment Document for 2,3,7,8-Tetrachlorodiobenzo(p)dioxins and Related Compounds, Volume II (Exposure Assessment), EPA/600/BP-92/001c, 1994.

TAPs selected for quantitative evaluation are found in emissions from all of these sources. Thus, if emissions of 1,3-butadiene implied a substantial off-site impact, reducing 1,3-butadiene emissions would likely involve reducing all emissions of volatile organics including those not on the list of TAPs of concern.

2.6.2 Deposition Modeling

Several TAPs of concern might deposit onto soils, leading to exposure via direct contact with contaminated soils or through ingestion of home-grown produce grown in contaminated soils. The screening level assessment examined the potential significance of these exposure pathways using the results of screening-level air dispersion modeling and simple modeling of deposition.

Deposition modeling for TAPs of concern for LAX suggests no significant potential impact for either on-site or off-site soils/dusts. The screening was performed using results from air dispersion modeling for the No Action/No Project Alternative in 2015. Build alternatives, which imply substantially greater numbers of aircraft landings and takeoffs, could result in greater soil deposition than that projected for 2015 for the No Action/No Project Alternative.

The HHRA would, however, reach the same conclusions concerning soil deposition even if the analysis was completed with air dispersion modeling estimates from one of the build alternatives. At most, the LAX Master Plan would result in something less than twice the number of aircraft landings and takeoffs. If soil deposition increased by a factor of 2, the result would still be insignificant. Soil deposition seems not to be an important issue for emissions of TAPs from LAX.

2.6.3 Waterborne Exposures

Human exposure to TAPs that deposit onto and dissolve into water (i.e., Pacific Ocean) is possible. However, TAP dilution in the water column will be rapid; TAP concentrations in water are, therefore, likely to be many orders of magnitude lower than soil concentrations resulting from TAP deposition. In addition, besides the Pacific Ocean, no significant recreational water bodies are present in the area immediately surrounding LAX. Waterborne exposures are considered insignificant relative to other pathways and, as a result, exposure to TAPs in water are not addressed further.

2.7 Summary

TAPs of concern for LAX were selected in a multi-step process designed to ensure a conservative and protective analysis. Final TAPs of concern include those that are the most toxic and would be emitted in the greatest quantities. The list of TAPs includes volatile organics, metals, and semivolatile organic chemicals. These TAPs were selected on the basis of inhalation only. Potential impacts to soil would be too small to be measurable against urban background, and no TAPs of concern are selected for this medium.

3. SCREENING LEVEL AIR DISPERSION MODELING FOR TOXIC AIR POLLUTANTS

This section identifies assumptions and methodologies used in conducting air dispersion modeling to support the screening-level assessment of toxic air pollutants. The section is divided into the following subsections:

- Input to Screening-Level Air Dispersion Modeling
- Description of Air Dispersion Modeling
- Summary and Conclusions

3.1 Input to Screening Level Air Dispersion Modeling

3.1.1 Meteorological Data

One year of hourly meteorological data provided by SCAQMD were used for refined dispersion modeling. The 1-year file consists of hourly surface data from the Hawthorne SCAQMD meteorological observation station (Station No. 094) and twice daily mixing height data from the SCAQMD for 1981. The data set

consists of hourly values of wind speed, wind direction, surface air temperature, Pasquill-Gifford stability class, and interpolated mixing heights. The 1981 data set is generally considered to produce conservative estimates of annual pollutant concentrations in the South Coast Air Basin. Results of modeling using this data set are therefore appropriate for a screening level assessment, for which concentration estimates should exaggerate likely actual impacts.

3.1.2 Emission Estimates

Estimates for release of TAPs from sources at LAX currently, and in the future, are described in detail in Section 2, *Selection of Preliminary TAPs of Concern*. These estimates were used as inputs for air dispersion modeling to assist in defining an area of potential impact for LAX-related emissions (Section 4, *Preliminary Exposure Assessment*), and to assist in the evaluation of deposition of TAPs onto soils (Section 2, *Selection of Preliminary TAPs of Concern*). Modeling was performed only for emissions for the No Action/No Project Alternative.

It should be noted that results of modeling cannot be used to estimate potential risks to surrounding communities. As discussed in more detail in subsequent sections, screening level modeling estimates greatly exaggerates potential downwind TAP concentrations in air, and any exposure and risk estimates derived from these concentrations would be highly inaccurate. Potential relative impacts of TAPs found in LAX-related sources are evaluated in detail in Section 2, *Selection of Preliminary TAPs of Concern*, using a toxicity screening method independent of air dispersion modeling.

3.2 Dispersion Modeling

3.2.1 Model Selection

Dispersion modeling of pollutants generated by airports requires a model that can simulate TAP emissions from multiple point, area, line, and volume sources. The third generation of the Industrial Source Complex – Short-Term dispersion model (ISCST3, Version 97363) was used to calculate dispersion impacts from toxic air pollutant emissions produced by airport-related sources. The ISCST3 dispersion model is a steady-state Gaussian dispersion model capable of examining the short-term and annual average impacts from "complicated sources," such as point, area, or volume sources.[44] The FAA approved the use of ISCST3 to assess toxic air pollutant emissions for the LAX Master Plan EIS/EIR.[45]

3.2.2 Modeling Domain

Pollutant concentrations produced from airport sources were predicted at sufficient locations to identify maximum ambient air quality impacts from the airport sources for evaluation of possible impacts to soil, and to assist in defining an area of impact for use in the final HHRA. A Cartesian (rectangular) grid system was used with grid spacing of 100 meters for locations within 1 kilometer of the LAX Theme Building as well as for any receptors beyond 1 kilometer of the theme building but within 100 meters of the LAX fence line. Maximum impacts are expected within the fence line. A coarser grid was used at greater distances. For locations more than 100 meters beyond the fence line and more than 1 kilometer beyond the theme building, but less than 2 kilometers from the theme building grid spacing was increased to 200 meters. Grid spacing was increased to 500 meters for locations more than 100 meters beyond the fence line and more than 2 kilometers beyond the theme building, but less than 3 kilometers from the theme building.

3.2.3 Modeling Considerations

Building Downwash and Cavity Effects

Aircraft operations occurring on the runways and taxiways are expected to be the main contributor to TAP emissions. These sources are far enough from airport structures to avoid being influenced by building downwash. Downwash occurs when the exhaust plume from an emission source is trapped in the recirculation (eddy) zone on the leeward side of a building or structure. Since the impacts from other emission sources are expected to be located well within the airport boundaries, any aerodynamic effects

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[44] USEPA, User's Guide for the Industrial Source Complex (ISC3) Dispersion Models, EPA-454/B-95-003a, 1995.

[45] Landrum and Brown, LAX EIS/EIR Meeting Summary, FAA Headquarters, November 24, 1997.

on stack emissions due to nearby structures would be insignificant at publicly accessible receptor locations. Therefore, analyses of building downwash and cavity impacts was not performed.

Plume Rise

The bulk of emissions from LAX during normal operations would be from jet exhaust (Section 2.4.2, *Phase II Emission Estimates*). Plume rise would expected to be significant for these emissions because of high initial exhaust temperatures. For the screening level assessment, however, plume rise was not taken into account. Since plume rise will result in lower modeled downwind concentrations, omission of plume rise in the modeling will cause modeled concentrations to overestimate actual concentrations resulting from LAX operations. Such overestimation is acceptable for screening since model results will only be used to assess potential impacts to soil, and to help define a reasonable area of impact for population-based risk estimates.

Aircraft Operations

Emission estimates for aircraft were compiled for several modes of operation, including queue, taxi, takeoff, and climb out (see Attachment B3, *Emission Estimates Spreadsheets*). Time in queue (aircraft waiting on tarmac for permission to takeoff) is responsible for the highest estimated emissions for any operational mode. Since aircraft emissions dominated total emissions of TAPs of concern (Section 2.4.2, *Phase II Emission Estimates*), emissions during queue have the greatest influence on predicted downwind concentrations when considering emissions from all sources. Emissions for queue mode were modeled using a large area source appropriate for queue times during the busiest hours of operations at LAX. Results of screening level air dispersion modeling are used only for evaluation of deposition to soils and definition of an area of impacts. A very conservative analysis of queue is appropriate for these uses.

Terrain

The South Coast Air Basin is characterized by relatively flat terrain, and receptors are not expected to be located at substantial heights above LAX emission sources. Possible exceptions could be workers in high-rise office buildings near the airport. However, the screening level modeling does not take into account plume rise, and most sources at LAX would occur at ground level. Thus, downwind concentrations will be maximal at ground level in the screening level modeling.

Deposition onto Soils

Deposition of TAPs onto soils was estimated by applying a deposition velocity to estimated annual average air concentrations. A generic and conservative deposition velocity of 0.0018 milligrams per second (mg/s)[46] was used in the screening. This deposition velocity is favored by USEPA for analysis of deposition of radionuclides as particulates onto soils. The value is reasonably applied to non-radioactive metals, PAHs, and TCDD present as particulates in emissions from LAX.

Multiplying air concentrations in $\mu g/m^3$ by the deposition velocity gives an estimate of the amount of deposition of soil in units of $\mu g/m^2/sec$. Resulting deposition rates are used to estimate steady-state soil concentrations as a result of LAX-operations as described in Section 2, *Selection of Preliminary TAPs of Concern.*

3.2.4 Model Output

Model outputs are annual average concentrations for TAPs of concern within the model domain described above. Annual average concentrations are appropriate for preliminary analyses of possible LAX-related impacts, because chronic exposure integrates (averages) exposures over extended time periods. The analysis focuses on potential impacts on or near LAX so that maximal impacts are used in subsequent analyses. In particular, possible impacts to soil are evaluated at the locations on-site and at the LAX fence line where the highest predicted annual average concentrations occur. Example model input and output are provided for 1,3-butadiene in Attachment B4, *Example Model Output*.

[46] USEPA, User's Guide for CAP88-PC, PC Version 1.0. Radiation Protection Programs. EPA 402-B-92-001, Las Vegas, NV, March 1992.

3.3 Summary and Conclusions

Emission estimates, aircraft operations, and site-specific meteorological data were used to produce conservative estimates of possible air concentrations and deposition onto soils of TAPs of concern as a result of emissions during LAX operations. The analysis uses ISCST3, a USEPA approved air dispersion model with capabilities appropriate for evaluation of the many complex sources of TAPs present at LAX. Modeling is intended to be "screening level." That is, the modeling is designed to provide upper-bound air concentrations suitable for a preliminary analysis of important exposure pathways and areas of impact.

Important considerations for the screening level modeling include:

- Use of a meteorological data set known to produce conservative estimates of long-term average air concentrations
- Omission of plume rise for emissions from jet exhaust thereby reducing the amount of dispersion of this dominant source of TAPs of concern
- Use of near maximum queue times thereby maximizing emissions during the operational mode associated with the greatest overall emissions

The results of the screening level modeling are very conservative estimates of potential air concentrations on and near LAX during future operations. The results are useful for assessing the importance of soil deposition and for helping to define an area of impact for additional evaluation.

Air dispersion modeling results are not appropriate for estimation of possible off-site exposures because they are based on dated emission estimates and unrealistic aircraft operational assumptions, and do not consider plume rise. Refined modeling was necessary to support quantitative exposure estimates in the final HHRA.

Finally, screening level air dispersion modeling addressed a future No Action/No Project Alternative. Increased emissions as a result of increased aircraft operations are not included in the modeling results. Even though the modeling is very conservative, some caution is employed in assessing soil deposition and in defining areas of impact (Section 4, *Preliminary Exposure Assessment*).

4. PRELIMINARY EXPOSURE ASSESSMENT

Exposure assessment is a process for estimating the amounts of chemicals that may be taken into the body through exposures to chemicals in air, soil, water, or other media. The assessment generally results in estimates of chronic daily intake of chemicals of concern for persons likely to be exposed to substances released from waste sites, industrial facilities, and other sources of toxic materials. For the LAX HHRA, the purpose of the exposure assessment was to develop estimates of chronic daily intake for TAPs of concern for people living or working on or near LAX.

For this preliminary analysis, no estimates of chronic daily intake were developed. However, the basis for such estimates was developed through consideration of ways in which people living near LAX could be exposed to TAPs from LAX operations, and the areas surrounding LAX where impacts are likely to be greatest.

Methods used follow the SCAQMD CEQA Handbook Chapter 10 on *Air Toxics*. An associated reference methods of California Air Pollution Control Officers Association (CAPCOA) and California Air Resources Board (CARB), referenced with the subtext of the SCAQMD CEQA Handbook - Chapter 10, *Air Toxics*, were also followed.

The objective of the preliminary exposure assessment was to describe a series of scenarios that represent the potential for human exposure to TAPs released during airport operations. An exposure scenario is a characterization of the ways in which people may come into contact with LAX-related TAPs via air, soil, or other media. The preliminary exposure assessment considered scenarios based on proximity to LAX, type of land use (e.g., residential, commercial, or industrial), sensitive receptors (e.g., school children), and possible exposure parameters (e.g., inhalation rate, exposure frequency, and exposure duration). Scenarios for the final HHRA were defined by the outcome of the preliminary assessment in terms of:

- Locations of sensitive receptors and residential areas near LAX under baseline and expected future conditions
- Areas potentially affected by emissions during LAX operations

- Exposure pathways that would contribute substantially to overall exposures
- Exposure parameters appropriate for human receptors residing, attending school, or working on or near LAX

Analyses in the remainder of this section are presented in five subsections. The exposure setting is discussed in Section 4.1*, Exposure Setting*, potential receptors and exposure pathways are evaluated in Section 4.2, *Evaluation of Potential Receptors and Exposure Pathways*, exposure assessment methods and exposure parameters for each scenario are developed in Section 4.3, *Exposure Assessment Methods*, areas of impact for LAX emissions are identified in Section 4.4, *Areas of Potential Impact for LAX Emissions*, and a summary is provided in Section 4.5, *Summary*.

4.1 Exposure Setting

The potential for exposure to TAPs released during LAX operations is influenced by the nature of TAP sources (e.g., aircraft, maintenance operations, ground vehicles), the chemical nature of the TAPs, physical characteristics of receptors (e.g., inhalation rates, body weights), locations of human receptors, receptor activities, and routes of exposure (inhalation, ingestion, and dermal absorption). To characterize the exposure setting for evaluation of Master Plan implementation, existing conditions for areas within the likely zone of impact from LAX operations were assumed. Existing land use near the airport consists of a mixture of residential, commercial, industrial, recreational, and other uses, and this mixed use is expected to continue indefinitely. For example, development of Westchester Southside is planned to include a golf course, 1.1 million square feet of mixed uses (office, hotel, conference center, retail, restaurant, and entertainment), 920,000 square feet for an R/D business park and education (college), and 480,000 square feet for resort hotels. Exposure scenarios based on current land uses are applicable to any foreseeable pattern of land use near LAX in the future.

4.1.1 Demographics

Existing land uses, and a description of existing land use plans and regulations relevant to future and current land use in the LAX area, are summarized in the Technical Report 1, *Land Use*. Current land use near the airport shows low- to medium-density residential housing immediately adjacent to the airport to the north in Playa del Rey and Westchester, and a mixture of low-and high-density residential housing to the south in El Segundo. High-density housing is found closest to the ends of runways to the east of the LAX north runways and also in Westchester. Residents at the ends of runways would be expected to experience the greatest impacts from jet exhaust. Further, jet exhaust would likely be the greatest source of LAX-related air toxics under both the No Action/No Project Alternative and the three build alternatives (Section 2, *Selection of Preliminary TAPs of Concern*). Residents closest to ends of runways would, therefore, be expected to represent maximally exposed receptors for residential scenarios under all conditions.

Locations of schools and some day care centers also have been identified in previous studies summarized in the Technical Report 17, *Schools*. Ninety-nine schools, including both private and public institutions, and preschool through high school levels exist within a few kilometers of the LAX fence line (see Technical Report 17, *Schools)*. Schools and day care centers located within a one-mile radius of LAX include children from infants to 18-years-old.

Certain subpopulations may be more sensitive or susceptible to negative health impacts caused by environmental contaminants than the population at large.[47] Higher risks may occur due to increased genetic susceptibility or sensitivity, behavior patterns that can result in higher exposure, participation in activities, which result in higher risk for health problems than others (e.g., smoking, consumption of alcoholic beverages), and/or current or past exposures from other sources. Subpopulations that may be more sensitive to environmental contaminants include, but are not limited to, infants, children, the elderly, pregnant and nursing women, and people with chronic illnesses. These critical subpopulations are discussed below.

[47] USEPA, Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual Interim Final, December 1989.

- Schools: School children include all students enrolled in kindergarten through high school. A survey was conducted of the study area in 1996, and 99 schools were identified. Of these, approximately 20 schools lie within one mile of the LAX fence line. The school nearest the current LAX fence line is the Imperial Avenue School located at 540 E. Imperial Avenue in El Segundo. The 1997/1998-projected enrollment for this school was 80 students, ranging from kindergarten to 8th grades.
- Day care centers and preschools: Day care centers and preschools within the noise impact area for LAX were also identified. Forty-one preschool/day care centers were also identified. Of these centers, 14 facilities are located within one mile of the LAX fence line. The center nearest the LAX fence line is St. John's Lutheran Child Development Center at 16111 East Sycamore Avenue in El Segundo.
- Hospitals, nursing homes, and retirement communities: Patients and residents in hospitals, nursing homes, and retirement communities are critical subpopulations with increased sensitivity to the environmental contaminants. According to the 1990 census, 8 percent of the local population is in excess of 65 years of age in the area surrounding LAX. No hospitals are, however, located within one mile of LAX. The nearest hospital, Centinela Hospital, lies approximately 1.6 miles to the east.
- Residential areas with children: Children living in the immediate vicinity of the site or within the potential impact zones are believed to have higher sensitivity or susceptibility to contaminants. The area surrounding LAX includes mixed use and residential communities. The 1990 census reported a population of 441,375 within an expanded population area subject to noise impacts. Of this population, 131,794 people were determined to be less than 16 years of age.

A detailed survey of area demographics is included separately in the Technical Report 14a, *Human Health Risk Assessment* (Section 3, *Exposure Assessment*).

4.1.2 Existing Ambient Air Quality

The SCAQMD maintains a network of air quality monitoring stations throughout the South Coast Air Basin (Basin). Air quality data for the two stations closest to LAX are summarized in Section 2, *Selection of Preliminary TAPs of Concern*. Existing annual mean TAP concentration results from downtown Los Angeles (1630 North Main Street) and Long Beach (3648 N. Long Beach Boulevard) are presented in **Table 12**, Existing Annual Mean Toxic Air Pollutant Concentrations in Los Angeles Region.

Air quality data exist for three TAPs of concern for future emissions from LAX, including benzene, 1,3-butadiene, and toluene. The three TAPs are all found in the exhaust of internal combustion engines of most automobiles and aircraft. Data for these three chemicals are useful in comparing the possible impacts of LAX operations with the impacts from unrelated sources of the same pollutants. The concentrations of benzene, 1,3-butadiene, and toluene measured at the two monitoring stations are similar. These stations can, at a screening level, be considered representative of ambient urban background levels for the South Coast Air Basin. Background concentrations are used in a preliminary determination of an area of impact around LAX within which LAX-related impacts will be assessed.

Table 12

Existing Annual Mean Toxic Air Pollutant Concentrations in Los Angeles Region

	Los Angeles (µg/m^3)			Long Beach (µg/m^3)		
Toxic Air Pollutant	**1994**	**1995**	**1996**	**1994**	**1995**	**1996**
Benzene	2.00	1.83	1.48	1.63	1.33	Not Available
1,3-Butadiene	0.525	0.536	0.471	0.385	0.385	Not Available
Toluene	6.18	5.98	4.44	4.04	3.29	Not Available

Source: CALEPA California Air Resources Board, Toxics Air Quality Data, Statewide Summaries, Online Database, http\:www.arb.ca.gov/homepage.htm, May, 2000.

4.1.3 Exposure Scenarios

Exposure scenarios are representations of potential conditions under which receptors within the LAX zone of impact may come into contact with LAX TAPs of concern. Receptors for which exposure scenarios are prepared are selected to provide the most conservative, and therefore, protective, values for health impact

assessment. By providing estimates for the most exposed individuals, it can be assumed that the general population would be protected.

Exposure scenarios include receptors and the various pathways by which they might be exposed to TAPs of concern. A complete exposure pathway consists of four parts:

- A TAP source (e.g., jet or automobile fuel combustion)
- A release mechanism (e.g., jet or automobile engine exhaust)
- A means of transport from point of release to point of exposure (e.g., local winds)
- A route of exposure (e.g., inhalation)

If any of these elements of an exposure pathway is absent, no exposure can take place and the pathway is considered incomplete and is not evaluated. An example of an incomplete pathway involves the contact with volatile chemicals in soil. Highly volatile TAPs do not efficiently deposit into soil, and quickly volatilize should they be deposited from the air. The pathway is incomplete because efficient transport of chemical from air to soil will occur. Exposure scenarios can be represented through the development of a site conceptual exposure model (SCEM), described in the following section.

4.1.4 Site Conceptual Exposure Model

A site conceptual exposure model is provided as the basis for identifying and evaluating pathways by which human receptors may be exposed to TAP emissions from LAX (**Figure B2**, Conceptual Exposure Model for LAX Master Plan Toxic Air Pollution Exposure Assessment). The objective of the model is to facilitate analysis of exposure routes and receptors, and to focus the assessment on those pathways and sources that drive potential impacts on human health risk.

The model presented on **Figure B2**, Conceptual Exposure Model for LAX Master Plan Toxic Air Pollution Exposure Assessment, provides a consistent roadmap to the evaluation of risk to human health implied by LAX operations. The model traces exposure pathways from sources through release mechanisms and exposure routes to affected receptors and shows which exposure pathways will be quantitatively evaluated for the final risk assessment.

Numerous potentially complete exposure pathways exist for receptors at or near LAX. On the right-hand side of **Figure B2**, Conceptual Exposure Model for LAX Master Plan Toxic Air Pollution Exposure Assessment, various combinations of exposure pathways and exposure media are tabulated. Each pathway and medium are evaluated for completeness and relative significance for each receptor. Pathways identified as being complete and significant are denoted with an "✓." These pathways will be quantitatively evaluated in the final HHRA. Pathways not expected to result in significant exposures for a given receptor are noted, along with an explanation of their exclusion from the quantitative risk analysis.

For clarity, exposure pathways for a number of possible receptors, such as visitors to local parks, recreational facilities, beaches, etc., are not included in the SCEM. Exposures to these receptors are likely, but would always be less than those for the receptors included in the model. Therefore, if LAX-related impacts were acceptable for the receptors presented in the SCEM, then impacts would also be acceptable for other receptors that receive less exposure. Other receptors are further discussed below in Section 4.2.5, *Other Potential Receptors*.

4.2 Evaluation of Potential Receptors and Exposure Pathways

The analysis of exposure pathways to be evaluated in the final HHRA is discussed in this section.

4.2.1 On-Airport Occupational Worker

Workers at LAX may represent the population for which exposures to TAPs may be greatest. LAX workers, especially baggage handlers at the gates and on the aprons, spend large amounts of their time at work in areas where exhaust from jet engines, GSE, and other sources may reach their highest concentrations (Section 2, *Selection of Preliminary TAPs of Concern*). These workers would also be active for long periods, typically 40 hours per week, in areas where elevated concentrations of TAPs are expected. If impacts to LAX workers at the gates and aprons fall within acceptable levels, so should impacts for any workers at LAX.

TAPs of concern for LAX operations include VOCs, carcinogenic PAHs, metals, and equivalents of TCDD (Section 3, *Screening Level Air Dispersion Modeling for Toxic Air Pollutants).* On-airport workers would inhale vapors and particulates emitted during normal LAX operations. VOCs would not efficiently deposit onto surfaces at LAX and would not be expected to exist in significant concentrations in dusts, soils or other particulates. Thus, workers would not be expected to be exposed to VOCs via pathways other than inhalation, such as incidental ingestion of dust/soil or dermal absorption of chemicals from dust/soil.

PAHs and metals would be released during LAX operations as fine particulates that could be inhaled by on-airport workers. Inhalation is therefore a potentially important exposure pathway for these TAPs. Because PAHs and metals would be expected to exist as particles, deposition on soils and as dust is possible. Exposure pathways involving direct contact with contaminated soil/dust are therefore theoretically complete for these TAPs. However, as discussed in Section 2, *Selection of Preliminary TAPs of Concern*, deposition of PAH and metals onto surfaces would be too small to result in measurable increases in urban background concentrations of these TAPs in soil. For TCDD, deposition rates onto soil would be very low compared to background deposition rates, and substantial impacts above background levels would not be expected. Thus, exposures via pathways associated with contaminated soils will not evaluated quantitatively in the final HHRA.

The on-airport worker scenario is defined as:

- Baggage handlers working near aircraft gate areas
- Workers exposed to VOCs via inhalation only
- Workers exposed to metals, PAHs, and TCDD via inhalation only

4.2.2 Off-Airport Elementary School Child

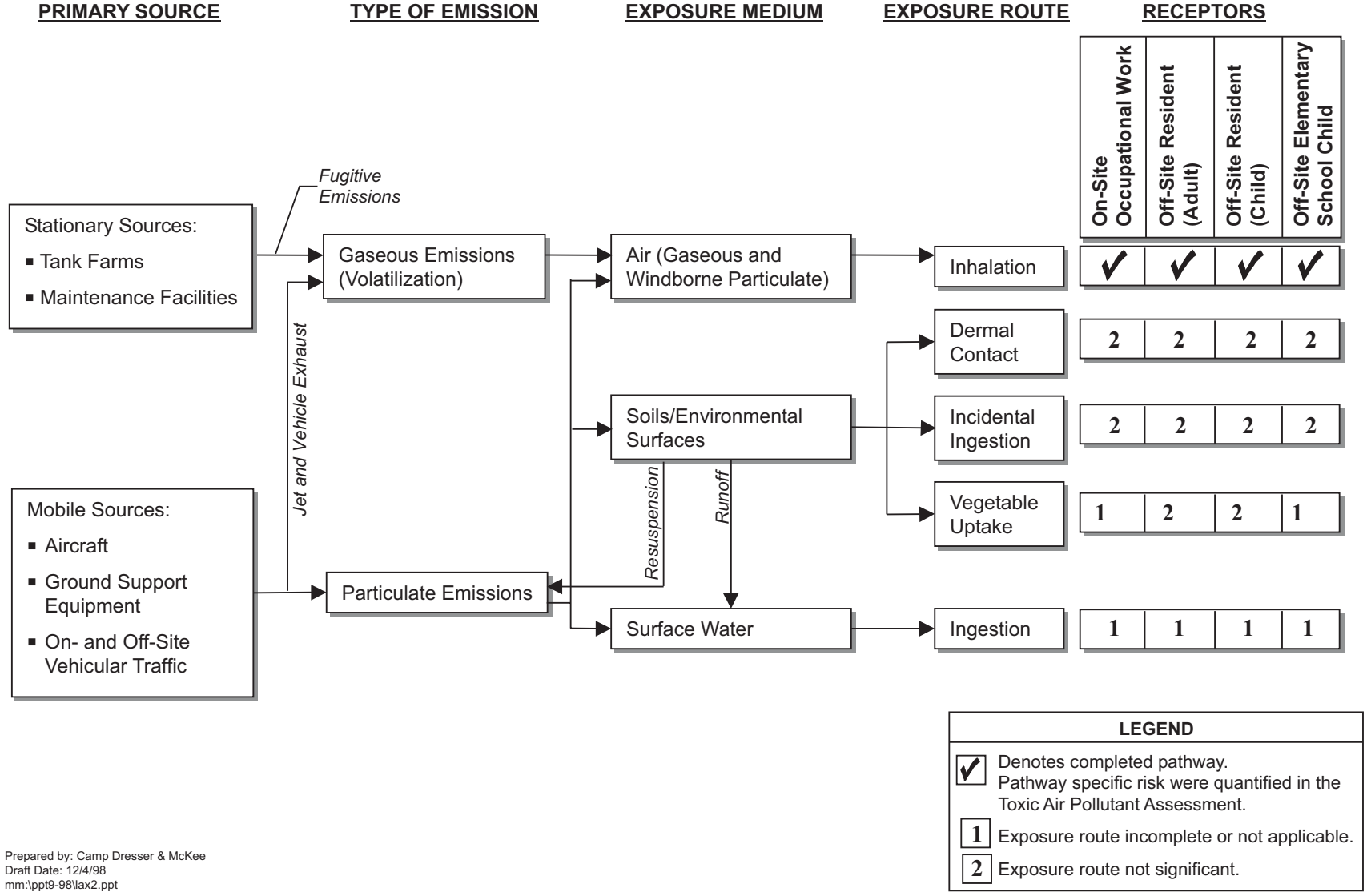
Children may attend school at locations close to LAX where impacts may be greater than those at their residences. Further, children may be more susceptible to air toxics because of relatively high inhalation rates and low body weights. These factors can combine to provide relatively high exposures expressed in terms of amount of material inhaled per kilogram of body weight. School children are therefore separately evaluated for potential impacts from LAX operations. Children ages 6 to 12 years old are considered an appropriate target population since this age range includes the youngest school ages, and it is sufficiently long for analysis of chronic exposures and risks.

TAPs of concern for LAX operations include VOCs, carcinogenic PAHs, metals, and TCDD (Section 2, *Selection of Preliminary TAPs of Concern).* Elementary school children may be exposed to VOCs released into the air via inhalation if the school is located in the direction of prevailing winds. Elementary school children attending an existing school at the location where downwind impacts are maximal are evaluated for exposure to VOCs via inhalation. For reasons discussed previously, VOCs are not expected to deposit effectively onto surfaces of playgrounds and are not, as a consequence, expected to exist in significant concentrations in dusts, soils, or other particulates. School children, therefore, would not be expected to be exposed to VOCs via pathways such as incidental ingestion of dust/soil and dermal absorption of chemicals from dust/soil that adheres to skin.

TCDD, PAHs, and metals would be released during LAX operations as fine particulates, which could be inhaled by school children. Inhalation is therefore a potentially important exposure pathway for these TAPs. This pathway will be evaluated quantitatively. However, as discussed in Section 2, *Selection of Preliminary TAPs of Concern,* deposition of PAHs and metals onto surfaces would be too small to result in measurable increases in urban background concentrations of these TAPs in soil. For TCDD, deposition rates onto soil would be very low compared to background deposition rates, and substantial impacts above background levels would not be expected. Thus, exposures via pathways associated with contaminated soils will not evaluated quantitatively in the final HHRA.

The elementary school child scenario is defined as:

- Elementary school children, ages 6 through 12 years old, who attend school at a location subject to the highest TAP concentrations
- School children exposed to VOCs via inhalation only
- School children exposed to metals, PAHs, and TCDD via inhalation only



Los Angeles International Airport Master Plan	Conceptual Exposure Model for LAX Master Plan - Toxic Air Exposure Assessment	Figure B2

4.2.3 Off-Airport Resident, Adult

Currently, residential areas are located immediately adjacent to LAX. Adult residents living at locations near the airport, especially in areas downwind, would be exposed to TAPs from LAX, possibly for long periods of time. Long periods of exposure are appropriate for evaluating carcinogenic risks, because exposures to carcinogens are averaged over an entire lifetime. Thus, the longer the period of exposure, the higher the potential risk. For quantitative assessment of cancer risks, adult residents are assumed to be exposed to TAPs from LAX from birth for 30 years.

TAPs of concern for LAX operations include VOCs, carcinogenic PAHs, metals, and TCDD (Section 2, *Selection of Preliminary TAPs of Concern*). Adult residents could be exposed to VOCs in air via inhalation in locations downwind from the airport. Therefore, an adult resident is assumed to live in current or projected future residential areas close to the airport, and in areas where the highest impacts from TAP emissions would be expected. This pathway will be evaluated quantitatively in the final HHRA. As previously discussed, VOCs would not be expected to deposit efficiently onto soils or other surfaces. Adult residents will not be evaluated for exposure to VOCs via direct contact with contaminated soils or dusts in the final HHRA.

TCDD, PAHs, and metals would be released during LAX operations as fine particulates, which could be inhaled by adult residents. Inhalation is therefore a potentially important exposure pathway for these TAPs. Because PAHs and metals would be expected to exist as particles, deposition on soils and as dust is also possible. Exposure pathways involving direct contact with contaminated soil/dust are therefore theoretically complete for these TAPs. However, as discussed in Section 2*, Selection of Preliminary TAPs of Concern,* deposition of PAHs and metals onto surfaces would be too small to result in measurable increases in urban background concentrations for these TAPs. For TCDD, deposition rates onto soil would be very low compared to background deposition rates, and substantial impacts above background levels would not be expected. Thus, exposures via pathways associated with contaminated soils will not be included in the quantitative exposure estimates in the final HHRA.

The adult resident scenario is defined as:

- Adult residents living in current or potential future residential locations determined to be subject to the highest TAP concentrations
- Adult residents exposed to VOCs via inhalation only
- Adult residents exposed to metals, PAHs, and TCDD via inhalation only

4.2.4 Off-Airport Resident, Young Child

Currently, residential areas are located immediately adjacent to LAX. Child residents living at locations near the airport, especially in areas downwind, could be exposed to TAPs from LAX, possibly for long periods of time. Children are separately evaluated because non-cancer impacts are evaluated on the basis of exceeding a threshold of exposure. Exposures for children are likely to be higher than those for adults because child body weights are lower and chemical intakes rates relatively high. Thus, the most conservative analysis for non-cancer risks is evaluation of young children for the shortest duration that can be termed chronic. For this assessment, children aged 0 to 6 years old living with adult residents were evaluated for non-cancer effects.

TAPs of concern for LAX operations include VOCs, carcinogenic PAHs, metals, and TCDD equivalents (Section 2, *Selection of Preliminary TAPs of Concern*). Child residents could be exposed to VOCs released into the air via inhalation in the same manner as adult residents. This pathway will be quantitatively evaluated in the final HHRA. VOCs would not be expected to deposit effectively onto residential surfaces and, therefore, would not be expected to exist in significant concentrations in dusts, soils, or other particulates. Thus, child residents would not be expected to be exposed to VOCs via pathways such as incidental ingestion of dust/soil and dermal absorption of chemicals from dust/soil that adheres to skin.

PAHs and metals would be released during LAX operations as fine particulates that could be inhaled by child residents. Inhalation is therefore a potentially important exposure pathway for these TAPs. This pathway will be quantitatively evaluated in the final HHRA.

Because PAHs and metals would be expected to exist as particles, deposition on soils and as dust would also be possible. Exposure pathways involving direct contact with contaminated soil/dust would therefore theoretically complete for these TAPs. However, as discussed in Section 2*, Selection of Preliminary TAPs*

of Concern, deposition of PAHs and metals onto surfaces would likely be too small to result in toxicologically significant impact. For TCDD, deposition rates onto soil would be very low compared to background deposition rates, and substantial impacts above background levels would not be expected. Thus, exposures via pathways associated with contaminated soils, including ingestion of or dermal exposure to TAPs present in dust or soils or which have been taken up into home-grown vegetables will not be included in the quantitative risk estimates in the final HHRA.

The child resident scenario is defined as:

- Child residents, ages 0 through 6 years old, living at the same locations as the adult resident
- Child residents exposed to VOCs via inhalation only
- Child residents exposed to metals, PAHs, and TCDD via inhalation only

4.2.5 Other Potential Receptors

Several other receptors could be exposed to emissions from LAX. These include visitors to parks and other recreational areas, visitors to sport and cultural event centers near the airport, commercial and industrial workers at facilities near LAX, and workers involved in construction projects near LAX. In all cases, exposures for these receptors would be less than those for residents living adjacent to LAX.

Visitors to recreational, sports and/or cultural facilities near the airport could be exposed to relatively high concentrations of TAPs when facilities are located downwind and close to the airport boundaries. However, residents will be assumed in the final HHRA to live adjacent to the airport in locations where maximum impacts from LAX emissions are expected. Visitors would be exposed much less frequently than people living in these locations. Thus, residents in areas of highest impacts represent a "worst case" for evaluating toxic air pollutant emissions from LAX. If potential impacts were determined to be less than significant for residents, they would also be less than significant for visitors to the LAX environs.

Similar arguments hold for workers near LAX. Although some of these workers could spend significant amounts of time close to LAX in areas of impact, exposure frequency and duration for residents would always be greater than those for workers. Again, residential land uses are located adjacent to airport boundaries where impacts from LAX emissions are expected to be greatest. Thus, if impacts for residents were determined to be less than significant, impacts to workers would also be less than significant.

4.3 Exposure Assessment Methods

For the LAX HHRA, four specific receptors were selected for quantitative evaluation. Each receptor represents a unique population and set of exposure conditions. As a whole, they cover a range of exposure scenarios for the potentially most affected human receptors (**Table 13**, Scenarios Evaluated in the HHRA).

Table 13

Scenarios Evaluated in the HHRA

Receptor	Pathways
On-Airport Worker	Inhalation of TAPs
Off-Airport Adult Resident	Inhalation of TAPs
Off-Airport Child Resident	Inhalation of TAPs
Off-Airport School Child	Inhalation of TAPs

Source: Camp Dresser & McKee Inc., 1998.

4.3.1 Evaluation of Toxic Air Pollutant Exposure for the On-Airport Occupational Worker

The on-airport occupational worker was assumed to be in contact with TAPs related to LAX operations as occupational exposure during business hours. Because this is occupational exposure and not incidental exposure related to residence or recreational status, it is appropriate to assess the extent of potential

toxicological impacts to the worker through comparison of the maximum average air concentrations of TAPs (conservative predictor of exposure) to thresholds of significance determined for workers by relevant governing bodies. Permissible Exposure Limits – Time-Weighted Average (PEL-TWA) are air concentrations for chemicals designed to represent maximum concentrations to which workers may be repeatedly exposed during business hours (8-hour time-weighted average) without developing adverse health effects.[48]

Occupational exposures are thus assessed by comparing maximum 8-hour concentrations of TAPs near gates and aprons, estimated through air dispersion modeling, with PEL-TWAs. Under ACGIH guidelines, if TAP concentrations are below PEL-TWAs, health impacts are unlikely for LAX worker. Eight-hour PEL-TWAs for most TAPs of concern for LAX are presented in **Table 14**, Permissible Exposure Limits (Time-Weighted Average) for TAPs for an On-Airport Occupational Worker.

Table 14

Permissible Exposure Limits (Time-Weighted Average) for TAPs for an On-Airport Occupational Worker

TAP	PEL-TWA (mg/m^3) [1]
Acetaldehyde	$1.8x10^{2}$
Acrolein	$2.5x10^{-1}$
Benzene [2]	$3.2x10^{-1}$
Butadiene, 1,3	$2.2x10^{0}$
Formaledehyde [2]	$3.7x10^{-1}$
Naphthalene	$5.0x10^{1}$
Toluene	$1.88x10^{2}$
Xylenes	$4.34x10^{2}$
Arsenic	$1.0x10^{-2}$
Beryllium	$2.0x10^{-3}$
Cadmium	$5.0x10^{-3}$
Chromium (as Cr(VI))	$5.0x10^{-2}$
Copper	$1.0x10^{0}$
Hexane	$1.76x10^{2}$
Lead	$1.5x10^{-1}$
Maganese	$5.0x10^{0}$
Nickel	$1.0x10^{-1}$
Selenium	$2.0x10^{-1}$
Zinc[3]	$1.0x10^{1}$

[1] CalOSHA. 2000. Table AC-1. Permissible Exposure Limits for Chemical Contaminants. http://www.dir.ca.gov/title8/5155a.htm.

[2] PEL unavailable; value is from American Conference of Governmental Industrial Hygienists (ACGIH), Documentation of the Threshold Limit Values and Biological Exposure Indices, 8th ed., Cincinnati, Ohio, 1998.

[3] PEL-TWA for zinc oxide dust.

4.3.2 Evaluation of TAP Exposure for the Off-Airport Elementary School Student and for Residential Receptors

This section defines the methods used for quantifying TAP exposure for potential human receptors for the three off-airport scenarios previously defined. The methodology is based on CalEPA and USEPA guidelines and will provide conservative estimates of exposure.

Exposure is defined for the LAX HHRA as contact of a person with a TAP. If exposure to a TAP occurs through inhalation, dermal contact, or ingestion, some of the TAP would be absorbed by the body. The amount of TAP inhaled or ingested is referred to as the contaminant intake. The fraction of the contaminant absorbed into the body (that which crosses membranes of the gastrointestinal or respiratory

[48] CalOSHA (California Occupational Safety and Health Agency) 2000 California Code of Regulation, Title 8, Section 5155. Airborne Contaminants.

system following ingestion or inhalation, or skin after dermal contact) is referred to as the dose or the absorbed dose of contaminant. Toxicity values (RfDs) and cancer potency or slope factors (CSFs) are generally developed in terms of contaminant intake, rather than absorbed doses. Thus, all exposure estimates for this HHRA will be calculated as TAP intakes.

4.3.2.1 Calculation of Chronic Daily Intakes (CDI)

To estimate potential cancer risks and the potential for adverse non-cancer health hazards, TAP intakes for each pathway for each receptor must be estimated. For both cancer and non-cancer risk assessment, average long-term daily intakes are used to estimate risk and hazards. Chronic daily intake (CDI) for TAPs is estimated as follows:[49]

$$CDI=(C \times IR \times EF \times ED) / (BW \quad AT)$$

Where:

CDI	=	chronic daily intake (mg/kg body weight/day)
C	=	chemical concentration in exposure medium (mg/kg)
IR	=	inhalation rate with exposure medium (mg/day)
EF	=	exposure frequency and duration (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	average time; e.g., the period over which exposure is averaged (days)

Averaging time for estimation of cancer risk is 70 years or 25,550 days. Cancer risk is evaluated as the lifetime average daily dose (LADD) according to CalEPA and USEPA guidance. Averaging time for estimation of non-cancer hazards is the duration of exposure, expressed in days. Non-cancer hazards are evaluated as average daily dose (ADD) over the period of exposure, again, following CalEPA and USEPA guidance.

4.3.2.2 Estimation of TAP Concentrations in Air

Evaluation of proposed the three build alternatives is prospective and no actual data for expected conditions will be available for the HHRA. Thus, all estimates for exposure concentrations are based on air dispersion/deposition modeling.

Methods used to estimate the quantities of chemicals released during LAX operations for different LAX configurations are discussed in detail in Section 2, *Selection of Preliminary TAPs of Concern*. Exposure concentrations in air will be estimated from these emission estimates using maximum annual average concentrations using meteorological data from LAX derived from ISC3 modeling (Section 3*, Screening Level Air Dispersion Modeling for Toxic Air Pollutants*). Exposure concentrations will be calculated for a modeling grid that becomes finer with decreasing distance from sources. In addition, air concentrations will be modeled for locations that will represent maximally exposed receptors for the four scenarios defined above.

4.3.2.3 Exposure Parameters

Exposure pathways used to assess receptor contaminant intakes are summarized in **Table 13**, Scenarios Evaluated in the HHRA. Exposure parameters used to calculate LADD and ADD for each of these pathways are summarized in **Table 15**, Parameters Used to Estimate Exposures to TAPs of Concern. Exposure parameters are based on the CalEPA Supplemental Guidance for Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities,[50] USEPA Exposure Factors Handbook,[51] and CAPCOA Air Toxics Assessment Manual.[52]

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[49] USEPA, Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual Interim Final, December 1989.

[50] CalEPA, Supplemental Guidance for Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities, 1993.

[51] USEPA, Exposure Factors Handbook, 1997.

[52] CAPCOA, Air Toxics Assessment Manual, 1993.

4.4 Areas of Potential Impact for LAX Emissions

Emissions from LAX would be carried primarily to the east by prevailing winds. As TAPs move downwind, they would be diluted, photodegraded, and/or deposited so that air concentrations would eventually be reduced to negligible levels. One goal of this screening analysis was to determine how large an area around LAX would need to be included in the air dispersion modeling to ensure that all potentially significant impacts would be included in the HHRA. To define this study area, screening level modeling was performed using ISC3 (Section 3, *Screening Level Air Dispersion Modeling for Toxic Air Pollutants*). This modeling was very limited, and incorporated a number of conservative, simplifying assumptions. Thus, calculated air concentrations significantly overestimate potential impacts of LAX emissions. However, because the modeling was conservative, the results can be used to define a study area around LAX.

Table 15

Parameters Used to Estimate Exposures to TAPs of Concern

Exposure Pathway	Off-Airport Receptors		
Inhalation of Particulates and Gases	Off-Site Resident		Off-Site Elementary School Child
	Adult	Child	
IR (m^3/d)	20	15	6
EF (d/yr)	350	350	200
ED (yr)	30	6	6
BW (kg)	70	15	40
AT Non-cancer (days)	10,950	2,190	2,190
AT Cancer (days)	25,550	25,550	25,550

Acronyms used in this table:
IR =Average inhalation rate
EF =Exposure frequency
ED =Exposure duration
BW =Body weight
AT =Averaging time

Source: Camp Dresser & McKee Inc., 2000.

To identify a study area, urban background concentrations of 1,3-butadiene, benzene, and toluene were estimated from recent data (**Table 12**, Existing Annual Mean Toxic Air Pollutant Concentrations in Los Angeles Region). These concentrations (1, 4, and 14 µg/m^3, respectively) were divided by two, and the resulting concentrations (0.5, 2, and 7 µg/m^3) used as target values in analyzing air dispersion modeling results, as described below.

ISC3 modeling was performed only for emissions for the No Action/No Project Alternative. The modeling grid for the ISC3 runs focused on current LAX property and the fine grid did not extend into off-airport areas. As a preliminary evaluation of a study area, annual average concentrations for each of the points of the fine grid on LAX property were used to generate isopleths for 1,3-butadiene for a concentration of 0.5 µg/m^3 (**Figure B3**, 1,3-Butadiene Isopleth at One Half the Background Level) for benzene for a concentration of 2 µg/m^3 (**Figure B4**, Benzene Isopleth at One Half the Background Level), and for toluene for a concentration of 7 µg/m^3 (**Figure B5**, Toluene Isopleth at One-Half the Background Level). In all three cases, the isopleths, representing a concentration of half of the expected urban background, stay within the LAX boundary.

The results of the preliminary evaluation suggest that concentrations of TAPs from LAX would merge with background relatively quickly in areas outside the LAX boundary. Since the ISC3 modeling runs exaggerate possible impacts on air quality, the study area for the HHRA could conceivably be very small. However, screening level air dispersion modeling was not performed for any of the LAX Master Plan alternatives. Since the LAX Master Plan would result in greatly increase air traffic, impacts could extend further than those implied by the screening level modeling.

Also apparent in **Figures B3** and **B4** is the east-west orientation of the isopleths. This result reflects the prevailing winds, mostly from the west during normal airport operations. Any impacts from LAX operations would be expected east of the airport, rather than to either the north or south.

Given the screening nature of the ISC3 modeling, the lack of air dispersion modeling for impacts for any of the three build alternatives, and the potential for impacts mainly to the east of the airport, a study area should be defined conservatively. However, the area should not be so large that modeling and assessment efforts are spent inefficiently for areas where impacts are negligible. A reasonable compromise would suggest that the study area should include an elliptical area that extends 2 kilometers (1.25 miles) north and south, and 4 kilometers (2.5 miles) east of the current LAX fence line for the No Action/No Project Alternative. This study area, based on current LAX configuration, is included in **Figures B3** through **B5**. Note that the LAX fence line would change somewhat both under the No Action/No Project Alternative and the three build alternatives.

4.5 Summary

The preliminary exposure assessment has five goals:

- To define the exposure setting on and near LAX for both current and future land uses
- To evaluate potential exposure pathways and receptors for TAPs released from LAX
- To develop representative exposure scenarios based on potentially important exposure pathways and on receptors for which impacts are potentially greatest
- To establish appropriate exposure parameters for quantitative evaluation of cancer and non-cancer risk for each representative exposure scenario
- To define an appropriate study area within which exposures will be quantified

Results of the preliminary assessment indicated that:

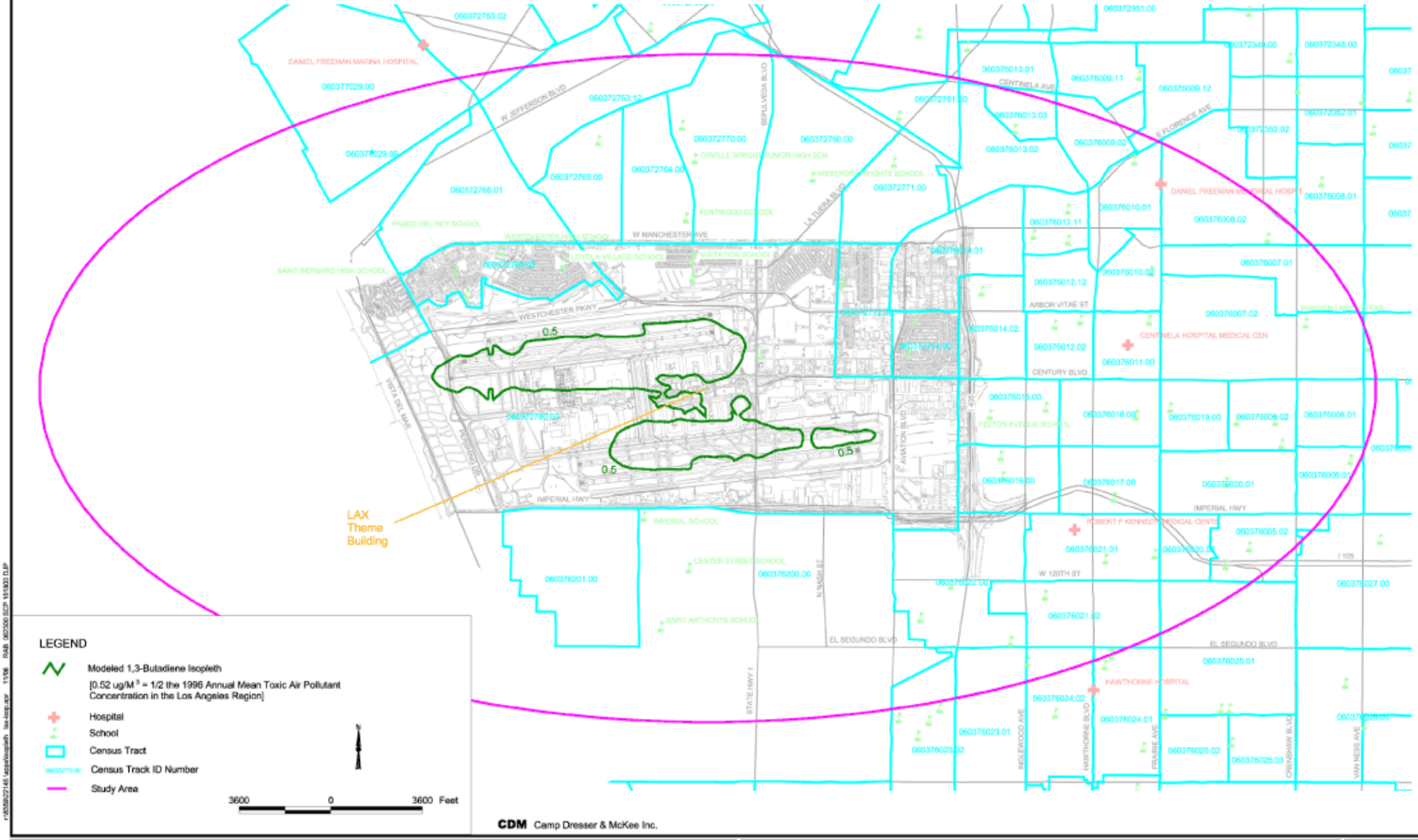
- Exposures on and near LAX for both the No Action/No Project Alternative and for the three build alternatives would occur in areas of mixed residential, commercial, industrial, and recreational uses.
- Significant exposures would be likely only for inhalation of TAPs released during LAX operations; deposition of TAPs onto soil and other surfaces would be too low to have any significant impact.
- Representative scenarios for the HHRA are
  - On-airport worker (baggage handler)
  - Off-airport elementary school child
  - Off-airport adult resident
  - Off-airport child resident
- For LAX workers, exposures and risks will be evaluated through comparison of potential air concentrations of TAPs (maximum 8-hour averages) with TLV-TWAs established by ACGIH.
- For other scenarios, exposure parameters and calculations of chronic daily intakes will be based on CalEPA and USEPA guidance.
- The study area for the HHRA should extend at least 2 kilometers north and south and 4 kilometers east of the current LAX fence line for the No Action/No Project Alternative and for the three build alternatives.

The final HHRA will use the above findings and conclusions to assess the potential health impacts associated with implementation of the LAX Master Plan as part of the EIS/EIR for the project.

5. TOXICITY ASSESSMENT

The toxicity assessment evaluates potential human health effects from exposure to TAPs related to aircraft and airport operations at LAX. Potential adverse effects from exposure to such pollutants include both carcinogenic and non-carcinogenic health effects.

The primary sources of toxicity information used in this assessment include CalEPA Office of Environmental Health Hazard Assessment (OEHHA) Cancer Potency Factors, USEPA's Integrated Risk Information System (IRIS), Health Effects Assessment Summary Tables (HEAST), Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles, and USEPA criteria documents.



Los Angeles International Airport Master Plan

1,3-Butadiene Isopleth at One Half the Background Level

Figure
B3

LAX
Theme
Building

LEGEND

- Modeled Benzene Isopleth
  [2.37 ug/M ^{3} = 1/2 the 1996 Annual Mean Toxic Air Pollutant Concentration in the Los Angeles Region]
- Hospital
- School
- Census Tract
- Census Track ID Number
- Study Area

3600 0 3600 Feet

CDM Camp Dresser & McKee Inc.

Los Angeles International Airport Master Plan	**Benzene Isopleth at One Half the Background Level**	Figure B4

LAX
Theme
Building

LEGEND

- Modeled Toluene Isopleth
  [7.47 ug/M^3 = 1/2 the 1996 Annual Mean Toxic Air Pollutant Concentration in the Los Angeles Region]
- Hospital
- School
- Census Tract
- Census Track ID Number
- Study Area

3600 0 3600 Feet

N

CDM Camp Dresser & McKee Inc.

Los Angeles International Airport Master Plan	**Toluene Isopleth at One Half the Background Level**	Figure B5

This section explains how toxicity criteria for carcinogens and non-carcinogens are developed and expressed, and summarizes toxicity values for each TAP of concern. Individual chemical profiles in support of toxicity values are presented in Attachment C, *Toxicological Profiles*. These profiles describe important toxicokinetic findings (absorption into, distribution in, metabolism by, and excretion from the body), outline major adverse effects, discuss uncertainties and important data gaps, and summarize important studies used in the derivation of toxicity values. Sections 5.1, *Carcinogens,* and 5.2*, Non-carcinogens*, present the basis for the development of toxicity values for carcinogens and non-carcinogens, respectively. Section 5.3, *Uncertainties Associated with Toxicity Assessment*, discusses uncertainties associated with the toxicity assessment.

5.1 Carcinogens

5.1.1 Evidence of Carcinogenicity

USEPA has developed a classification system for carcinogens which characterizes the overall weight of evidence of carcinogenicity based on the availability of human, animal, and other supportive data. Three major factors are considered:

- The quality of evidence from human studies
- The quality of evidence from animal studies
- Other supportive data that are assessed to determine whether the overall weight of evidence should be modified

The USEPA classification system for the characterization of the overall weight of carcinogenicity has the following five categories:

- Group A - Human Carcinogen. This category indicates that there is sufficient evidence from epidemiological studies to support a causal association between an agent and cancer.
- Group B - Probable Human Carcinogen. This category generally indicates that there is at least limited evidence from epidemiological studies of carcinogenicity to humans (Group B1) or that, in the absence of adequate data on humans, there is sufficient evidence of carcinogenicity in animals (Group B2).
- Group C - Possible Human Carcinogen. This category indicates that there is limited evidence of carcinogenicity in animals in the absence of adequate data on humans.
- Group D - Not Classified. This category indicates that the evidence for carcinogenicity in animals is inadequate.
- Group E - Evidence of Non-carcinogenicity to Humans. This category indicates that there is evidence for non-carcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies.

5.1.2 Cancer Slope Factors

The USEPA Cancer Review and Validation Effort (CRAVE), formerly the Cancer Assessment Group (CAG), has used a variety of specialized models to estimate the upper bound risk of carcinogenesis for more than 50 compounds. Data from animal or epidemiological studies are used to determine slope factors, which are expressed as $(mg/kg\text{-}day)^{-1}$. The cancer slope factor (CSF) describes the increase in an individual's risk of developing cancer over a 70-year lifetime per unit of exposure where exposure is expressed as mg/kg-day.

CSFs are calculated using methods intended to be protective of human health and are based on the assumption that cancer risks decrease linearly with decreasing dose. The 95 percent upper confidence limit estimate for the slope is used in most cases to compensate for animal to human extrapolation and other uncertainties. The resulting CSFs are considered to be upper range estimates that are unlikely to underestimate carcinogenic potential in humans.

When the upper-bound CSF is multiplied by the lifetime average daily dose of a potential carcinogen, the product is the upper-bound lifetime individual cancer risk associated with exposure at that dose. The calculated risk is thus an estimate of the increased likelihood of cancer resulting from exposure to a chemical. For example, if the product of the CSF and the average daily dose is 1×10^{-6}, the predicted upper-bound cancer risk for the exposed population is one in one million, or 0.0001 percent. This risk is in addition to any "background" risk of cancer not related to the chemical exposure.

It should be noted that calculation of risk relies on data derived from human epidemiological studies or chronic animal bioassays. The likelihood that a chemical is a human carcinogen is a function of the following factors:

- The number of tissues affected by the chemical
- The number of animal species, strains, sexes, and number of experiments and doses showing a carcinogenic response
- The occurrence of clear-cut dose-response relationships as well as a high level of statistical significance of the increased tumor incidence in treated compared to control groups
- A dose-related decrease in time-to-tumor occurrence or time-to-death with tumor
- A dose-related increase in the proportion of tumors that are malignant

Animal studies are usually conducted using relatively high doses in order to observe adverse effects. Because humans are expected to be exposed at lower doses, data are adjusted by using a mathematical model. Data from animal studies are fitted to a linearized multi-stage model and a dose-response curve is obtained. The low-dose slope of the dose-response curve is subjected to various adjustments (e.g., calculation of 95 percent upper confidence limit), and inter-species scaling factors are often applied to derive slope factors for humans. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an individual basis. These models provide conservative but plausible estimates of upper limits on lifetime risk. Although the actual risk is unlikely to be higher than the estimated risk, it could be considerably lower and may even be zero.

CSFs for carcinogenic TAPs of concern for the LAX Master Plan are listed in **Table 16**, Cancer Slope Factors. Data used to develop CSFs for TAPs of concern are summarized in toxicity profiles provided in Attachment C, *Toxicological Profiles*.

5.2 Non-carcinogens

RfDs are toxicity values developed by USEPA for chemicals exhibiting non-carcinogenic effects. CalEPA has not separately developed RfDs. RfDs are usually derived from no-observable-adverse-effect levels (NOAELs) taken either from human studies, often involving workplace exposures, or from animal studies, and are adjusted downward using uncertainty or modifying factors. Uncertainty factors are generally applied to adjust for the possibility that humans are more sensitive than experimental animals and that there may be sensitive subpopulations of humans (e.g., children, pregnant women, individuals with hay fever or asthma). Depending upon the information available, other modifying factors may also be applied. For example, a modifying factor of 2 to 10 may be applied if the database on a particular chemical lacks information on reproductive or developmental toxicity.

The RfD is intended as an estimate of the daily exposure to a chemical that would not cause adverse effects even if the exposure occurs continuously over a lifetime. RfDs are presented in units of mg/kg-day for comparison with estimated chronic daily intake into the body. Intakes that are less than the RfD are not likely to cause adverse health effects. Chronic daily intakes that are greater than the RfD indicate a possibility for adverse effects. Whether such exposures actually produce adverse effects, however, is a function of a number of factors such as accuracy of uncertainty factors applied to the NOAEL, appropriateness of animal models used in studies extrapolated to humans, and potential for the chemical to cause effects in organs or systems (e.g., reproductive and immune systems) that have not been adequately studied. It is generally accepted, however, that protective assumptions made by USEPA in deriving RfDs will, in most cases, mean that exposures slightly in excess of the RfD will be associated with a low risk for adverse effects, with the probability of adverse effects increasing with increasing exposure.

RfDs for oral exposure have not been developed by CalEPA; therefore, for purposes of this analysis, all oral RfDs are taken from USEPA sources. Reference doses for inhalation exposure are available from USEPA sources for many TAPs of concern. RfDs for inhalation are generally calculated from Reference Concentrations (RfC). RfCs are derived in a fashion analogous to that for oral RfDs, but are expressed in units of mg/m^3. RfCs are intended as a estimate of the ambient air concentration for a chemical that could be present for a lifetime without causing adverse effects. An RfC is converted to an inhalation RfD by multiplying by inhalation rate and dividing by body weight. Standard CalEPA and USEPA parameters, 20 m^3/day for inhalation rate and 70 kilograms for body weight, were used for these calculations.

Reference Exposure Levels (RELs) have been proposed by CalEPA. RELs are analogous to RfCs, and could be used in risk assessment for inhalation exposure. However, current RELs are in review and are

subject to change. RELs should not, therefore, be the sole source of inhalation toxicity criteria used in the preliminary assessment.

As explained in Section 2, *Selection of Preliminary TAPs of Concern*, toxicity screening for selection for TAPs of concern (Step 5) was performed both with and without proposed RELs. This approach assures that TAPs of concern include all chemicals that might pass the toxicity screening criteria in the near future. Use of RELs for calculation of quantitative risk estimates in the final HHRA is a more difficult issue because the criteria are still under review and many values are likely to change. Use of RELs in the final HHRA will be predicated on recommendations of CalEPA scientists, professional judgement, and the magnitude of possible risks.

RfDs for COPCs for the LAX Master Plan are presented in **Table 17**, Toxicity Criteria for Systemic Toxicants.

Table 16

Cancer Slope Factors

Chemical	Oral Cancer Slope Factor [(mg/kg/day)][1]	Inhalation Cancer Slope Factor [(mg/kg/day)$^{-1}$]	Tumor Site		Cancer Classification
			Oral	Inhalation	
Organics					
Acetaldehyde	NA	0.00945	NA	Nasal, Larynx	B2
Acrolein	NA	NA	NA	NA	C
Benzene	0.029	0.102	NA	Blood	A
1,3-Butadiene	NA	0.595	NA	Reproductive Sys., Blood, Lung, GI	B2
Formaldehyde	NA	0.021	NA	Respiratory	B1
TCDD Equivalents	156,000	133,000	GI, Immune System, Reproductive System, Kidney	GI, Immune System, Reproductive System, Kidney	A
Polycyclic Aromatic Hydrocarbons (PAHs)					
Benzo(a) anthracene	1.2	0.39	NA	NA	B2
Benzo(b) fluoranthene	1.2	0.39	NA	NA	B2
Benzo(k) fluoranthene	1.2	0.39	NA	NA	B2
Benzo(a) pyrene	12	3.9	GI	GI, Respiratory	B2
Chrysene	0.12	0.039	NA	NA	B2
Dibenz(a,h) anthracene	4.1	4.2	Respiratory	NA	B2
Indeno(1,2,3-cd) pyrene	1.2	0.39	NA	NA	B2
Inorganics					
Arsenic	1.5[1]	11.6	Skin, Lung	Respiratory System	A
Beryllium	4.3[1]	7.0[3]	NA	Lung	B1
Cadmium	NA	14.7	NA	Respiratory System	B1
Chromium (Total)	NA	NA	NA	NA	NA
Chromium (VI)	42	525	NA	Lung	A
Lead (Inorganic)	NA	NA	NA	NA	B2
Manganese	NA	NA	NA	NA	D
Nickel	NA	0.91[2]	NA	Respiratory System	A[2]

Notes: NA - No data available
GI - Gastrointestinal System
CNS - Central Nervous System
All Toxicity Criteria from CalEPA Office of Environmental and Human Health Assessment, Cancer Potency Factors, 1994, except as noted.

1 USEPA, Integrated Risk Information System (IRIS) Online Database, 1998.
2 For nickel in refinery dust
3 Beryllium oxide value

Cancer Classification
Group A – Human Carcinogen
Group B (B1 and B2) – Probable Human Carcinogen
Group C – Possible Human Carcinogen
Group D – Not classified

Source: Camp Dresser & McKee Inc., 1998.

Table 17

Toxicity Criteria for Systemic Toxicants

TAP of Concern	USEPA Chronic Oral RfD (mg/kg-day)[1]	USEPA Chronic Inhalation RfD (mg/kg-day)[1]	CalEPA Chronic Inhalation RfD (mg/kg-day)	Target Organ		Uncertainty Factor		
				Oral	Inhalation	Oral	Inhalation (USEPA RfD)	Inhalation (CalEPA RfD)
Organics								
Butadiene, 1,3	NA	NA	$2.3x10^{-3}$	NA	Reproductive System	NA	NA	300
Acetaldehyde	NA	$2.57x10^{-3}$	NA	NA	Nasal	NA	1000	NA
Acrolein	$2x10^{-2}$	$5.71x10^{-6}$	NA	NA	Nasal	NA	1000	NA
Benzene	NA	$1.71x10^{-3}$(3)	$1.7x10^{-2}$	NA	NA	NA	NA	NA
Formaldehyde	$2x10^{-1}$	NA	$5.7x10^{-4}$	Body Weight	NA	100	NA	NA
Hexane	$6x10^{-2}$(2)	$5.7x10^{-2}$	NA	NA	CNS	10000	300	NA
Napthalene	$2x10^{-2}$	$8.57x10^{-4}$	$2.6x10^{-3}$	Body Weight	Resp. System, Blood	3000	3000	1,000
Toluene	$2x10^{-1}$	$1.1x10^{-1}$	NA	Liver, Kidney	CNS	1000	300	NA
Xylene	$2x10^{0}$	NA	$5.7x10^{-2}$	Body Weight	CNS, Resp.System	100	NA	100
Inorganics								
Arsenic	$3x10^{-4}$	NA	$8.57x10^{-6}$	Skin	NA	3	NA	3
Beryllium	$2x10^{-3}$	$5.7x10^{-6}$	$2.9x10^{-7}$	GI	Resp. System	300	10	300
Cadmium	$1x10^{-3}$ (Food)	$5.7x10^{-5}$(4)	$2.9x10^{-6}$	Kidney	Kidney, Resp. System	10	NA	NA
Chromium (VI)	$3x10^{-3}$	$2.86x10^{-5}$	$2.3x10^{-7}$	NA	NA	300	NA	300
Copper	$4x10^{-2}$(3)	NA	$5.7x10^{-6}$	GI	Resp. System	NA	NA	100
Lead (Inorganic)	NA	NA	NA	NA	NA	NA	NA	NA
Manganese	$1.4x10^{-1}$ (Food)	$1.4x10^{-5}$	NA	CNS	CNS	1	1000	NA
Nickel	$2x10^{-2}$	NA	$1.4x10^{-5}$	Body, Organ Weight	Lung	300	NA	30
Selenium	$5x10^{-3}$	NA	$2.3x10^{-5}$	NA	NA	3	NA	3000
Zinc	$3x10^{-1}$	NA	$2.6x10^{-4}$	Blood	NA	3	NA	100

NA – No data available
CNS - Central Nervous System
GI – Gastrointestinal System
Resp – Respiratory System

1 From USEPA, Integrated Risk Information System (IRIS) Online Database, 1998, unless otherwise noted
2 From Health Effects Assessment Tables (HEAST)
3 National Center for Environmental Assessment (NCEA) Regional Support Provisional Value
4 Withdrawn from IRIS

Source: Camp Dresser & McKee Inc, 1998.

5.3 Uncertainties Associated with Toxicity Assessment

5.3.1 General Uncertainties

A potentially large source of uncertainty is inherent in the derivation of the CalEPA and USEPA toxicity criteria (i.e., oral and inhalation RfDs, and cancer slope factors). In many cases, data must be extrapolated from animals to sensitive humans by the application of uncertainty factors to an estimated NOAEL or lowest-observed adverse effects level (LOAEL) for non-cancer effects. While designed to be protective, it is likely in many cases that uncertainty factors overestimate the magnitude of differences that may exist between human and animals, and among humans.

In some cases, however, toxicity criteria may be based on studies that did not detect the most sensitive adverse effects. For example, many past studies have not measured possible toxic effects on the immune system. Moreover, some chemicals may cause subtle effects not easily recognized in animal studies.

In addition, derivation of cancer slope factors often involves linear extrapolation of effects at high doses to potential effects a lower doses commonly seen in environmental exposure settings. Currently, it is not known whether linear extrapolation is appropriate. Probably, the shape of the dose-response curve for carcinogenesis varies with different chemicals and mechanisms of action. It is not possible at this time, however, to describe such differences in quantitative terms. In addressing uncertainties, USEPA recognizes that risks calculated at very low levels of exposure could be overestimated by the linear extrapolation process and may even be zero.

Finally, CalEPA proposed RELs are still under review and re-evaluation. Use of these criteria for the screening described in Section 2, *Selection of Preliminary TAPs of Concern*, is reasonable since (1) no quantitative risk estimates were derived, and (2) the design of the toxicity screening would allow RELs to include additional TAPs, but would not allow exclusion of TAPs. Uncertainties in proposed RELs are, however, too great to allow use in quantification or risks in the final HHRA without discussions with CalEPA scientists who derived the RELs, and additional evaluation of the toxicological data that support individual RELs.

5.3.2 Uncertainties for 1,3-Butadiene and Acrolein

Two TAPs of concern may dominate risk estimates for LAX emissions. 1,3-Butadiene and acrolein may be the "risk drivers" for cancer and non-cancer effects for the final HHRA (Section 2*, Selection of Preliminary TAPs of Concern*). That is, any mitigation that might be necessary based on releases of TAPs would probably have to be based on reducing 1,3-butadiene and acrolein impacts to acceptable levels. The toxicity of these TAPs should therefore receive additional consideration. Recently, a new health assessment for 1,3-butadiene has been published by EPA,[53] and currently, Toxicological Excellence in Risk Assessment (TERA) is conducting a toxicological review of acrolein. Both these recent reviews were evaluated, and new toxicological insights incorporated into toxicological profiles (Attachment C, *Toxicological Profiles*), and uncertainties discussions in the final HHRA.

6. SUMMARY AND CONCLUSIONS

LAWA has proposed expanding LAX in response to increasing demand for air and freight services in the area. As part of the EIS/EIR, a screening HHRA was conducted to provide a preliminary evaluation of potential impacts associated with proposed Master Plan alternatives. The results of the screening evaluation were used to focus the final HHRA on the most important exposure and risk issues that may be associated with emissions from LAX, for example, areas most likely to be affected, chemicals contributing most to overall impacts, and most sensitive human populations at and near LAX. The screening evaluation consisted of the following:

- Identification of TAPs of concern
- Screening level air dispersion and deposition modeling
- Preliminary exposure assessment

[53] USEPA, Health Assessment for 1,3-Butadiene, October 1998.

- Preliminary toxicity assessment

Results and conclusions from these analyses are summarized in the following sections.

6.1 Identification of TAPs of Concern

TAPs of concern were selected based on their potential presence in current and future emissions at LAX, identification as TAPs in federal and state regulations, estimated concentrations in LAX emissions, and toxicity. TAPs were selected for the No Action/No Project Alternative (baseline) and Alternative B. Results of the selection process include:

- Potential sources of TAPs at LAX include aircraft maintenance facilities, existing and planned tank farms, parking facilities, the Central Utilities Plant, aircraft, on-airport vehicles, off-airport vehicles, and ground support equipment.
- All but three TAPs identified in LAX emission sources are listed as TAPs in at least one of the following state or federal regulations: SCAQMD Rules 1401 and 1402, AB2588 and AB1807/2728, and/or the Clean Air Act (CAA).
- Comparison of emission estimates for different TAP sources indicated that aircraft emissions contribute most to overall emissions from LAX (over 90 percent for almost all TAPs). Implementation of the LAX Master Plan would result in increased number of aircraft and flights at LAX, and, therefore, increased aircraft emissions. Aircraft emissions are therefore also expected to dominate in the future. It is thus reasonable to focus further analyses in the HHRA on emissions from aircraft.
- Nine carcinogens were estimated to contribute individually more than 0.1 percent and together over 99.9 percent to total relative impacts from LAX. These chemicals, including acetaldehyde, arsenic, benzene, 1,3-butadiene, beryllium, cadmium, chromium, formaldehyde, and nickel were retained as TAPs of concern. PAHs (including benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)anthracene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene) were not eliminated in the toxicity screening step, even though they do not meet the screening criterion, since the public has expressed a special interest in impacts associated with this group of chemicals.
- For systemic toxicants, one chemical, acrolein, dominates potential impacts from LAX. Acrolein was estimated to contribute at least 95 percent to overall relative non-carcinogenic impacts from emissions at LAX. In addition to acrolein, the following chemicals were selected as TAPs of concern based on potential of systemic effects: arsenic, acetaldehyde, benzene, beryllium, 1,3-butadiene, cadmium, chromium, copper, formaldehyde, hexane, manganese, naphthalene, nickel, selenium, toluene, xylene, and zinc.
- Since standard toxicity criteria are not available for lead, toxicity screening for lead was not conducted. However, since releases of lead at LAX could theoretically be significant, lead was retained as a TAP of potential concern.
- Evaluation of potential impacts from deposition of TAPs released at LAX onto soil indicates that contributions from emissions would be negligible compared to background concentrations. Therefore, no assessment of secondary exposure pathways associated with contaminated soil is necessary to adequately evaluate total exposure and risks in the final HHRA.

Preliminary TAPs of concern for LAX are listed in **Table 18**, Preliminary TAPs of Concern for LAX.

Table 18

Preliminary TAPs of Concern for LAX

Substance	Carcinogen	Systemic Toxicant
Acetaldehyde	x	x
Acrolein		x
Arsenic	x	x
Benz(a)anthracene	x	
Benzene	x	x
Benzo(b)fluoranthene	x	
Benzo(k)fluoranthene	x	
Benzo(a)pyrene	x	
Beryllium[1]	x	x
1,3-Butadiene	x	x
Cadmium		x
Chromium (evaluated as hexavalent)	x	x
Chrysene	x	
Copper[1]		x
Dibenz(a,h)anthracene	x	
Formaldehyde	x	x
Hexane		x
Indeno(1,2,3-cd)pyrene	x	
Lead		x
Manganese		x
Naphthalene		x
Nickel	x	x
Selenium[1]		x
Toluene		x
Xylene[1]		x
Zinc[1]		x

[1] Selected only if proposed CalEPA RELs are adopted

Source: Camp Dresser McKee Inc., 2000.

6.2 Screening Level Air Dispersion Modeling

Screening air level dispersion modeling was conducted for the No Action/No Project Alternative (baseline) to assist in evaluation of soil deposition and to help identify an area of impact around LAX within which the analyses in the final HHRA will be focused. The modeling made several simplifying assumptions that result in overestimation of actual likely concentrations of TAPs from LAX emissions. These assumptions include: (1) plume rise for jet exhaust was not included in the model, (2) upper range estimates for time in queue were used, and (3) deposition to soil was estimated for on-airport locations where the highest annual average air concentrations were predicted.

Results indicate that:

- Air concentrations are predicted with an unrealistic model that is inappropriate for estimation of human health risks.
- Air concentrations predicted by screening modeling are overestimated and can be used to assess deposition of TAPs onto soil and to define areas of possible impact from airport operations.
- Areas of possible impact extend mainly east and west from LAX runways in the directions of prevailing winds. TAPs released during airport operations are not expected to be efficiently transported into communities north and south of the airport.

6.3 Preliminary Exposure Assessment

A preliminary exposure assessment was conducted to identify human populations that may be affected by emissions from LAX now or in the future and pathways through which these populations may be exposed. Identification of key exposure pathways for LAX emissions will result in a focused HHRA, with greater effort applied to the most critical exposure and risk issues.

The preliminary exposure assessment evaluated:

- The exposure setting for LAX and surrounding areas for both current and future land uses
- Potential exposure pathways and receptors for TAPs released from LAX
- Representative exposure scenarios
- Appropriate exposure parameters for quantitative evaluation of cancer and non-cancer risk for each representative exposure scenario
- Areas of potentially significant impact within which exposures will be quantified

Results of the preliminary assessment indicated that:

- Future land use near LAX will be similar to current land use and will consist of mixed residential, commercial, industrial, and recreational uses.
- The following populations are representative and will be evaluated quantitatively in the final HHRA:
  - On-airport worker (baggage handler)
  - Off-airport elementary school child
  - Off-airport adult resident
  - Off-airport child resident
- Significant exposures would be likely only for inhalation of TAPs released during LAX operations; this exposure pathway will be the focus for the HHRA. Pathways associated with deposition of TAPs onto soil (e.g., soil ingestion) would not be important, since deposition of TAPs onto soil would be negligible compared to background concentrations of TAPs in soil.
- For LAX workers, exposures and risks will be evaluated through comparison of potential air concentrations of TAPs (maximum 8-hour averages) with PEL-TWAs established by ACGIH.
- For residents and school children, exposure parameters and calculations of chronic daily intakes will be based on CalEPA and USEPA guidance.
- The area for which potential impacts will be evaluated during the HHRA should extend 2 kilometers north and south and 4 kilometers east of the LAX fence line for No Action/No Project Alternative and for the three build alternatives.

6.4 Preliminary Toxicity Assessment

Toxicity assessment evaluates potential human health effects from exposure to TAPs. Potential adverse effects from exposure to TAPs include both carcinogenic and non-carcinogenic health effects. In the preliminary toxicity assessment, derivation of toxicity criteria for carcinogens and non-carcinogens was explained and toxicity criteria were presented for all TAPs of concern. The toxicity assessment identified and resolved several issues of importance to the final HHRA.

- Both USEPA and CalEPA toxicity criteria were identified for many TAPs. CalEPA values were used in preference to USEPA values. This preference will be extended to the final HHRA. The only exception will be cases where appropriate CalEPA toxicity values are not available. Because of the importance of inhalation for the final HHRA, inhalation criteria will be used in preference to oral toxicity criteria regardless of source.
- CalEPA has proposed RELs that could be used in preference to USEPA RfCs. Since RELs are proposed only, their use may be premature. Some or all of the values may change before the final values are accepted for use in risk assessment in California. In the screening level assessment, TAPs of concern were identified with and without inclusion of RELS. Use of RELs in the final HHRA will be predicated on discussions with the Health Effects Research Division (HERD) and on professional judgement, unless the RELs are finalized prior to the completion of the final HHRA.
- A few TAPs of concern, notably 1,3-butadiene and acrolein, are likely to dominate exposures and risks associated with LAX emissions. New health assessments have recently been completed, or are ongoing, for both of these TAPs. The final HHRA considered the latest toxicological evaluations for both of these chemicals.

Attachment B1
List of Chemicals to be Considered

Table B1-1

AB2588 Substances for which Emissions Must be Quantified

Substance Name	See Note	Chemical Abstract Number
Acetaldehyde	C	75-07-0
Acetamide	C	60-35-5
Acrolein		107-02-8
Acrylamide	C	79-06-1
Acrylonitrile	C	107-13-1
Allyl chloride		107-05-1
2-Aminoanthraquinone	C	117-79-3
Amitrole	C	61-82-5
Ammonia		766-441-7
Arsenic	C	744-038-2
Arsenic compounds, inorganic	C	*
Arsine		778-442-1
Asbestos	C	133-221-4
Benzene	C	71-43-2
Benzidene (and its salts)	C	92-87-5
Benzidene – based dyes	C	
Benz(a)anthracene	C	56-55-3
Benzo(b)fluoranthene	C	205-99-2
Benzo(k)fluoranthene	C	207-08-9
Benzo(a)pyrene	C	50-32-8
Benzyl chloride		100-44-7
Beryllium	C	7440-41-7
Bis(chloromethyl)ether	C	542-88-1
Bromine		7726-95-6
Bromine compounds(inorganic)		*
1,3-Butadiene	C	106-99-0
Cadmium	C	7440-43-9
Cadmium compounds	C	*
Carbon black extracts	C	
Carbon tetrachloride	C	56-23-5
Carrageenan (degraded)	C	
Chlorinated fluorocarbon (CFC-113)		76-13-1
Chlorine		7782-50-5
Chloramphenicol	C	56-75-7
Chlorobenzene		108-90-7
1(2-Chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea (methyl CCNU)	C	13909-09-0
Chloroform	C	67-66-3
Chlorphenols	C	*
Chloropicrin		76-06-2
Chloroprene		126-99-8
4-Chloro-o-phenylenediamine	C	95-83-0
p-Chloro-o-toluidine	C	95-69-2
Chromium (hexavalent)	C	18540-29-9
Coke oven emissions	C	8007-45-2
Copper		7440-50-8
Creosotes	C	
p-Cresidine	C	120-71-8
Cresols		1319-77-3
Cupferron	C	135-20-6
Cycloheximide		66-81-9
Dialkylnitrosamines		*
2,4-Diaminoanisol	C	615-05-4
2,4-Diaminotoluene	C	95-80-7
Dibenzo(a,h)anthracene	C	53-70-3
Dibenzofurans(chlorinated)	C	*
1,2-Dibromo-3-chloropropane	C	96-12-8
p-Dichlorobenzene (1,4-dichlorobenzene)	C	106-46-7
3,3'-Dichlorobenzidine	C	91-94-1
Di(2-ethylhexyl)phthalate	C	117-81-7
Dimethylamine		124-40-3
p-Dimethylaminoazobenzene	C	60-11-7

Table B1-1

AB2588 Substances for which Emissions Must be Quantified

Substance Name	See Note	Chemical Abstract Number
1,1-Dimethylhydrazine	C	57-14-7
Dimethyl sulfate	C	77-78-1
1,4-Dioxane	C	123-91-1
Dioxins (chlorinated dibenzodioxins)	C	
Environmental tobacco smoke	C	
Epichlorohydrin	C	106-89-8
Ethyl acrylate	C	140-88-5
Ethyl chloride		75-00-3
Ethylene dibromide (1,2-dibromoethane)	C	106-93-4
Ethylene dichloride (1,2-dichloroethane)	C	107-06-2
Ethylene oxide	C	75-21-8
Ethylene thiourea	C	96-45-7
Fluorocarbons(chlorinated and brominated)		*
Formaldehyde	C	50-00-0
Gasoline vapors		
Glutaraldehyde		111-30-8
Glycol ethers		*
Griseofulvin	C	126-07-8
Hexachlorobenzene	C	118-74-1
Hexachlorocyclohexanes	C	*
Hexachlorocyclopentadiene		77-47-4
Hydrazene	C	302-01-2
Hydrochloric acid		7647-01-0
Hydrocyanic acid		74-90-8
Hydrogen fluoride		7664-39-3
Hydrogen sulfide		7783-06-4
Indeno(1,2,3,-cd)pyrene	C	193-39-5
Isocyanates		*
Lead	C	7439-92-1
Lead compounds (inorganic)	C	*
Maleic anhydride		108-31-6
Manganese		7439-96-5
Mercuric chloride		748-794-7
Mercury		7439-97-6
Methanol		67-56-1
Methyl bromide (bromomethane)		74-83-9
Methyl chloroform (1,1,1-trichloroethane)		71-55-6
Methyl isocyanate		624-83-9
Methyl methacrylate		80-62-6
4,4'-methylene bis (2-chloroaniline) (MOCA)	C	10-114-4
Methylene chloride (dichloromethane)	C	75-09-2
4,4'-Methylene dianiline (and its dichloride)	C	101-77-9
Methyl mercury (Dimethylmercury)		593-74-8
Metronidazole	C	443-48-1
Michler's ketone	C	90-94-8
Mineral fibers		
Napthalene		91-20-3
Nickel	C	7440-02-0
Nickel carbonyl	C	13463-39-3
Nickel subsulfide	C	12035-72-2
Niridazole	C	61-57-4
Nitrobenzene		98-95-3
Nitrogen mustard N-oxide	C	302-70-5
2-Nitropropane	C	79-46-9
N-Nitrosodiethyleneamine	C	55-18-5
N-Nitrosodimethyamine	C	62-75-9
p-Nitrosodiphenylamine	C	156-10-5
N-Nitrosondi-n-butylamine	C	924-16-3
N-Nitrosondi-n-propylamine	C	621-64-7
N-Nitrosomethylethylamine	C	10595-95-6
N-Nitrosomorpholine	C	59-89-2
N-Nitrosopiperidine	C	100-75-4

Table B1-1

AB2588 Substances for which Emissions Must be Quantified

Substance Name	See Note	Chemical Abstract Number
N-Nitrosopyrrolidine	C	930-55-2
Oxymetholone	C	434-07-1
PAHs (Polycyclic aromatic hydrocarbons)		**
PCBs (polychlorinated biphenyls)	C	1336-36-3
Perchloroethylene (tetrachloroethene)		127-18-4
Phenobarbitol	C	50-06-6
Phenol		108-95-2
Phosgene		75-44-5
Phosphine		7803-51-2
Phosphorus		7723-14-0
Phthalic anhydride		85-44-9
Potassium brominate	C	7758-01-2
Progesterolone	C	57-83-0
1,3-Propane sultone	C	1120-71-4
Propylene		115-07-1
Propylene oxide	C	75-56-9
Radionuclides		
Selenium		7782-49-2
Selenium compounds	C	*
Silica crystalline	C	
Sodium hydroxide		1310-73-2
Styrene	C	100-42-5
2,3,7,8-Tetrachlorodibenzodioxin (TCDD)	C	1746-01-6
Thioacetamide	C	62-55-5
Thiourea	C	62-55-6
Toluene		108-88-3
Toluene-2,4-diisocyanate	C	584-84-9
Toluene-2,6-diisocyanate	C	91-08-7
Trichloroethylene		79-01-6
2,4,6-Trichlorophenol	C	88-06-2
Urethane	C	51-79-6
Vinyl chloride	C	75-01-4
Vinylidene chloride		75-35-4
Xylenes		*
Zinc		7440-66-6
Zinc Oxide		1314-13-2

***** Denotes a chemical category, therefore no CAS number applies.

Note: The letter “C” in the notes column indicates that for the purpose of AB2588 the substance shall be treated as a human carcinogen or a potential human carcinogen.

Source: “Technical Guidance Document to the Criteria and Guidelines for AB2588” State of California Air Resources Board, August, 1989.

Table B1-2

CARB Toxic Air Contaminants
Listed Under AB 1807 and AB2728 (includes all Hazardous Air Pollutants listed in the Clean Air Act Amendments of 1990)

Chemical	CAS Number
Acetaldehyde	75-07-0
Acetamide	60-35-5
Acetonitrile	75-05-8
Acetophenone	98-86-2
2-Acetylaminofluorene	53-96-3
Acrolein	107-02-8
Acrylamide	79-06-1
Acrylic acid	79-01-7
Acrylonitrile	107-13-1
Allyl chloride	107-05-1
4-Aminobiphenyl	92-67-1
Aniline	62-53-3
o-Anisidine	90-04-0
Asbestos	1332-21-4
Benzene	71-43-2
Benzidine	92-87-5
Benzotrichloride	98-07-7
Benzyl chloride	100-44-7
Biphenyl	92-52-4
Bis(2-ethylhexyl)phthalate	117-81-7
Bis(chloromethyl)ether	542-88-1
Bromoform	75-25-2
1,3-Butadiene	106-99-0
Cadmium (metallic cadmium and cadmium compounds)	*
Calcium cyanamide	156-62-7
Caprolactam	105-60-2
Captan	133-06-2
Carbaryl	63-25-2
Carbon disulfide	75-15-0
Carbon tetrachloride	52-23-5
Carbonyl sulfide	463-58-1
Catechol	120-80-9
Chloramben	133-90-4
Chlordane	57-74-9
Chlorine	7782-50-5
Chlorinated dioxins and dibenzofurans (15 species)	
Chloroacetic acid	79-11-8
2-Chloroacetophenone	532-27-4
Chlorobenzene	108-90-7
Chlorobenzilate	510-15-6
Chloroform	67-66-3
Chloromethyl methyl ether	107-30-2
Chloroprene	126-99-8
Chromium VI	
Cresols/Cresylic acid	1319-77-3
o-Cresol	95-48-7
m-Cresol	108-39-4
p-Cresol	106-44-5
Cumene	98-82-8
2,4-D, salts and esters	94-75-7
DDE	3547-04-4
Diazomethane	334-88-3
Dibenzofurans	132-64-9
1,2-Dibromo-3-chloropropane	96-12-8
Dibutylphthalate	84-74-2
1,4-Dichlorobenzene	106-46-7
3,3-Dichlorobenzidene	91-94-1
Dichloroethyl ether (bis(2-chloroethyl)ether)	111-44-4

Table B1-2

CARB Toxic Air Contaminants
Listed Under AB 1807 and AB2728 (includes all Hazardous Air Pollutants listed in the Clean Air Act Amendments of 1990)

Chemical	CAS Number
1,3-Dichloropropene	542-75-6
Dichlorvos	62-73-7
Diethanolamine	111-42-2
N,N-Dimethyl aniline	121-69-7
Diethyl sulfate	64-67-5
3,3-Dimethoxybenzidine	119-90-4
Dimethyl aminoazobenzene	60-11-7
3,3-Dimethylbenzidene	119-93-7
Dimethyl carbamoyl chloride	79-44-7
Dimethyl formamide	68-12-2
1,1-Dimethyl hydrazine	57-14-7
Dimethyl phthlate	131-11-3
Dimethyl sulfate	77-78-1
4,6-Dinitro-o-cresol, and its salts	534-52-1
2,4-Dinitrophenol	51-28-5
2,4-Dinitrotoluene	121-14-2
1,4-Dioxane	123-91-1
1,2-Diphenylhydrazine	122-66-7
Epichlorohydrin	106-89-8
1,2-Epoxybutane	106-88-7
Ethyl acrylate	140-88-5
Ethyl benzene	100-41-4
Ethyl carbamate (urethane)	51-79-6
Ethyl chloride (chloroethane)	75-00-3
Ethylene dibromide (dibromoethane)	106-93-4
Ethylene dichloride (1,2 dichloroethane)	107-06-2
Ethylene glycol	107-21-1
Ethylene imine (Aziridine)	151-56-4
Ethylene oxide	75-21-8
Ethylene thiourea	96-45-7
Ethylidene dichloride (1,1-dichloroethane)	75-34-3
Formaldehyde	50-00-0
Heptachlor	76-44-8
Hexachlorobenzene	118-74-1
Hexachlorobutadiene	87-68-3
Hexachlorocyclopentadiene	77-47-4
Hexachloroethane	67-72-1
Hexamethylene-1,6-diisocyanate	822-06-0
Hexamethylphosphoramide	680-31-9
Hexane	110-54-3
Hydrazine	302-01-2
Hydrochloric acid	7647-01-1
Hydrogen fluoride	7664-39-3
Hydroquinone	123-31-9
Isophorone	78-59-1
Lindane (all isomers)	58-89-9
Maleic anhydride	108-31-6
Methanol	67-56-1
Methoxychlor	72-43-5
Methyl bromide (bromomethane)	74-83-9
Methyl chloride	74-87-3
Methyl chloroform (1,1,1-trichloroethane)	71-55-6
Methyl ethyl ketone (2-butanone)	78-93-3
Methyl hydrazine	60-34-8
Methyl iodide	74-88-4
Methyl isobutyl ketone (hexone)	108-10-1
Methyl isocyanate	624-83-9
Methyl methacrylate	80-62-6
Methyl-t-butyl ether	1634-04-4
4,4'-Methylene-bis(2-chloroaniline)	101-14-4

Table B1-2

CARB Toxic Air Contaminants
Listed Under AB 1807 and AB2728 (includes all Hazardous Air Pollutants listed in the Clean Air Act Amendments of 1990)

Chemical	CAS Number
Methylene chloride (dichloromethane)	75-09-2
Methylene diphenyl diisocyanate (MDI)	101-68-8
4,4'-Methylenedianiline	101-77-9
Napthalene	91-20-3
Nickel and nickel compounds	*
Nitrobenzene	98-95-3
4-Nitrobiphenyl	92-93-3
4-Nitrophenol	100-02-7
2-Nitropropane	79-46-9
N-Nitroso-N-methylurea	684-93-5
N-Nitrosodimethylamine	62-75-9
N-Nitrosomorpholine	59-89-2
Parathion	56-38-2
Pentachloronitrobenzene	82-68-8
Pentachlorophenol	87-86-5
Phenol	108-95-2
p-Phenylenediamine	106-50-3
Phosgene	75-44-5
Phosphine	7803-51-2
Phosphorus	7723-14-0
Phthalic anhydride	85-44-9
Polychlorinated biphenyls (Aroclors)	1336-36-3
1,3-Propane sultone	1120-71-4
Beta-propiolactone	57-57-8
Proprionaldehyde	123-38-6
Propoxur (Baygon)	114-26-1
Propylene dichloride (1,2-dichloropropane)	78-87-5
Propylene oxide	75-56-9
1,2-Propylenimine (2-methyl aziridine)	75-55-8
Quinoline	91-22-5
Quinone	106-51-4
Styrene	100-42-5
Styrene oxide	96-09-3
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1746-01-6
1,1,2,2-Tetrachloroethane	79-34-5
Tetrachloroethylene (perchloroethylene)	127-18-4
Titanium tetrachloride	7550-45-0
Toluene	108-88-3
2,4-Toluene diamine	95-80-7
2,4-Toluene diisocyanate	584-84-9
o-Toluidine	95-53-4
Toxaphene (chlorinated camphene)	8001-35-2
1,2,4-Trichlorobenzene	120-82-1
1,1,2-Trichlorothane	79-00-5
Trichloroethylene	79-01-6
2,4,5-Trichlorophenol	95-95-4
2,4,6-Trichlorophenol	88-06-2
Triethylamine	121-44-8
Trifluoralin	1582-09-8
2,2,4-Trimethylpentane	540-84-1
Vinyl acetate	108-05-4
Vinyl bromide	593-60-2
Vinyl chloride	75-01-4
Vinylidene chloride (1,1-dichloroethylene)	75-35-4
Xylenes (isomers and mixture)	1330-20-7
o-Xylenes	95-47-6
m-Xylenes	108-38-3
p-Xylenes	106-42-3
Antimony Compounds	
Arsenic compounds (inorganic including arsine)	

Table B1-2

CARB Toxic Air Contaminants
Listed Under AB 1807 and AB2728 (includes all Hazardous Air Pollutants listed in the Clean Air Act Amendments of 1990)

Chemical	CAS Number
Beryllium compounds	
Cadmium compounds	
Chromium compounds	
Cobalt compounds	
Coke oven emissions	
Cyanide compounds[1]	
Glycol ethers[2]	
Lead compounds	
Manganese compounds	
Mercury compounds	
Fine mineral fibers[3]	
Nickel Compounds	
Polycyclic organic matter[4]	
Radionuclides (including radon)[5]	
Selenium compounds	

Note: For all listings above which contain the word "compounds" and for glycol ethers the following applies: Unless otherwise specified, these listings are defined as including any unique chemical substance that contains the named chemical (i.e., antimony, arsenic, etc.) as part of that chemical's structure.

[1] 'CN where X=H' or any other group where a formal dissociation may occur. For example KCNB or $CA(CN)_2$

[2] Includes mono- and di- ethers of ethylene glycol, diethylene glycol, and triethylene glycol $R\text{-}OCH_2CH_2)_n\text{-}$ OR' where:

n=1,2,or 3
R = alkyl or aryl groups
R' = R, H, or groups which, when removed, yield glycol ethers with the structure:
$R\text{-}(OCH_2CH)_n\text{-}OH$

Polymers are excluded from the glycol category.

[3] Includes mineral rock fiber emissions from facilities manufacturing or processing glass, rock ,or slag fibers (or other mineral derived fibers) of average diameter 1 micrometer or less.

[4] Includes organic compounds with more than one benzene ring , and which have a boiling point greater than or equal to 100° C.

[5] A type of atom which spontaneously undergoes radioactive decay.

Source: June 1996 ARB Toxic Air Contaminants

Table B1-3

SCQMD Rule 1401/1402 Pollutants

Substance	CAS Number[a]	Date of Listing
SCAQMD Rule 1401 Toxic Air Pollutants		
Acetaldehyde	75-07-0	December 7, 1990
Acrylamide	79-06-01	December 7, 1990
Acrylonitrile	107-13-1	December 7, 1990
Arsenic (inorganic)	7440-38-2	December 7, 1990
Asbestos	1332-21-4	June 1, 1990
Benzene	71-43-2	June 1, 1990
Benzidene	92-87-5	December 7, 1990
Polynuclear Aromatics Hydrocarbons (PAH)	*	
Benz(a)anthracene	56-55-3	December 7, 1990
Benzo(a)pyrene	50-32-8	December 7, 1990
Benzo(b)fluoranthene	205-99-2	December 7, 1990
Benzo(k)fluoranthene	207-08-9	December 7, 1990
Chrysene	218-01-9	December 7, 1990
Dibenzo(a,h)anthracene	53-70-3	December 7, 1990
Indenopyrene	193-39-5	December 7, 1990
Beryllium	7440-41-7	December 7, 1990
Bis(2-chloroethyl)ether	111-44-4	December 7, 1990
Bis(chloromethyl)ether	542-88-1	December 7, 1990
1,3-Butadiene	106-99-0	December 7, 1990
Cadmium	7440-43-9	June 1, 1990
Carbon Tetrachloride	56-23-5	June 1, 1990
Chlorinated Dioxins and Dibenzofurans (TCDD equivalents)[b]	*	June 1, 1990
Chloroform	67-66-3	December 7, 1990
Chromium, hexavalent	7440-47-3	June 1, 1990
3,3-Dichlorobenzidene	91-94-1	December 7, 1990
2,4-Dinitrotoluene	121-14-2	December 7, 1990
1,4-Dioxane	123-91-1	December 7, 1990
Diphenylhydrazine	122-66-7	December 7, 1990
Epichlorohydrin	106-89-8	December 7, 1990
Ethylene dibromide	106-93-4	June 1, 1990
Ethylene dichloride (1,2-dichloroethane)	107-06-2	June 1, 1990
Ethylene oxide	75-21-8	June 1, 1990
Formaldehyde	50-00-0	December 7, 1990
Hexachlorobenzene	118-74-1	December 7, 1990
Hexachlorocyclohexane	-------	
Technical Grade	--------*	December 7, 1990
Alpha isomer	319-84-6	December 7, 1990
Methylene chloride	75-09-2	June 1, 1990
Nickel		
Refinery dust	-------*	December 7, 1990
Subsulfide	0120-35-722	December 7, 1990
N-Nitroso Compounds		
Dimethylnitrosamine	62-75-9	December 7, 1990
Diethylnitrosamine	55-18-5	December 7, 1990
Dibutylnitrosamine	924-16-3	December 7, 1990
N-Nitrososopyrrolidine	930-55-2	December 7, 1990
N-Nitrosodiphenylamine	86-30-6	December 7, 1990
N-Nitroso-N-ethylurea	759-73-9	December 7, 1990
N-Nitroso-N-methylurea	684-93-5	December 7, 1990
Polychlorinated biphenyls	1336-36-3	December 7, 1990
Trichloroethylene	79-01-6	December 7, 1990
2,4,6-trichlorophenol	88-06-2	December 7, 1990
Vinyl chloride	75-01-4	December 7, 1990
SCAQMD Rule 1401/1402 Pollutants		
Acetaldehyde	75-07-0	C, CH
Acrolein	107-02-8	CH, AH
Acrylamide	79-06-1	C,CH
Acrylonitrile	107-13-1	C,CH

Table B1-3

SCQMD Rule 1401/1402 Pollutants

Substance	CAS Number[a]	Date of Listing
Ammonia	7664-41-7	CH, AH
Arsenic	7440-38-2	C, CH
Arsenic and compounds (inorganic)	*	C
Arsine	7784-42-1	AH
Asbestos	1332-21-4	C
Benzene	71-43-2	C,CH
Benzidene and its salts	92-87-5	C, CH
Bez(a)anthracene	56-55-3	C
Benzo(b)fluoranthene	205-99-2	C
Benzo(k)fluoranthene	207-08-9	C
Benzo(a)pyrene	50-32-8	C
Bemzyl chloride	100-44-7	CH AH
Berylium	7440-41-7	C, CH
Bis(2-chloroethyl)ether	111-44-4	C
Bis(chloromethyl)ether	542-88-1	C
Bromine	7726-95-6	CH
Bromine pentafluoride	7789-30-2	CH
1,3-Butadiene	106-99-0	C
Cadmium	7440-43-9	C, CH
Cadmium compounds	*	C
Carbon tetrachloride	56-23-5	C, CH, AH
Chlorinated fluorocarbon (CFC-113)	76-13-1	CH
Chlorine	7782-50-5	CH, AH
Chlorobenzene	108-90-7	CH
Chloroform	67-66-3	C, CH
2-Chlorophenol	95-57-8	CH
Chloropicrin	76-06-2	CH
Chloroprene	126-99-8	C, CH
Chromium, hexavalent	7440-47-3	C, CH
Chrysene	218-01-9	C
Coke oven emission	8007-45-2	C
Copper	7440-50-8	CH, AH
Copper compounds	*	CH, AH
Cresols	1319-77-3	CH
Dibenzo(a,h)anthracene	53-70-3	
Dibenzofurans(chlorinated)	*	C, CH
1,2-Dibromo-3-chloropropane	96-12-8	C, CH
p-dichlorobenzene (1,4-dichlorobenzene)	106-46-7	C, CH
3,3'-dichlorbenzidene	91-94-1	C
Di(2-ethylhexyl)phthalate	117-81-7	C, CH
Dimethylamine	124-40-3	CH
2,4-Dinitrotolune	121-14-2	C
1,4-Dixoane	123-91-1	C, CH, AH
Dioxins (chlorinated dibenzodioxins)	*	C, CH
Diphenylhydrazene	122-66-7	C
Epichlorohydrin	106-89-8	C, CH
Ethyl acrylate	140-88-5	CH
Ethyl chloride	75-00-3	CH
Ethylene dibromide (1,2-dibromethane)	106-93-4	C, CH
Ethylene dichloride (1,2-dichloroethane)	107-06-2	C, CH
Ethylene glycol butyl ether	111-76-2	CH, AH
Ethylene glycol ethyl ether	110-80-5	CH, AH
SCQMD Rule 1401/1402 Pollutants		
Ethylene glycol ethylether acetate	111-15-9	CH, AH
Ethylene glycol methyl ether	109-86-4	CH, AH
Ethylene glycol methylethyl ether acetate	110-49-6	CH
Ethylene oxide	75-21-8	C, CH
Formaldehyde	50-00-0	C, CH, AH
Glutaraldehyde	111-30-8	CH
Hexachlorobenzene	118-74-1	C, CH
Hexachlorcyclohexanes	*	C
Hexachlorcyclohexane (gamma isomer)	58-89-9	CH

Table B1-3

SCQMD Rule 1401/1402 Pollutants

Substance	CAS Number[a]	Date of Listing
Hexchlorocyclopentadiene	77-47-4	CH
Hydrazine	302-01-2	C, CH
Hydrochloric acid	7647-01-0	CH, AH
Hydrogen bromide	10035-10-6	CH
Hydrogen cyanide	74-90-8	CH, AH
Hydrogen fluoride	7664-39-3	CH, AH
Hydrogen sulfide	7783-06-4	CH, AH
Indeno(1,2,3-cd)pyrene	193-39-5	C
Lead	7439-92-1	CH
Lead compounds	*	CH
Maleic anhydride	108-31-6	CH, AH
Manganese	7439-96-5	CH
Manganese compounds	*	CH
Mercury	7439-97-6	CH
Mercury and compounds (inorganic)	*	CH, AH
Methanol	67-56-1	CH
Methyl bromide	74-83-9	CH
Methyl chloroform (1,1,1-trichloroethane	71-55-6	CH, AH
Methy isocyanate	624-83-9	CH
Methyl methacrylate	80-62-6	CH
Methylene chloride (dichloromethane)	75-09-2	C, CH, AH
4,4'-methylene dianiline (and its dichloride)	101-77-9	CH
Methyl mercury	593-74-8	CH
Mineral fibers	*	CH
Napthalene	91-20-3	CH
Nickel	7440-02-0	C, CH, AH
Nickel compounds	*	CH, AH
Nickel carbonyl	13463-39-3	C
Nickel subsulfide	012035-72-2	C
Nitrobenzene	98-95-3	CH
n-Nitrosodiethyleneamine	55-18-5	C
n-Nitrosodimethylamine	62-75-9	C
n-Nitroso-di-n-butylamine	924-16-3	C
n-Nitroso-di-n-propylamine	621-64-7	C
n-Nitrosomethylethylamine	10595-95-6	C
n-Nitrosodiphenylamine	86-30-6	C
n-Nitrosopyrrollidine	930-55-2	C
n-Nitros-n-ethylurea	759-73-9	C
n-Nitroso-n-methylurea	684-93-5	C
PCBs (polychlorinated biphenyls)	1336-36-3	C, CH
Pentachlorophenol	87-86-5	C, CH
Perchloroethylene	127-18-4	C, CH, AH
Phenol	108-95-2	CH
Phosgene	75-44-5	AH
Phosphine	7803-51-2	CH
Phosphorus	7723-14-0	CH
Phthalic anhydride	85-44-9	CH
Propylene oxide	75-56-9	C, CH, AH
Selenium	7782-49-2	CH, AH
Selenium compounds	*	CH, AH
Sodium hydroxide	1310-73-2	CH, AH
Styrene	100-42-5	CH
Tetrachlorophenols	*	CH
Toluene	108-88-3	CH
Toluene-2,4-diisocyanate	584-84-9	CH
Toluene-2,6-diidocyanate	91-08-7	CH
Trichloroethylene	79-01-6	C, CH
2,4,6-Trichlorophenol	88-06-2	C
Urethane	51-79-6	C
Vinyl chloride	75-01-4	C,CH
Vinylidene chloride	75-35-4	C,CH
Xylenes	*	CH, AH

Table B1-3

SCQMD Rule 1401/1402 Pollutants

Substance	CAS Number[a]	Date of Listing
Zinc	7440-66-6	CH
Zinc compounds	*	CH

Note: Rule 1402 is similar to Rule 1401, except it applies to existing sources of Toxic Air Pollutants. The chemicals which must be considered under Rule 1402 are given in Table 4-5.

* Indicates a class of compounds, therefore no CAS number applies

Table B1-4

U.S. EPA Hazardous Air Pollutants - CAAA Section 112(b)

Chemical	CAS Number
Acetaldehyde	75-07-0
Acetamide	60-35-5
Acetonitrile	75-05-8
Acetophenone	98-86-2
2-Acetylaminofluorene	53-96-3
Acrolein	107-02-8
Acrylamide	79-06-1
Acrylic acid	79-01-7
Acrylonitrile	107-13-1
Allyl chloride	107-05-1
4-Aminobiphenyl	92-67-1
Aniline	62-53-3
o-Anisidine	90-04-0
Asbestos	1332-21-4
Benzene	71-43-2
Benzidine	92-87-5
Benzotrichloride	98-07-7
Benzyl chloride	100-44-7
Biphenyl	92-52-4
Bis(2-ethylhexyl)phthalate	117-81-7
Bis(chloromethyl)ether	542-88-1
Bromoform	75-25-2
1,3-Butadiene	106-99-0
Calcium cyanamide	156-62-7
Caprolactam	105-60-2
Captan	133-06-2
Carbaryl	63-25-2
Carbon disulfide	75-15-0
Carbon tetrachloride	52-23-5
Carbonyl sulfide	463-58-1
Catechol	120-80-9
Chloramben	133-90-4
Chlordane	57-74-9
Chlorine	7782-50-5
Chloroacetic acid	79-11-8
2-Chloroacetophenone	532-27-4
Chlorobenzene	108-90-7
Chlorobenzilate	510-15-6
Chloroform	67-66-3
Chloromethyl methyl ether	107-30-2
Chloroprene	126-99-8
Cresols/Cresylic acid	1319-77-3
o-Cresol	95-48-7
m-Cresol	108-39-4
p-Cresol	106-44-5
Cumene	98-82-8
2,4-D, salts and esters	94-75-7
DDE	3547-04-4
Diazomethane	334-88-3
Dibenzofurans	132-64-9
1,2-Dibromo-3-chloropropane	96-12-8
Dibutylphthalate	84-74-2
1,4-Dichlorobenzene	106-46-7
3,3-Dichlorobenzidene	91-94-1
Dichloroethyl ether (bis(2-chloroethyl)ether	111-44-4
1,3-Dichloropropene	542-75-6
Dichlorvos	62-73-7
Diethanolamine	111-42-2
N,N-Diethyl aniline	121-69-7
Diethyl sulfate	64-67-5
3,3-Dimethoxybenzidine	119-90-4

Table B1-4

U.S. EPA Hazardous Air Pollutants - CAAA Section 112(b)

Chemical	CAS Number
Dimethyl aminobenzene	60-11-7
3,3-Dimethylbenzidine	119-93-7
Dimethyl carbamoyl chloride	79-44-7
Dimethyl formamide	68-12-2
1,1-Dimethyl hydrazine	57-14-7
Dimethyl phthlate	131-11-3
Dimethyl sulfate	77-78-1
4,6-Dinitro-o-cresol, and its salts	534-52-1
2,4-Dinitrophenol	51-28-5
2,4-Dinitrotoluene	121-14-2
1,4-Dioxane	123-91-1
1,2-Diphenylhydrazine	122-66-7
Epichlorohydrin	106-89-8
1,2-Epoxybutane	106-88-7
Ethyl acrylate	140-88-5
Ethyl benzene	100-41-4
Ethyl carbamate (urethane)	51-79-6
Ethyl chloride (chloroethane)	75-00-3
Ethylene dibromide (dibromoethane)	106-93-4
Ethylene dichloride (1,2 dichloroethane)	107-06-2
Ethylene glycol	107-21*-1
Ethylene imine (Aziridine)	151-56-4
Ethylene oxide	75-21-8
Ethylene thiourea	96-45-7
Ethylidene dichloride (1,1-dichloroethane)	75-34-3
Formaldehyde	50-00-0
Heptachlor	76-44-8
Hexachlorobenzene	118-74-1
Hexachlorobutadiene	87-68-3
Hexachlorocyclopentadiene	77-47-4
Hexachloroethane	67-72-1
Hexamethylene-1,6-diisocyanate	822-06-0
Hexamethylphosphoramide	680-31-9
Hexane	110-54-3
Hydrazine	302-01-2
Hydrochloric acid	7647-01-1
Hydrogen fluoride	7664-39-3
Hydrogen sulfide	7783-06-4
Hydroquinone	123-31-9
Isophorone	78-59-1
Lindane (all isomers)	58-89-9
Malic anhydride	108-31-6
Methanol	67-56-1
Methoxychlor	72-43-5
Methyl bromide (bromomethane)	74-83-9
Methyl chloride	74-87-3
Methyl chloroform (1,1,1-trichloroethane)	71-55-6
Methyl ethyl ketone (2-butanone)	78-93-3
Methyl hyrazine	60-34-8
Methyl iodide	74-88-4
Methyl isobutyl ketone (hexone)	108-10-1
Methyl isocyanate	624-83-9
Methyl methacrylate	80-62-6
Methyl-t-butyl ether	1634-04-4
4,4'-Methylene-bis(2-chloroaniline)	101-14-4
Methylene chloride (dichloromethane)	75-09-2
Methylene diphenyl diisocyanate (MDI)	101-68-8
4,4'-Methylenedianiline	101-77-9
Napthalene	91-20-3
Nitrobenzene	98-95-3
4-Nitrobiphenyl	92-93-3
4-Nitrophenol	100-02-7

Table B1-4

U.S. EPA Hazardous Air Pollutants - CAAA Section 112(b)

Chemical	CAS Number
2-Nitropropane	79-46-9
N-Nitroso-N-methylurea	684-93-5
N-Nitrosodimethylamine	62-75-9
N-Nitrosomorpholine	59-89-2
Parathion	56-38-2
Pentachloronitrobenzene	82-68-8
Pentachlorophenol	87-86-5
Phenol	108-95-2
p-Phenylenediamine	106-50-3
Phosgene	75-44-5
Phosphine	7803-51-2
Phosphorus	7723-14-0
Phthalic anhydride	85-44-9
Polychlorinated biphenyls (Aroclors)	1336-36-3
1,3-Propane sultone	1120-71-4
Beta-Propiolactone	57-57-8
Proprionaldehyde	123-38-6
Propoxur (Baygon)	114-26-1
Propylene dichloride (1,2-dichloropropane)	78-87-5
Propylene oxide	75-56-9
1,2-Propylenimine (2-Methyl aziridine)	75-55-8
Quinoline	91-22-5
Quinone	106-51-4
Styrene	100-42-5
Styrene oxide	96-09-3
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1746-01-6
1,1,2,2-Tetrachloroethane	79-34-5
Tetrachloroethylene (perchloroethylene)	127-18-4
Titanium tetrachloride	7550-45-0
Toluene	108-88-3
2,4-Toluene diamine	95-80-7
2,4-Toluene diisocyanate	584-84-9
o-Toluidine	95-53-4
Toxaphene (chlorinated camphene)	8001-35-2
1,2,4-Trichlorobenzene	120-82-1
1,1,2-Trichlorothane	79-00-5
Trichloroethylene	79-01-6
2,4,5-Trichlorophenol	95-95-4
2,4,6 Trichlorophenol	88-06-2
Triethylamine	121-44-8
Trifluoralin	1582-09-8
2,2,4-Trimethylpentane	540-84-1
Vinyl acetate	108-05-4
Vinyl bromide	593-60-2
Vinyl chloride	75-01-4
Vinylidene chloride (1,1-dichloroethylene)	75-35-4
Xylenes (isomers and mixture)	1330-20-7
o-Xylenes	95-47-6
m-Xylenes	108-38-3
p-Xylenes	106-42-3
Antimony Compounds	
Arsenic compounds (inorganic including arsine)	
Beryllium compounds	
Cadmium compounds	
Chromium compounds	
Cobalt compounds	
Coke oven emissions	
Cyanide compounds (1)	
Glycol ethers (2)	
Lead compounds	
Manganese compounds	

Table B1-4

U.S. EPA Hazardous Air Pollutants - CAAA Section 112(b)

Chemical	CAS Number
Mercury compounds	
Fine mineral fibers (3)	
Nickel Compounds	
Polycyclic organic matter (4)	
Radionuclides (including radon) (5)	
Selenium compounds	

Note: For all listings above which contain the word "compounds" and for glycol ethers, the following applies: Unless otherwise specified, these listings are defined as including any unique chemical substance that contains the named chemical (i.e., antimony, arsenic, etc.) as part of that chemical's structure.

[1] X'CN where X=H' or any other group where a formal dissociation may occur. For example KCNB or $CA(CN)_2$

[2] Includes mono- and di-ethers of ethylene glycol, diethylene glycol, and triethylene glycol

$R\text{-}OCH_2CH_2)_n\text{-}$ OR' where:

n=1,2,or 3
R = alkyl or aryl groups
R' = R, H, or groups which, when removed, yield glycol ethers with the structure: $R\text{-}(OCH_2CH)_n\text{-}OH$

Polymers are excluded from the glycol category.

[3] Includes mineral rock fiber emissions from facilities manufacturing or processing glass, rock, or slag fibers (or other mineral derived fibers) of average diameter 1 micrometer or less.

[4] Includes organic compounds with more than one benzene ring , and which have a boiling point greater than or equal to 100° C.

[5] A type of atom which spontaneously undergoes radioactive decay.

Source: U.S. EPA Clean Air Act Section 112

Table B1-5

Draft CAA Emissions Inventory – Aircraft (September 5, 1997)

Compound	CAS Number	Present in Rule 1401 List?
Benzene	71-43-2	Yes
1,3-Butadiene	106-99-0	Yes
Formaldehyde	50-00-0	Yes
16- PAH[(a)]	----	
Acenapthalene		
Acenapthylene		
Anthracene		
Benzo(ghi)perylene		
Fluoranthene		
Fluorene		
Napthalene	91-20-3	Yes
Phenanthrene		
Pyrene		
7-PAH[(b)]	----	
Benz(a)anthracene	556-55-3	Yes
Benzo(b)fluoranthene	205-99-20	Yes
Benzo(k)fluoranthene	207-08-9	Yes
Benzo(a)pyrene	50-32-8	Yes
Dibenzo(a,h) anthracene	53-70-3	Yes
Chrysene	218-01-9	Yes
Indeno(1,2,3-cd)pyrene	193-39-5	Yes
Acetaldehyde	75-07-0	Yes
Acrolein	107-02-8	No
Styrene	100-42-5	No
Lead	7439-92-1	No

Source: U.S. EPA Clean Air Act Section 112(k)

Attachment B2
Calculation of Estimated Soil Concentrations from Particle Deposition

Table B2-1

Preliminary
Future Baseline 2015 w/o LCD, for Comparison to Alternative A
Annual Averages

	Maximum Concentrations in µg/m^3				Maximum Deposition Rates in µg/s/m^2 *			
TAP	@Fenceline	@Resident**	@School***	On Airport	@Fenceline	@Resident**	@School***	On Airport
Acetaldehyde	3.83E+00	6.96E-01	6.64E-01	8.17E+01	****	****	****	****
Acrolein	1.86E+00	3.38E-01	3.23E-01	3.99E+01	****	****	****	****
Benzene	1.90E+00	3.39E-01	3.20E-01	3.42E+01	****	****	****	****
1,3-Butadiene	1.56E+00	2.80E-01	2.67E-01	3.17E+01	****	****	****	****
Formaldehyde	1.24E+01	2.25E+00	2.14E+00	2.64E+02	****	****	****	****
Toluene	3.22E+00	1.48E-01	1.35E-01	9.16E+00	****	****	****	****
Benzo(a)anthracene	1.56E-04	1.41E-05	1.28E-05	5.99E-04	2.80E-07	2.53E-08	2.30E-08	1.08E-06
Benzo(a)pyrene	6.87E-05	7.23E-06	6.64E-06	2.53E-04	1.24E-07	1.30E-08	1.20E-08	4.56E-07
Benzo(b)fluoranthene	4.71E-05	3.60E-06	3.31E-06	1.83E-04	8.48E-08	6.49E-09	5.97E-09	3.30E-07
Benzo(k)fluoranthene	5.08E-05	3.85E-06	3.54E-06	1.98E-04	9.14E-08	6.93E-09	6.38E-09	3.57E-07
Chrysene	1.14E-04	9.34E-06	8.52E-06	4.47E-04	2.05E-07	1.68E-08	1.53E-08	8.04E-07
Dibenz(a,h)anthracene	2.20E-05	1.91E-06	1.78E-06	8.70E-05	3.97E-08	3.43E-09	3.20E-09	1.57E-07
Indeno(1,2,3-cd)pyrene	2.21E-05	1.92E-06	1.79E-06	8.79E-05	3.98E-08	3.45E-09	3.22E-09	1.58E-07
Arsenic	2.16E-01	4.74E-02	4.44E-02	1.16E+00	3.89E-04	8.54E-05	7.98E-05	2.09E-03
Cadmium	2.03E-02	4.49E-03	4.20E-03	1.09E-01	3.66E-05	8.07E-06	7.56E-06	1.97E-04
Chromium (Total)	2.16E-01	4.76E-02	4.45E-02	1.16E+00	3.89E-04	8.56E-05	8.01E-05	2.09E-03
Lead	2.24E-01	4.93E-02	4.61E-02	1.21E+00	4.04E-04	8.87E-05	8.29E-05	2.17E-03
Manganese	2.26E-04	2.22E-05	1.97E-05	7.37E-04	4.06E-07	4.00E-08	3.54E-08	1.33E-06
Nickel	2.04E-02	4.51E-03	4.21E-03	1.09E-01	3.67E-05	8.12E-06	7.59E-06	1.97E-04
Trichloroethylene*****	0.00E+00	0.00E+00	0.00E+00	0.00E+00	****	****	****	****

*Deposition velocity assumed to be 0.0018 m/s for non-volatile compounds.
**Maximum Impacts occur near the 1100 block of E. Acacia Ave., in El Segundo.
***Maximum Impacts at Imperial Ave. School.
****Volatile compounds, no significant deposition assumed to occur.
*****Trichloroethylene will not be used by Airport tenants in future operations.

Table B2-2

Preliminary
Future Baseline 2015 w/LCD, for Comparison to Alternative A
Annual Averages

	Maximum Concentrations in µg/m^3				Maximum Deposition Rates in µg/s/m^2 *			
TAP	@Fenceline	@Resident**	@School***	On Airport	@Fenceline	@Resident**	@School***	On Airport
Acetaldehyde	3.83E+00	6.96E-01	6.64E-01	8.17E+01	****	****	****	****
Acrolein	1.86E+00	3.38E-01	3.23E-01	3.99E+01	****	****	****	****
Benzene	1.97E+00	3.52E-01	3.32E-01	3.42E+01	****	****	****	****
1,3-Butadiene	1.56E+00	2.81E-01	2.68E-01	3.17E+01	****	****	****	****
Formaldehyde	1.24E+01	2.25E+00	2.14E+00	2.64E+02	****	****	****	****
Toluene	3.67E+00	1.71E-01	1.56E-01	9.22E+00	****	****	****	****
Benzo(a)anthracene	1.56E-04	1.41E-05	1.29E-05	5.99E-04	2.80E-07	2.55E-08	2.31E-08	1.08E-06
Benzo(a)pyrene	6.87E-05	7.27E-06	6.68E-06	2.53E-04	1.24E-07	1.31E-08	1.20E-08	4.56E-07
Benzo(b)fluoranthene	4.72E-05	3.64E-06	3.35E-06	1.83E-04	8.49E-08	6.55E-09	6.03E-09	3.30E-07
Benzo(k)fluoranthene	5.09E-05	3.89E-06	3.58E-06	1.98E-04	9.15E-08	7.00E-09	6.44E-09	3.57E-07
Chrysene	1.14E-04	9.40E-06	8.57E-06	4.47E-04	2.05E-07	1.69E-08	1.54E-08	8.05E-07
Dibenz(a,h)anthracene	2.21E-05	1.92E-06	1.79E-06	8.70E-05	3.97E-08	3.45E-09	3.22E-09	1.57E-07
Indeno(1,2,3-cd)pyrene	2.21E-05	1.93E-06	1.80E-06	8.79E-05	3.98E-08	3.47E-09	3.23E-09	1.58E-07
Arsenic	2.16E-01	4.74E-02	4.44E-02	1.16E+00	3.89E-04	8.54E-05	7.98E-05	2.09E-03
Cadmium	2.03E-02	4.49E-03	4.20E-03	1.09E-01	3.66E-05	8.07E-06	7.56E-06	1.97E-04
Chromium (Total)	2.16E-01	4.76E-02	4.45E-02	1.16E+00	3.89E-04	8.56E-05	8.01E-05	2.09E-03
Lead	2.24E-01	4.93E-02	4.61E-02	1.21E+00	4.04E-04	8.87E-05	8.29E-05	2.17E-03
Manganese	3.54E-04	2.57E-05	2.28E-05	7.41E-04	6.37E-07	4.62E-08	4.10E-08	1.33E-06
Nickel	2.04E-02	4.51E-03	4.22E-03	1.09E-01	3.67E-05	8.12E-06	7.59E-06	1.97E-04
Trichloroethylene*****	0.00E+00	0.00E+00	0.00E+00	0.00E+00	****	****	****	****

*Deposition velocity assumed to be 0.0018 m/s for non-volatile compounds.
**Maximum Impacts occur near the 1100 block of E. Acacia Ave., in El Segundo.
***Maximum Impacts at Imperial Ave. School.
****Volatile compounds, no significant deposition assumed to occur.
*****Trichloroethylene will not be used by Airport tenants in future operations.

Table B2-3

Preliminary
Future Baseline 2015 w/o LCD, for Comparison to Alternative B
Annual Averages

	Maximum Concentrations in µg/m^3				Maximum Deposition Rates in µg/s/m^2 *			
TAP	@Fenceline	@Resident**	@School***	On Airport	@Fenceline	@Resident**	@School***	On Airport
Acetaldehyde	3.83E+00	6.96E-01	6.64E-01	8.17E+01	****	****	****	****
Acrolein	1.86E+00	3.38E-01	3.23E-01	3.99E+01	****	****	****	****
Benzene	1.90E+00	3.39E-01	3.20E-01	3.42E+01	****	****	****	****
1,3-Butadiene	1.56E+00	2.80E-01	2.67E-01	3.17E+01	****	****	****	****
Formaldehyde	1.24E+01	2.25E+00	2.14E+00	2.64E+02	****	****	****	****
Toluene	3.22E+00	1.49E-01	1.36E-01	9.17E+00	****	****	****	****
Benzo(a)anthracene	1.56E-04	1.41E-05	1.28E-05	5.99E-04	2.80E-07	2.54E-08	2.31E-08	1.08E-06
Benzo(a)pyrene	6.87E-05	7.24E-06	6.66E-06	2.53E-04	1.24E-07	1.30E-08	1.20E-08	4.56E-07
Benzo(b)fluoranthene	4.72E-05	3.62E-06	3.33E-06	1.83E-04	8.49E-08	6.52E-09	5.99E-09	3.30E-07
Benzo(k)fluoranthene	5.09E-05	3.86E-06	3.56E-06	1.98E-04	9.16E-08	6.96E-09	6.40E-09	3.57E-07
Chrysene	1.14E-04	9.36E-06	8.54E-06	4.47E-04	2.05E-07	1.69E-08	1.54E-08	8.05E-07
Dibenz(a,h)anthracene	2.21E-05	1.92E-06	1.79E-06	8.71E-05	3.98E-08	3.46E-09	3.23E-09	1.57E-07
Indeno(1,2,3-cd)pyrene	2.22E-05	1.93E-06	1.80E-06	8.79E-05	4.00E-08	3.48E-09	3.25E-09	1.58E-07
Arsenic	2.16E-01	4.74E-02	4.44E-02	1.16E+00	3.89E-04	8.54E-05	7.98E-05	2.09E-03
Cadmium	2.03E-02	4.49E-03	4.20E-03	1.09E-01	3.66E-05	8.07E-06	7.56E-06	1.97E-04
Chromium (Total)	2.16E-01	4.76E-02	4.45E-02	1.16E+00	3.89E-04	8.56E-05	8.01E-05	2.09E-03
Lead	2.24E-01	4.93E-02	4.61E-02	1.21E+00	4.04E-04	8.87E-05	8.29E-05	2.17E-03
Manganese	2.26E-04	2.22E-05	1.97E-05	7.37E-04	4.06E-07	4.00E-08	3.54E-08	1.33E-06
Nickel	2.04E-02	4.51E-03	4.21E-03	1.09E-01	3.67E-05	8.12E-06	7.59E-06	1.97E-04
Trichloroethylene*****	0.00E+00	0.00E+00	0.00E+00	0.00E+00	****	****	****	****

*Deposition velocity assumed to be 0.0018 m/s for non-volatile compounds.
**Maximum Impacts occur near the 1100 block of E. Acacia Ave., in El Segundo.
***Maximum Impacts at Imperial Ave. School.
****Volatile compounds, no significant deposition assumed to occur.

Table B2-4

Preliminary
Future Baseline 2015 w/LCD, for Comparison to Alternative B
Annual Averages

	Maximum Concentrations in µg/m^3				Maximum Deposition Rates in µg/s/m^2 *			
TAP	@Fenceline	@Resident**	@School***	On Airport	@Fenceline	@Resident**	@School***	On Airport
Acetaldehyde	3.83E+00	6.96E-01	6.64E-01	8.17E+01	****	****	****	****
Acrolein	1.86E+00	3.38E-01	3.23E-01	3.99E+01	****	****	****	****
Benzene	1.97E+00	3.52E-01	3.32E-01	3.42E+01	****	****	****	****
1,3-Butadiene	1.56E+00	2.81E-01	2.68E-01	3.17E+01	****	****	****	****
Formaldehyde	1.24E+01	2.25E+00	2.14E+00	2.64E+02	****	****	****	****
Toluene	3.67E+00	1.72E-01	1.56E-01	9.22E+00	****	****	****	****
Benzo(a)anthracene	1.56E-04	1.42E-05	1.29E-05	5.99E-04	2.80E-07	2.55E-08	2.32E-08	1.08E-06
Benzo(a)pyrene	6.88E-05	7.28E-06	6.69E-06	2.53E-04	1.24E-07	1.31E-08	1.20E-08	4.56E-07
Benzo(b)fluoranthene	4.72E-05	3.66E-06	3.36E-06	1.84E-04	8.50E-08	6.58E-09	6.05E-09	3.30E-07
Benzo(k)fluoranthene	5.09E-05	3.90E-06	3.59E-06	1.98E-04	9.17E-08	7.03E-09	6.47E-09	3.57E-07
Chrysene	1.14E-04	9.42E-06	8.58E-06	4.47E-04	2.05E-07	1.69E-08	1.55E-08	8.05E-07
Dibenz(a,h)anthracene	2.21E-05	1.93E-06	1.80E-06	8.71E-05	3.98E-08	3.48E-09	3.24E-09	1.57E-07
Indeno(1,2,3-cd)pyrene	2.22E-05	1.94E-06	1.81E-06	8.79E-05	4.00E-08	3.49E-09	3.26E-09	1.58E-07
Arsenic	2.16E-01	4.74E-02	4.44E-02	1.16E+00	3.89E-04	8.54E-05	7.98E-05	2.09E-03
Cadmium	2.03E-02	4.49E-03	4.20E-03	1.09E-01	3.66E-05	8.07E-06	7.56E-06	1.97E-04
Chromium (Total)	2.16E-01	4.76E-02	4.45E-02	1.16E+00	3.89E-04	8.56E-05	8.01E-05	2.09E-03
Lead	2.24E-01	4.93E-02	4.61E-02	1.21E+00	4.04E-04	8.87E-05	8.29E-05	2.17E-03
Manganese	3.54E-04	2.57E-05	2.28E-05	7.41E-04	6.37E-07	4.62E-08	4.10E-08	1.33E-06
Nickel	2.04E-02	4.51E-03	4.22E-03	1.09E-01	3.67E-05	8.12E-06	7.59E-06	1.97E-04
Trichloroethylene*****	0.00E+00	0.00E+00	0.00E+00	0.00E+00	****	****	****	****

*Deposition velocity assumed to be 0.0018 m/s for non-volatile compounds.
**Maximum Impacts occur near the 1100 block of E. Acacia Ave., in El Segundo.
***Maximum Impacts at Imperial Ave. School.
****Volatile compounds, no significant deposition assumed to occur.
*****Trichloroethylene will not be used by Airport tenants in future operations.

Table B2-5

Comparison of Estimated Soil Concentrations from Particle Deposition with Background Concentrations for Soil

Element	Concentration in Soil at the Fenceline (Cs)	LN of Concentration in Soil at the Fenceline (Cs)	Concentration in Soil Onsite (Cs)	LN Concentration in Soil Onsite (Cs)	Background Geomean	Ln (Background)	GSD	Standardize Onsite	Onsite Percentile	Standardize Offsite	Offsite Percentile
	mg/Kg	mg/Kg	mg/Kg	mg/Kg							
Arsenic	4.85E-01	-7.25E-01	2.61E+00	9.58E-01	5.50E+00	1.70E+00	1.98E+00	-3.77E-01	35.30%	-1.23E+00	10.99%
Cadmium	4.57E-02	-3.09E+00	2.46E-01	-1.40E+00							
Chromium Hexavalent	1.14E-02	-4.47E+00	6.14E-02	-2.79E+00	4.10E+01	3.71E+00	2.19E+00	-2.97E+00	0.15%	-3.74E+00	0.01%
Chromium (Total)	4.85E-01	-7.24E-01	2.61E+00	9.58E-01	4.10E+01	3.71E+00	2.19E+00	-1.26E+00	10.41%	-2.03E+00	2.14%
Lead	5.03E-01	-6.87E-01	2.71E+00	9.95E-01	1.70E+01	2.83E+00	1.80E+00	-1.02E+00	15.36%	-1.96E+00	2.52%
Manganese	7.93E-04	-7.14E+00	1.66E-03	-6.40E+00			1.80E+00				
Nickel	4.57E-02	-3.08E+00	2.46E-01	-1.40E+00	1.50E+01	2.71E+00	2.10E+00	-1.96E+00	2.51%	-2.76E+00	0.29%
Benzo(a)anthracene	2.99E-03		1.15E-02								
Benzo(a)pyrene	3.52E-04		1.30E-03								
Benzo(b)fluoranthene	1.93E-03		7.48E-03								
Benzo(k)fluoranthene	2.08E-03		8.09E-03								
Chrysene	1.07E-03		4.21E-03								
Dibenz(a,h)anthracene	4.56E-04		1.79E-03								
Indeno(1,2,3-cd)pyrene	3.25E-04		1.29E-03								

Table B2-6

Calculation of Estimated Soil Concentrations from Particle Deposition

Cs=Dep x X/(Ks x SD x BD x Tt)
X = [{EXP(-Ks x Tt) - EXP(-Ks x To }/Ks] + Tt
Ks = 0.693/half-life

Element	Concentration in Soil at the Fenceline (Cs)	Concentration in Soil at the Residence (Cs)	Concentration in Soil Onsite (Cs)	Annual Air Concentration at the Fenceline	Annual Air Concentration at Residence	Annual Air Concentration Onsite	Deposition Velocity	CF	Deposition Rate at the Fenceline (Dep)	Deposition Rate at Residence (Dep)	Deposition Rate Onsite (Dep)	Half-life	Soil Elimination (Ks)	Soil Mixing Depth (SD)	Soil Bulk Density (BD)	Total Days (Tt)	To	X
	mg/Kg	mg/Kg	mg/Kg	ug/m^3	ug/m^3	ug/m^3	m/s	sec/day	ug/m^2/d	ug/m^2/d	ug/m^2/d		1/day	m	kg/m3	days	days	
Acetaldehyde	****	****	****	3.83E+00	6.96E-01	8.17E+01	0.0018	86400	****	****	****	NA	****	0.01	1000	36500	365	****
Acrolein	****	****	****	1.86E+00	3.38E-01	3.99E+01	0.0018	86400	****	****	****	NA	****	0.01	1000	36500	365	****
Benzene	****	****	****	1.97E+00	3.52E-01	3.42E+01	0.0018	86400	****	****	****	190.20	3.64E-03	0.01	1000	36500	365	3.64E+04
1,3-Butadiene	****	****	****	1.56E+00	2.81E-01	3.17E+01	0.0018	86400	****	****	****	NA	****	0.01	1000	36500	365	****
Formaldehyde	****	****	****	1.24E+01	2.25E+00	2.64E+02	0.0018	86400	****	****	****	NA	****	0.01	1000	36500	365	****
Toluene	****	****	****	3.67E+00	1.72E-01	9.22E+00	0.0018	86400	****	****	****	28.43	2.44E-02	0.01	1000	36500	365	3.65E+04
Benzo(a)anthracene	2.99E-03	2.72E-04	1.15E-02	1.56E-04	1.42E-05	5.99E-04	0.0018	86400	2.42E-02	2.20E-03	9.32E-02	878.29	7.89E-04	0.01	1000	36500	365	3.55E+04
Benzo(a)pyrene	3.52E-04	3.73E-05	1.30E-03	6.88E-05	7.28E-06	2.53E-04	0.0018	86400	1.07E-02	1.13E-03	3.94E-02	228.63	3.03E-03	0.01	1000	36500	365	3.64E+04
Benzo(b)fluoranthene	1.93E-03	1.49E-04	7.48E-03	4.72E-05	3.66E-06	1.84E-04	0.0018	86400	7.35E-03	5.69E-04	2.85E-02	1,948.62	3.56E-04	0.01	1000	36500	365	3.40E+04
Benzo(k)fluoranthene	2.08E-03	1.59E-04	8.09E-03	5.09E-05	3.90E-06	1.98E-04	0.0018	86400	7.92E-03	6.07E-04	3.08E-02	1,948.62	3.56E-04	0.01	1000	36500	365	3.40E+04
Chrysene	1.07E-03	8.86E-05	4.21E-03	1.14E-04	9.42E-06	4.47E-04	0.0018	86400	1.77E-02	1.46E-03	6.95E-02	423.39	1.64E-03	0.01	1000	36500	365	3.62E+04
Dibenz(a,h)anthracene	4.56E-04	3.98E-05	1.79E-03	2.21E-05	1.93E-06	8.71E-05	0.0018	86400	3.44E-03	3.01E-04	1.35E-02	944.89	7.33E-04	0.01	1000	36500	365	3.55E+04
Indeno(1,2,3-cd)pyrene	3.25E-04	2.84E-05	1.29E-03	2.22E-05	1.94E-06	8.79E-05	0.0018	86400	3.45E-03	3.02E-04	1.37E-02	665.00	1.04E-03	0.01	1000	36500	365	3.58E+04
Arsenic	4.85E-01	1.06E-01	2.61E+00	2.16E-01	4.74E-02	1.16E+00	0.0018	86400	3.36E+01	7.38E+00	1.81E+02	100.00	6.93E-03	0.01	1000	36500	365	3.65E+04
Cadmium	4.57E-02	1.01E-02	2.46E-01	2.03E-02	4.49E-03	1.09E-01	0.0018	86400	3.16E+00	6.98E-01	1.70E+01	100.00	6.93E-03	0.01	1000	36500	365	3.65E+04
Chromium Hexavalent	1.14E-02	2.52E-03	6.14E-02	5.09E-03	1.12E-03	2.74E-02	0.0018	86400	7.91E-01	1.74E-01	4.26E+00	100.00	6.93E-03	0.01	1000	36500	365	3.65E+04
Chromium (Total)	4.85E-01	1.07E-01	2.61E+00	2.16E-01	4.76E-02	1.16E+00	0.0018	86400	3.36E+01	7.40E+00	1.81E+02	100.00	6.93E-03	0.01	1000	36500	365	3.65E+04
Lead	5.03E-01	1.10E-01	2.71E+00	2.24E-01	4.93E-02	1.21E+00	0.0018	86400	3.49E+01	7.66E+00	1.88E+02	100.00	6.93E-03	0.01	1000	36500	365	3.65E+04
Manganese	7.93E-04	5.76E-05	1.66E-03	3.54E-04	2.57E-05	7.41E-04	0.0018	86400	5.50E-02	3.99E-03	1.15E-01	100.00	6.93E-03	0.01	1000	36500	365	3.65E+04
Nickel	4.57E-02	1.01E-02	2.46E-01	2.04E-02	4.51E-03	1.09E-01	0.0018	86400	3.17E+00	7.02E-01	1.70E+01	100.00	6.93E-03	0.01	1000	36500	365	3.65E+04

NA = Not Available
**** = Not Calculated

Table B2-7

Calculations for TCDD Impacts to Soils on and near LAX

	Soil Concentration	Deposition Rate	Soil Elimination	Soil Mixing Depth	Soil Bulk Density	Total Days	X
	ug/kg	ug/m^2/d	d	m	kg/m^3	d	
On-LAX	0	0	0.000693	0.01	1600	36500	35379.49
Fence	0	0	0.000693	0.01	1600	36500	35379.49
Bckgrnd1	479.0078912	5.479452055	0.000693	0.01	1600	36500	35379.49
Bckgrnd2	1437.023673	16.43835616	0.000693	0.01	1600	36500	35379.49

Background deposition 2-6 ng/m^2/yr From EPA 1994

Rate ng/m^2/y	ng/m^2/d	ug/m^2/d
2	0.005479452	5.479452055
6	0.016438356	16.43835616

Attachment B3
Emissions Estimates Spreadsheets

2015 Future Baseline Air Pollutant Emissions, lbs/year

Source Category	CO	THC	VOC	NOx	SO_2	PM_{10}	Acetaldehyde	Acrolein	Benzene	1,3-Butadiene	Formaldehyde	Hexane	Propylene	Propylene oxide	Styrene	Toluene	Xylene, m- or p-	Xylene, o-	Xylene (total)	Acenaphthene	Acenaphthylene	Anthracene	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(g,h,i)perylene	Benzo(k)fluoranthene	Chrysene	Dibenz(a,h)anthracene
Aircraft:																													
Comm. Jet (TF)																													
APU	113,656	4,160	4,080	63,256	0		3.10E+01	4.73E+01	1.22E+02	6.53E+01	1.16E+02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.73E-03	1.29E-02	4.66E-03	1.04E-02	5.73E-03	9.69E-03	3.55E-02	0.00E+00
Taxi/Idle	9,594,200	1,716,952	1,879,547	1,389,104	178,724	202,973	9.75E+04	4.76E+04	4.07E+04	3.78E+04	3.15E+05	0.00E+00	1.07E+05	0.00E+00	8.27E+03	1.08E+04	6.03E+03	3.95E+03	9.98E+03	0.00E+00	0.00E+00	7.52E-01	9.93E-02	5.28E-02	0.00E+00	8.77E-03	0.00E+00	1.06E-01	0.00E+00
Takeoff	49,304	15,496	16,963	2,831,782	50,426	57,268	8.80E+02	4.29E+02	3.67E+02	3.41E+02	2.84E+03	0.00E+00	9.66E+02	0.00E+00	7.46E+01	9.75E+01	5.44E+01	3.56E+01	9.00E+01	0.00E+00	0.00E+00	2.48E-02	1.77E-02	1.27E-02	0.00E+00	2.84E-03	0.00E+00	3.42E-03	0.00E+00
Climbout	74,812	21,064	23,059	3,009,764	66,942	76,025	1.20E+03	5.83E+02	5.00E+02	4.63E+02	3.87E+03	0.00E+00	1.31E+03	0.00E+00	1.01E+02	1.33E+02	7.39E+01	4.84E+01	1.22E+02	0.00E+00	0.00E+00	3.37E-02	2.41E-02	1.72E-02	0.00E+00	3.86E-03	0.00E+00	4.65E-03	0.00E+00
Approach	257,246	31,410	34,385	925,490	49,322	56,014	1.78E+03	8.70E+02	7.45E+02	6.91E+02	5.76E+03	0.00E+00	1.96E+03	0.00E+00	1.51E+02	1.98E+02	1.10E+02	7.22E+01	1.83E+02	0.00E+00	0.00E+00	3.76E-02	1.39E-01	8.76E-02	0.00E+00	1.23E-02	0.00E+00	1.47E-02	0.00E+00
Eng. Testing	45,967	7,081	7,752	258,174	5	6	4.02E+02	1.96E+02	1.68E+02	1.56E+02	1.30E+03	0.00E+00	4.41E+02	0.00E+00	3.41E+01	4.46E+01	2.49E+01	1.63E+01	4.11E+01	0.00E+00	0.00E+00	8.56E-03	1.20E-02	7.88E-03	0.00E+00	1.35E-03	0.00E+00	1.72E-03	0.00E+00
AT/GA Turbine (TP)																													
APU	0	0	0	0	0		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Aircraft	524,830	49,582	52,711	189,876	16,270	61	2.58E+03	1.23E+03	1.07E+03	9.39E+02	8.46E+03	0.00E+00	0.00E+00	0.00E+00	2.21E+02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.81E-02	2.69E-02	1.70E-02	0.00E+00	2.61E-03	0.00E+00	5.38E-03	0.00E+00
AT/GA Piston (P)			0				0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Aircraft Total, lbs/yr	10,660,015	1,845,745	2,018,496	8,667,446	361,689	392,347	1.04E+05	5.09E+04	4.37E+04	4.04E+04	3.37E+05	0.00E+00	1.12E+05	0.00E+00	8.85E+03	1.13E+04	6.29E+03	4.12E+03	1.04E+04	0.00E+00	0.00E+00	8.90E-01	3.32E-01	2.00E-01	1.04E-02	3.74E-02	9.69E-03	1.71E-01	0.00E+00
Aircraft Total, tpy	5,330	923	1,009	4,334	181	196	5.22E+01	2.55E+01	2.18E+01	2.02E+01	1.69E+02	0.00E+00	5.59E+01	0.00E+00	4.43E+00	5.64E+00	3.15E+00	2.06E+00	5.21E+00	0.00E+00	0.00E+00	4.45E-04	1.66E-04	9.99E-05	5.20E-06	1.87E-05	4.85E-06	8.55E-05	0.00E+00
GSE																													
Diesel	73,284	73,052	71,640	507,940	13,320	28,990	1.06E+01	3.27E+00	5.90E+02	1.15E+03	1.54E+02	0.00E+00	1.04E+03	0.00E+00	0.00E+00	1.87E+02	0.00E+00	0.00E+00	6.40E+01	2.05E+00	3.11E+00	6.81E-01	2.94E-01	2.42E-01	3.42E-01	2.76E-01	2.39E-01	3.96E-01	2.51E-01
Gasoline	1,275,618	70,642	63,970	53,584	1,114	2,756	1.41E+02	4.48E+01	3.19E+03	5.12E+02	6.52E+02	0.00E+00	0.00E+00	0.00E+00	1.92E+01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-02	1.74E-01	1.02E-01	8.91E-02	3.42E-01	9.63E-02	1.31E-01	1.95E-02
LPG																													
CNG/LNG																													
GSE Total, lbs/yr	1,348,902	143,694	1.36E+05	561,524	14,434	31,746	1.51E+02	4.80E+01	3.78E+03	1.66E+03	8.07E+02	0.00E+00	1.04E+03	0.00E+00	1.92E+01	1.87E+02	0.00E+00	0.00E+00	6.40E+01	2.05E+00	3.11E+00	6.94E-01	4.68E-01	3.44E-01	4.31E-01	6.19E-01	3.36E-01	5.27E-01	2.70E-01
GSE Total, tpy	674	72	68	281	7	16	7.57E-02	2.40E-02	1.89E+00	8.29E-01	4.03E-01	0.00E+00	5.21E-01	0.00E+00	9.60E-03	9.35E-02	0.00E+00	0.00E+00	3.20E-02	1.03E-03	1.55E-03	3.47E-04	2.34E-04	1.72E-04	2.15E-04	3.09E-04	1.68E-04	2.63E-04	1.35E-04
Stationary Total lbs/yr	445,955	222,269	222,269	702,482	10,882	105,820	1.07E+02	2.80E+01	7.24E+02	3.78E+01	4.44E+02	2.17E+02	1.59E+03	4.01E+01	1.70E+01	9.02E+02			7.59E+02	1.57E+00	1.94E+01	1.48E+00	1.86E+00	4.47E-01	6.60E-01	4.58E-01	2.78E-01	1.20E+00	1.12E-01
Stationary Total, tpy	223	111	111	351	5	53	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Motor Vehicle, On Airport																													
Diesel					1,043																								
Running Exhaust	5,583	5,463	5,358	31,155		211	4.07E+01	6.22E+01	1.61E+02	8.57E+01	1.53E+02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.53E-03	1.69E-02	6.12E-03	1.36E-02	7.53E-03	1.27E-02	4.66E-02	0.00E+00
Start Exhaust	242	0	0	0			0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Gasoline					1,728								5.12E+02																
Running Exhaust	523,066	23,357	21,151	57,095		3,702	1.17E+02	1.48E+01	9.58E+02	1.17E+02	2.57E+02	0.00E+00	5.12E+02	0.00E+00	8.67E+01	2.43E+03	0.00E+00	0.00E+00	1.37E+03	0.00E+00	0.00E+00	6.85E-04	9.71E-03	5.71E-03	4.97E-03	1.91E-02	5.37E-03	7.31E-03	1.09E-03
Start Exhaust	214,357	16,961	15,359	29,546			8.48E+01	1.08E+01	6.95E+02	8.48E+01	1.87E+02	0.00E+00	3.71E+02	0.00E+00	6.30E+01	1.76E+03	0.00E+00	0.00E+00	9.94E+02	0.00E+00	0.00E+00	4.98E-04	7.05E-03	4.15E-03	3.61E-03	1.39E-02	3.90E-03	5.31E-03	7.88E-04
Evaporation		15,389	13,935				0.00E+00	0.00E+00	1.07E+02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.20E+01	0.00E+00	0.00E+00	2.09E+01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
MV, On Airport Total, lb/yr	743,249	61,170	55,803	117,796	2,771	3,913	2.42E+02	8.77E+01	1.92E+03	2.87E+02	5.96E+02	0.00E+00	1.39E+03	0.00E+00	1.50E+02	4.29E+03	0.00E+00	0.00E+00	2.38E+03	0.00E+00	0.00E+00	8.71E-03	3.37E-02	1.60E-02	2.22E-02	4.05E-02	2.20E-02	5.92E-02	1.87E-03
MV, On Airport Total, tpy	372	31	28	59	1	2	1.21E-01	4.39E-02	9.61E-01	1.44E-01	2.98E-01	0.00E+00	6.97E-01	0.00E+00	7.48E-02	2.14E+00	0.00E+00	0.00E+00	1.19E+00	0.00E+00	0.00E+00	4.35E-06	1.69E-05	7.99E-06	1.11E-05	2.02E-05	1.10E-05	2.96E-05	9.36E-07
Motor Vehicle, Off Airport					54,933	58,090																							
Running Exhaust	5,460,631	237,066	214,675	1,067,882			1.19E+03	1.50E+02	9.72E+03	1.19E+03	2.61E+03	0.00E+00	5.19E+03	0.00E+00	8.80E+02	2.47E+04	0.00E+00	0.00E+00	1.39E+04	0.00E+00	0.00E+00	6.96E-03	9.85E-02	5.80E-02	5.04E-02	1.94E-01	5.45E-02	7.42E-02	1.10E-02
Start Exhaust	494,292	24,089	21,814	33,341			1.20E+02	1.53E+01	9.88E+02	1.20E+02	2.65E+02	0.00E+00	5.28E+02	0.00E+00	8.94E+01	2.51E+03	0.00E+00	0.00E+00	1.41E+03	0.00E+00	0.00E+00	7.07E-04	1.00E-02	5.89E-03	5.12E-03	1.97E-02	5.54E-03	7.54E-03	1.12E-03
Evaporation		1,056,968	957,138				0.00E+00	0.00E+00	7.37E+03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.32E+03	0.00E+00	0.00E+00	1.44E+03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
MV, Off Airport Total, lb/yr	5,954,923	1,318,123	1,193,627	1,101,223	54,933	58,090	1.31E+03	1.66E+02	1.81E+04	1.31E+03	2.87E+03	0.00E+00	5.72E+03	0.00E+00	9.70E+02	3.35E+04	0.00E+00	0.00E+00	1.67E+04	0.00E+00	0.00E+00	7.66E-03	1.09E-01	6.39E-02	5.56E-02	2.13E-01	6.00E-02	8.17E-02	1.21E-02
MV, Off Airport Total, tpy	2,977	659	597	551	27	29	6.53E-01	8.28E-02	9.04E+00	6.53E-01	1.44E+00	0.00E+00	2.86E+00	0.00E+00	4.85E-01	1.67E+01	0.00E+00	0.00E+00	8.37E+00	0.00E+00	0.00E+00	3.83E-06	5.43E-05	3.19E-05	2.78E-05	1.07E-04	3.00E-05	4.09E-05	6.07E-06
Motor Vehicles, Parking																													
MV, Parking, lb/yr	506,402	103,172	93,427	36,709		549	5.16E+02	6.54E+01	4.23E+03	5.16E+02	1.13E+03	0.00E+00	2.26E+03	0.00E+00	3.83E+02	1.07E+04	0.00E+00	0.00E+00	6.05E+03	0.00E+00	0.00E+00	3.03E-03	4.29E-02	2.52E-02	2.19E-02	8.43E-02	2.37E-02	3.23E-02	4.79E-03
MV, Parking, tpy	253	52	47	18		0	2.58E-01	3.27E-02	2.12E+00	2.58E-01	5.67E-01	0.00E+00	1.13E+00	0.00E+00	1.92E-01	5.36E+00	0.00E+00	0.00E+00	3.02E+00	0.00E+00	0.00E+00	1.51E-06	2.14E-05	1.26E-05	1.10E-05	4.21E-05	1.19E-05	1.61E-05	2.40E-06
Fugitive Dust																													
Runways						46,914																							
Roadways, On-Airport						543,791																							
Roadways, Off-Airport																													
Fugitive Dust, Total lbs/yr						590,705																							
Fugitive Dust, Total tpy						295																							
Total Operating, lbs/yr	19,659,445	3,694,172	3,719,233	11,187,180	444,710	1,183,170	1.07E+05	5.13E+04	7.24E+04	4.42E+04	3.43E+05	2.17E+02	1.24E+05	4.01E+01	1.04E+04	6.09E+04	6.29E+03	4.12E+03	3.64E+04	3.62E+00	2.25E+01	3.08E+00	2.84E+00	1.10E+00	1.20E+00	1.45E+00	7.29E-01	2.07E+00	4.01E-01
Total Operating, tpy	9,830	1,847	1,860	5,594	222	592	5.34E+01	2.57E+01	3.62E+01	2.21E+01	1.72E+02	1.09E-01	6.19E+01	2.00E-02	5.20E+00	3.04E+01	3.15E+00	2.06E+00	1.82E+01	1.81E-03	1.13E-02	1.54E-03	1.42E-03	5.48E-04	6.00E-04	7.26E-04	3.64E-04	1.04E-03	2.00E-04
Total Operating, kg/yr	8.93E+06	1.68E+06	1.69E+06	5.08E+06	2.02E+05	5.37E+05	4.85E+04	2.33E+04	3.29E+04	2.01E+04	1.56E+05	9.86E+01	5.62E+04	1.82E+01	4.72E+03	2.76E+04	2.86E+03	1.87E+03	1.65E+04	1.64E+00	1.02E+01	1.40E+00	1.29E+00	4.98E-01	5.45E-01	6.59E-01	3.31E-01	9.41E-01	1.82E-01
Acquisition Areas																													
Alternative 1, lbs/yr	57,357	15,202	15,202	349,119	0	573	1.32E+01	1.29E+01	6.71E+00	0.00E+00	1.36E+01	0.00E+00	1.33E+03	0.00E+00	0.00E+00	9.26E+01	0.00E+00	0.00E+00	5.36E+01	3.99E-03	3.47E-02	4.62E-03	4.04E-03	3.27E-03	3.27E-03	3.58E-03	3.27E-03	3.99E-03	3.27E-03
Alternative 2, lbs/yr	144,045	38,047	38,047	861,687	0	1,435	3.32E+01	3.25E+01	1.69E+01	0.00E+00	3.42E+01	0.00E+00	3.34E+03	0.00E+00	0.00E+00	2.33E+02	0.00E+00	0.00E+00	1.35E+02	1.00E-02	8.72E-02	1.16E-02	1.02E-02	8.21E-03	8.21E-03	9.01E-03	8.21E-03	1.00E-02	8.21E-03
Alternative 3, lbs/yr	89,659	23,762	23,762	531,581	0	895	2.07E+01	2.02E+01	1.05E+01	0.00E+00	2.13E+01	0.00E+00	2.08E+03	0.00E+00	0.00E+00	1.45E+02	0.00E+00	0.00E+00	8.38E+01	6.23E-03	5.42E-02	7.22E-03	6.32E-03	5.11E-03	5.11E-03	5.60E-03	5.11E-03	6.23E-03	5.11E-03
Chrome Plating																													
LCD																													
Motor Vehicle					6,497	9,027																							
Running Exhaust	1,019,310	42,401	38,396	143,529			2.12E+02	2.69E+01	1.74E+03	2.12E+02	4.66E+02	0.00E+00	9.29E+02	0.00E+00	1.57E+02	4.41E+03	0.00E+00	0.00E+00	2.48E+03	0.00E+00	0.00E+00	1.24E-03	1.76E-02	1.04E-02	9.02E-03	3.46E-02	9.74E-03	1.33E-02	1.97E-03
Start Exhaust	199,406	9,771	8,848	143,529			4.89E+01	6.19E+00	4.01E+02	4.89E+01	1.07E+02	0.00E+00	2.14E+02	0.00E+00	3.63E+01	1.02E+03	0.00E+00	0.00E+00	5.73E+02	0.00E+00	0.00E+00	2.87E-04	4.06E-03	2.39E-03	2.08E-03	7.98E-03	2.25E-03	3.06E-03	4.54E-04
Evaporation		274,488	248,563				0.00E+00	0.00E+00	1.91E+03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.64E+03	0.00E+00	0.00E+00	3.73E+02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Northside - NG Use	2,903	770	770	17,420	0	30	6.69E-01	6.55E-01	3.40E-01	0.00E+00	6.90E-01	0.00E+00	6.72E+01	0.00E+00	0.00E+00	4.69E+00	0.00E+00	0.00E+00	2.71E+00	2.02E-04	1.76E-03	2.34E-04	2.05E-04	1.65E-04	1.65E-04	1.81E-04	1.65E-04	2.02E-04	1.65E-04
Continental City - NG Use	1,510	400	400	9,058	0	15	3.48E-01	3.40E-01	1.77E-01	0.00E+00	3.59E-01	0.00E+00	3.49E+01	0.00E+00	0.00E+00	2.44E+00	0.00E+00	0.00E+00	1.41E+00	1.05E-04	9.13E-04	1.22E-04	1.06E-04	8.60E-05	8.60E-05	9.44E-05	8.60E-05	1.05E-04	8.60E-05
LCD, Total lbs/yr	1,223,129	327,830	296,977	313,535	6,497	9,072	262	34	4,054	261	575	0	1,245	0	194	7,074	0	0	3,434	0	0	0	0	0	0	0	0	0	0
LCD, Total tpy	612	164	148	157	3	5	0	0	2	0	0	0	1	0	0	4	0	0	2	0	0	0	0	0	0	0	0	0	0

2015 Future Baseline Air Po																													
Source Category	Fluoranthene	Fluorene	Indeno(1,2,3-cd)pyrene	Naphthalene	Phenanthrene	Pyrene	2,3,7,8 TCDD Equivalents	Dioxin (tetrachloro total)	Dioxin (pentachloro total)	Dioxin (hexachloro total)	Dioxin (heptachloro total)	Dioxin (octachloro)	Furan (tetrachloro total)	Furan (pentachloro total)	Furan (hexachloro total)	Furan (heptachloro total)	Furan (octachloro)	Antimony	Arsenic	Beryllium	Cadmium	Chromium Hexavalent	Chromium (Total)	Cobalt	Copper	Lead	Manganese	Mercury	Nickel
Aircraft:																													
Comm. Jet (TF)																													
APU	4.66E-02	0.00E+00	0.00E+00	0.00E+00	4.37E-02	8.10E-02																							
Taxi/Idle	1.55E+00	0.00E+00	0.00E+00	8.11E+02	6.85E+00	1.90E+00		8.64E-05	1.65E-04	2.08E-04	3.88E-04	2.47E-03	7.71E-04	1.08E-03	5.57E-04	3.86E-04	1.99E-04	0.00E+00	1.08E+03	0.00E+00	1.01E+02	0.00E+00	1.08E+03	0.00E+00	0.00E+00	1.12E+03	0.00E+00	0.00E+00	1.01E+02
Takeoff	7.71E-02	0.00E+00	0.00E+00	8.05E-01	5.23E-01	6.17E-02		2.25E-05	4.31E-05	5.42E-05	1.01E-04	6.45E-04	2.01E-04	2.81E-04	1.45E-04	1.01E-04	5.19E-05	0.00E+00	3.04E+02	0.00E+00	2.86E+01	0.00E+00	3.04E+02	0.00E+00	0.00E+00	3.15E+02	0.00E+00	0.00E+00	2.86E+01
Climbout	1.05E-01	0.00E+00	0.00E+00	1.09E+00	7.11E-01	8.39E-02		3.05E-05	5.82E-05	7.33E-05	1.37E-04	8.72E-04	2.72E-04	3.80E-04	1.96E-04	1.36E-04	7.01E-05	0.00E+00	4.03E+02	0.00E+00	3.80E+01	0.00E+00	4.03E+02	0.00E+00	0.00E+00	4.18E+02	0.00E+00	0.00E+00	3.80E+01
Approach	2.83E-01	0.00E+00	0.00E+00	2.74E+00	1.22E+00	2.33E-01		2.37E-05	4.52E-05	5.70E-05	1.06E-04	6.77E-04	2.11E-04	2.96E-04	1.53E-04	1.06E-04	5.45E-05	0.00E+00	2.97E+02	0.00E+00	2.80E+01	0.00E+00	2.97E+02	0.00E+00	0.00E+00	3.08E+02	0.00E+00	0.00E+00	2.80E+01
Eng. Testing	3.52E-02	0.00E+00	0.00E+00	1.17E+00	1.95E-01	2.92E-02		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.30E-02	0.00E+00	3.12E-03	0.00E+00	3.30E-02	0.00E+00	0.00E+00	3.43E-02	0.00E+00	0.00E+00	3.12E-03
AT/GA Turbine (TP)																													
APU	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00																							
Aircraft	9.43E-02	0.00E+00	0.00E+00	1.97E+01	4.46E-01	9.20E-02												0.00E+00	3.23E-01	0.00E+00	3.04E-02	0.00E+00	3.23E-01	0.00E+00	0.00E+00	3.35E-01	0.00E+00	0.00E+00	3.04E-02
AT/GA Piston (P)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00												0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Aircraft Total, lbs/yr	2.19E+00	0.00E+00	0.00E+00	8.36E+02	9.98E+00	2.48E+00	7.30E-04	1.63E-04	3.12E-04	3.92E-04	7.32E-04	4.66E-03	1.46E-03	2.04E-03	1.05E-03	7.28E-04	3.75E-04	0.00E+00	2.08E+03	0.00E+00	1.96E+02	0.00E+00	2.08E+03	0.00E+00	0.00E+00	2.16E+03	0.00E+00	0.00E+00	1.96E+02
Aircraft Total, tpy	1.10E-03	0.00E+00	0.00E+00	4.18E-01	4.99E-03	1.24E-03	3.65E-07	8.15E-08	1.56E-07	1.96E-07	3.66E-07	2.33E-06	7.28E-07	1.02E-06	5.25E-07	3.64E-07	1.88E-07	0.00E+00	1.04E+00	0.00E+00	9.81E-02	0.00E+00	1.04E+00	0.00E+00	0.00E+00	1.08E+00	0.00E+00	0.00E+00	9.81E-02
GSE																													
Diesel	1.00E+00	2.95E+00	2.58E-01	4.87E+01	1.08E+01	8.05E-01												0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.03E+00	0.00E+00	1.45E+00	0.00E+00	2.03E+00	5.80E-01	8.70E-01
Gasoline	1.14E-01	0.00E+00	1.54E-02	0.00E+00	4.10E-02	7.27E-02												0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.65E-01	0.00E+00	4.41E-01	0.00E+00	3.31E-01	2.76E-02	1.93E-01
LPG																													
CNG/LNG																													
GSE Total, lbs/yr	1.12E+00	2.95E+00	2.73E-01	4.87E+01	1.08E+01	8.78E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.19E+00	0.00E+00	1.89E+00	0.00E+00	2.36E+00	6.07E-01	1.06E+00
GSE Total, tpy	5.59E-04	1.47E-03	1.37E-04	2.44E-02	5.42E-03	4.39E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.10E-03	0.00E+00	9.45E-04	0.00E+00	1.18E-03	3.04E-04	5.31E-04
Stationary Total lbs/yr	2.13E+00	3.12E+00	1.14E-01	5.45E+01	1.07E+01	2.39E+00	6.84E-07											0.00E+00	3.68E-02	9.89E-03	1.05E+01	1.97E-03	5.44E+01		1.82E-01	1.11E-01	1.88E+00	4.94E-04	8.89E+00
Stationary Total, tpy	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Motor Vehicle, On Airport																													
Diesel																													
Running Exhaust	6.12E-02	0.00E+00	0.00E+00	0.00E+00	5.74E-02	1.06E-01		3.53E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.74E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.48E-02	0.00E+00	1.06E-02	0.00E+00	1.48E-02	4.22E-03	6.34E-03
Start Exhaust	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00																							
Gasoline																													
Running Exhaust	6.34E-03	0.00E+00	8.57E-04	4.67E+01	2.28E-03	4.05E-03		2.96E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.81E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.22E-01	0.00E+00	5.92E-01	0.00E+00	4.44E-01	3.70E-02	2.59E-01
Start Exhaust	4.60E-03	0.00E+00	6.22E-04	3.39E+01	1.66E-03	2.94E-03																							
Evaporation	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00																							
MV, On Airport Total, lb/yr	7.21E-02	0.00E+00	1.48E-03	8.06E+01	6.13E-02	1.13E-01	7.86E-07	2.99E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.87E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.37E-01	0.00E+00	6.03E-01	0.00E+00	4.59E-01	4.12E-02	2.65E-01
MV, On Airport Total, tpy	3.61E-05	0.00E+00	7.39E-07	4.03E-02	3.07E-05	5.67E-05	3.93E-10	1.50E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.43E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.18E-04	0.00E+00	3.01E-04	0.00E+00	2.30E-04	2.06E-05	1.33E-04
Motor Vehicle, Off Airport								6.41E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.04E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.49E+00	0.00E+00	9.29E+00	0.00E+00	6.97E+00	5.81E-01	4.07E+00
Running Exhaust	6.43E-02	0.00E+00	8.69E-03	4.74E+02	2.32E-02	4.12E-02																							
Start Exhaust	6.54E-03	0.00E+00	8.83E-04	4.82E+01	2.36E-03	4.18E-03																							
Evaporation	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00																							
MV, Off Airport Total, lb/yr	7.09E-02	0.00E+00	9.58E-03	5.22E+02	2.55E-02	4.53E-02	1.68E-05	6.41E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.04E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.49E+00	0.00E+00	9.29E+00	0.00E+00	6.97E+00	5.81E-01	4.07E+00
MV, Off Airport Total, tpy	3.54E-05	0.00E+00	4.79E-06	2.61E-01	1.28E-05	2.27E-05	8.41E-09	3.20E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.20E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.74E-03	0.00E+00	4.65E-03	0.00E+00	3.49E-03	2.90E-04	2.03E-03
Motor Vehicles, Parking																													
MV, Parking, lb/yr	2.80E-02	0.00E+00	3.78E-03	2.06E+02	1.01E-02	1.79E-02	1.32E-06	5.01E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.15E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.29E-02	0.00E+00	8.78E-02	0.00E+00	6.58E-02	5.49E-03	3.84E-02
MV, Parking, tpy	1.40E-05	0.00E+00	1.89E-06	1.03E-01	5.05E-06	8.96E-06	6.58E-10	2.51E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.07E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.65E-05	0.00E+00	4.39E-05	0.00E+00	3.29E-05	2.74E-06	1.92E-05
Fugitive Dust																													
Runways																		0.00E+00	4.69E-01	0.00E+00	1.41E+00	0.00E+00	1.03E+01	0.00E+00	1.69E+01	7.06E+02	4.79E+01	0.00E+00	9.38E+00
Roadways, On-Airport																		0.00E+00	5.44E+00	0.00E+00	1.63E+01	0.00E+00	1.20E+02	0.00E+00	1.96E+02	8.18E+03	5.55E+02	0.00E+00	1.09E+02
Roadways, Off-Airport																		0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Fugitive Dust, Total lbs/yr							0.00E+00											0.00E+00	5.91E+00	0.00E+00	1.77E+01	0.00E+00	1.30E+02	0.00E+00	2.13E+02	8.89E+03	6.03E+02	0.00E+00	1.18E+02
Fugitive Dust, Total tpy																		0.00E+00	2.95E-03	0.00E+00	8.86E-03	0.00E+00	6.50E-02	0.00E+00	1.06E-01	4.45E+00	3.01E-01	0.00E+00	5.91E-02
Total Operating, lbs/yr	5.61E+00	6.07E+00	4.02E-01	1.75E+03	3.16E+01	5.92E+00	7.50E-04	1.70E-04	3.12E-04	3.92E-04	7.32E-04	4.66E-03	1.57E-03	2.04E-03	1.05E-03	7.28E-04	3.75E-04	0.00E+00	2.09E+03	9.89E-03	2.24E+02	1.97E-03	2.27E+03	0.00E+00	2.25E+02	1.10E+04	6.14E+02	1.24E+00	3.29E+02
Total Operating, tpy	2.80E-03	3.04E-03	2.01E-04	8.74E-01	1.58E-02	2.96E-03	3.75E-07	8.51E-08	1.56E-07	1.96E-07	3.66E-07	2.33E-06	7.87E-07	1.02E-06	5.25E-07	3.64E-07	1.88E-07	0.00E+00	1.04E+00	4.94E-06	1.12E-01	9.83E-07	1.13E+00	0.00E+00	1.12E-01	5.52E+00	3.07E-01	6.18E-04	1.64E-01
Total Operating, kg/yr	2.55E+00	2.76E+00	1.83E-01	7.94E+02	1.43E+01	2.69E+00	3.40E-04	7.73E-05	1.42E-04	1.78E-04	3.32E-04	2.12E-03	7.14E-04	9.24E-04	4.77E-04	3.30E-04	1.70E-04	0.00E+00	9.47E+02	4.49E-03	1.02E+02	8.93E-04	1.03E+03	0.00E+00	1.02E+02	5.02E+03	2.79E+02	5.61E-01	1.49E+02
Acquisition Areas																													
Alternative 1, lbs/yr	3.41E-02	1.32E-02	3.27E-03	6.80E-01	9.66E-02	1.61E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Alternative 2, lbs/yr	8.57E-02	3.31E-02	8.21E-03	1.71E+00	2.43E-01	4.04E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Alternative 3, lbs/yr	5.33E-02	2.06E-02	5.11E-03	1.06E+00	1.51E-01	2.51E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chrome Plating																													
LCD																													
Motor Vehicle								6.41E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.04E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.42E-01	0.00E+00	1.44E+00	0.00E+00	1.08E+00	9.03E-02	6.32E-01
Running Exhaust	1.15E-02	0.00E+00	1.56E-03	8.48E+01	4.15E-03	7.36E-03																							
Start Exhaust	2.65E-03	0.00E+00	3.58E-04	1.95E+01	9.56E-04	1.70E-03																							
Evaporation	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00																							
Northside - NG Use	1.73E-03	6.66E-04	1.65E-04	3.44E-02	4.89E-03	8.13E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Continental City - NG Use	8.98E-04	3.46E-04	8.60E-05	1.79E-02	2.54E-03	4.23E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
LCD, Total lbs/yr	0	0	0	104	0	0	1.68E-05	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0	1	0	1
LCD, Total tpy	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

2015 Future Baseline Air Po

Source Category	Phosphorus	Selenium	Zinc	Ammonia	Bromine	Hydrogen chloride	Benzaldehyde	N-Butanol	Secondary Butanol	Ethyl Alcohol	Ethylbenzene	Ethylene Glycol Ethers	Hydrogen fluoride	Methanol	Methyl ethyl ketone	Methylene Chloride	Perchloroethylene	Trichloroethane, 1,1,1	Trichloroethylene
Aircraft:																			
Comm. Jet (TF)																			
APU																			
Taxi/Idle	0.00E+00	1.01E+02	1.12E+03	0.00E+00	0.00E+00	0.00E+00													
Takeoff	0.00E+00	2.86E+01	3.15E+02	0.00E+00	0.00E+00	0.00E+00													
Climbout	0.00E+00	3.80E+01	4.18E+02	0.00E+00	0.00E+00	0.00E+00													
Approach	0.00E+00	2.80E+01	3.08E+02	0.00E+00	0.00E+00	0.00E+00													
Eng. Testing	0.00E+00	3.12E-03	3.43E-02	0.00E+00	0.00E+00	0.00E+00													
AT/GA Turbine (TP)																			
APU																			
Aircraft	0.00E+00	3.04E-02	3.35E-01	0.00E+00	0.00E+00	0.00E+00													
AT/GA Piston (P)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
Aircraft Total, lbs/yr	0.00E+00	1.96E+02	2.16E+03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Aircraft Total, tpy	0.00E+00	9.81E-02	1.08E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
GSE																			
Diesel	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
Gasoline	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
LPG																			
CNG/LNG																			
GSE Total, lbs/yr	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
GSE Total, tpy	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Stationary Total lbs/yr		1.53E-03					1.34E+01	5.96E+01	1.96E+00	1.39E+03	1.55E+02	2.88E+02	3.91E+01	1.17E+04	3.14E+02	9.51E+02	1.81E+02	4.16E+02	0.00E+00
Stationary Total, tpy	0	0	0	0	0	0	0	0	0	1	0	0	0	6	0	0	0	0	0
Motor Vehicle, On Airport																			
Diesel																			
Running Exhaust	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
Start Exhaust																			
Gasoline																			
Running Exhaust	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
Start Exhaust																			
Evaporation																			
MV, On Airport Total, lb/yr	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
MV, On Airport Total, tpy	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Motor Vehicle, Off Airport	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
Running Exhaust																			
Start Exhaust																			
Evaporation																			
MV, Off Airport Total, lb/yr	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
MV, Off Airport Total, tpy	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Motor Vehicles, Parking																			
MV, Parking, lb/yr	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
MV, Parking, tpy	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Fugitive Dust																			
Runways	2.67E+01	0.00E+00	2.72E+02	0.00E+00	4.69E-01	0.00E+00													
Roadways, On-Airport	3.10E+02	0.00E+00	3.15E+03	0.00E+00	5.44E+00	0.00E+00													
Roadways, Off-Airport	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
Fugitive Dust, Total lbs/yr	3.37E+02	0.00E+00	3.42E+03	0.00E+00	5.91E+00	0.00E+00													
Fugitive Dust, Total tpy	1.68E-01	0.00E+00	1.71E+00	0.00E+00	2.95E-03	0.00E+00													
Total Operating, lbs/yr	3.37E+02	1.96E+02	5.58E+03	0.00E+00	5.91E+00	0.00E+00	1.34E+01	5.96E+01	1.96E+00	1.39E+03	1.55E+02	2.88E+02	3.91E+01	1.17E+04	3.14E+02	9.51E+02	1.81E+02	4.16E+02	0.00E+00
Total Operating, tpy	1.68E-01	9.81E-02	2.79E+00	0.00E+00	2.95E-03	0.00E+00	6.70E-03	2.98E-02	9.81E-04	6.94E-01	7.77E-02	1.44E-01	1.95E-02	5.83E+00	1.57E-01	4.76E-01	9.07E-02	2.08E-01	0.00E+00
Total Operating, kg/yr	1.53E+02	8.91E+01	2.53E+03	0.00E+00	2.68E+00	0.00E+00	6.08E+00	2.71E+01	8.91E-01	6.30E+02	7.06E+01	1.31E+02	1.77E+01	5.29E+03	1.42E+02	4.32E+02	8.23E+01	1.89E+02	0.00E+00
Acquisition Areas																			
Alternative 1, lbs/yr	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Alternative 2, lbs/yr	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Alternative 3, lbs/yr	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chrome Plating																			
LCD																			
Motor Vehicle	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00													
Running Exhaust																			
Start Exhaust																			
Evaporation																			
Northside - NG Use	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Continental City - NG Use	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
LCD, Total lbs/yr	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
LCD, Total tpy	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Emission Inventory 2015 Future Baseline	CO	THC	VOC	NOx	SO_2	PM_{10}	Acetaldehyde	Acrolein	Benzene	1,3-Butadiene	Formaldehyde	Hexane	Propylene	Propylene oxide	Styrene	Toluene	Xylene, m- or p-	Xylene, o-	Xylene (total)	Acenaphthene	Acenaphthylene	Anthracene	Benzo(a)anthracene	Benzo(a)pyrene	Benzo(b)fluoranthene	Benzo(g,h,i)perylene	Benzo(k)fluoranthene	Chrysene	Dibenz(a,h)anthracene
Future Baseline w/o LCD:																													
for comp. to Alt 1, lbs/yr	19,716,802	3,709,374	3,734,435	11,536,299	444,710	1,183,743	106,762	51,320	72,426	44,240	343,256	217	125,049	40	10,391	60,954	6,291	4,122	36,458	4	23	3	3	1	1	1	1	2	0
for comp. to Alt 1, tpy	9,858	1,855	1,867	5,768	222	592	53	26	36	22	172	0	63	0	5	30	3	2	18	0	0	0	0	0	0	0	0	0	0
for comp. to Alt 2, lbs/yr	19,803,490	3,732,219	3,757,280	12,048,867	444,710	1,184,605	106,782	51,339	72,436	44,240	343,277	217	127,057	40	10,391	61,094	6,291	4,122	36,539	4	23	3	3	1	1	1	1	2	0
for comp. to Alt 2, tpy	9,902	1,866	1,879	6,024	222	592	53	26	36	22	172	0	64	0	5	31	3	2	18	0	0	0	0	0	0	0	0	0	0
for comp. to Alt 3, lbs/yr	19,749,104	3,717,934	3,742,995	11,718,761	444,710	1,184,065	106,770	51,327	72,430	44,240	343,264	217	125,796	40	10,391	61,006	6,291	4,122	36,488	4	23	3	3	1	1	1	1	2	0
for comp. to Alt 3, tpy	9,875	1,859	1,871	5,859	222	592	53	26	36	22	172	0	63	0	5	31	3	2	18	0	0	0	0	0	0	0	0	0	0
Future Baseline w/LCD:																													
for comp. to Alt 1, lbs/yr	20,939,931	4,037,204	4,031,412	11,849,834	451,207	1,192,815	107,024	51,354	76,479	44,501	343,831	217	126,293	40	10,585	68,028	6,291	4,122	39,892	4	23	3	3	1	1	1	1	2	0
for comp. to Alt 1, tpy	10,470	2,019	2,016	5,925	226	596	54	26	38	22	172	0	63	0	5	34	3	2	20	0	0	0	0	0	0	0	0	0	0
for comp. to Alt 2, lbs/yr	21,026,619	4,060,049	4,054,257	12,362,402	451,207	1,193,677	107,044	51,373	76,489	44,501	343,852	217	128,302	40	10,585	68,168	6,291	4,122	39,973	4	23	3	3	1	1	2	1	2	0
for comp. to Alt 2, tpy	10,513	2,030	2,027	6,181	226	597	54	26	38	22	172	0	64	0	5	34	3	2	20	0	0	0	0	0	0	0	0	0	0
for comp. to Alt 3, lbs/yr	20,972,233	4,045,764	4,039,972	12,032,296	451,207	1,193,137	107,031	51,361	76,483	44,501	343,839	217	127,041	40	10,585	68,080	6,291	4,122	39,922	4	23	3	3	1	1	2	1	2	0
for comp. to Alt 3, tpy	10,486	2,023	2,020	6,016	226	597	54	26	38	22	172	0	64	0	5	34	3	2	20	0	0	0	0	0	0	0	0	0	0

Emission Inventory 2015 Future Baseline	Fluoranthene	Fluorene	Indeno(1,2,3-cd)pyrene	Naphthalene	Phenanthrene	Pyrene	2,3,7,8 TCDD Equivalents	Dioxin (tetrachloro total)	Dioxin (pentachloro total)	Dioxin (hexachloro total)	Dioxin (heptachloro total)	Dioxin (octachloro)	Furan (tetrachloro total)	Furan (pentachloro total)	Furan (hexachloro total)	Furan (heptachloro total)	Furan (octachloro)	Antimony	Arsenic	Beryllium	Cadmium	Chromium Hexavalent	Chromium (Total)	Cobalt	Copper	Lead	Manganese	Mercury	Nickel
Future Baseline w/o LCD:																													
for comp. to Alt 1, lbs/yr	6	6	0	1,749	32	6	0	0	0	0	0	0	0	0	0	0	0	0	2,085	0	224	0	2,270	0	225	11,048	614	1	329
for comp. to Alt 1, tpy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	6	0	0	0
for comp. to Alt 2, lbs/yr	6	6	0	1,750	32	6	0	0	0	0	0	0	0	0	0	0	0	0	2,085	0	224	0	2,270	0	225	11,048	614	1	329
for comp. to Alt 2, tpy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	6	0	0	0
for comp. to Alt 3, lbs/yr	6	6	0	1,750	32	6	0	0	0	0	0	0	0	0	0	0	0	0	2,085	0	224	0	2,270	0	225	11,048	614	1	329
for comp. to Alt 3, tpy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	6	0	0	0
Future Baseline w/LCD:																													
for comp. to Alt 1, lbs/yr	6	6	0	1,854	32	6	0	0	0	0	0	0	0	0	0	0	0	0	2,085	0	224	0	2,270	0	226	11,048	615	1	329
for comp. to Alt 1, tpy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	6	0	0	0
for comp. to Alt 2, lbs/yr	6	6	0	1,855	32	6	0	0	0	0	0	0	0	0	0	0	0	0	2,085	0	224	0	2,270	0	226	11,048	615	1	329
for comp. to Alt 2, tpy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	6	0	0	0
for comp. to Alt 3, lbs/yr	6	6	0	1,854	32	6	0	0	0	0	0	0	0	0	0	0	0	0	2,085	0	224	0	2,270	0	226	11,048	615	1	329
for comp. to Alt 3, tpy	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	6	0	0	0

Emission Inventory 2015 Future Baseline	Phosphorus	Selenium	Zinc	Ammonia	Bromine	Hydrogen chloride	Benzaldehyde	N-Butanol	Secondary Butanol	Ethyl Alcohol	Ethylbenzene	Ethylene Glycol Ethers	Hydrogen fluoride	Methanol	Methyl ethyl ketone	Methylene Chloride	Perchloroethylene	Trichloroethane, 1,1,1	Trichloroethylene
Future Baseline w/o LCD:																			
for comp. to Alt 1, lbs/yr	337	196	5,578	0	6	0	13	60	2	1,388	155	288	39	11,652	314	951	181	416	0
for comp. to Alt 1, tpy	0	0	3	0	0	0	0	0	0	1	0	0	0	6	0	0	0	0	0
for comp. to Alt 2, lbs/yr	337	196	5,578	0	6	0	13	60	2	1,388	155	288	39	11,652	314	951	181	416	0
for comp. to Alt 2, tpy	0	0	3	0	0	0	0	0	0	1	0	0	0	6	0	0	0	0	0
for comp. to Alt 3, lbs/yr	337	196	5,578	0	6	0	13	60	2	1,388	155	288	39	11,652	314	951	181	416	0
for comp. to Alt 3, tpy	0	0	3	0	0	0	0	0	0	1	0	0	0	6	0	0	0	0	0
Future Baseline w/LCD:																			
for comp. to Alt 1, lbs/yr	337	196	5,578	0	6	0	13	60	2	1,388	155	288	39	11,652	314	951	181	416	0
for comp. to Alt 1, tpy	0	0	3	0	0	0	0	0	0	1	0	0	0	6	0	0	0	0	0
for comp. to Alt 2, lbs/yr	337	196	5,578	0	6	0	13	60	2	1,388	155	288	39	11,652	314	951	181	416	0
for comp. to Alt 2, tpy	0	0	3	0	0	0	0	0	0	1	0	0	0	6	0	0	0	0	0
for comp. to Alt 3, lbs/yr	337	196	5,578	0	6	0	13	60	2	1,388	155	288	39	11,652	314	951	181	416	0
for comp. to Alt 3, tpy	0	0	3	0	0	0	0	0	0	1	0	0	0	6	0	0	0	0	0

Attachment B4
Example Model Output

1 ISCST3 - (DATED 96113)

IBM-PC VERSION (3.04) ISCST3R
(C) COPYRIGHT 1992-1996, TRINITY CONSULTANTS, INC.

Run Began on 2/17/1998 at 14:09:23

** BREEZE AIR SUITE (ISCST3) - I:\8359-111\AIRQUAL\ISCST3\1996X2B.DAT
** Trinity Consultants Incorporated, Dallas, TX

```
CO STARTING
CO TITLEONE  LAX Master Plan - Test Run 1
CO TITLETWO  Camp Dresser & McKee Inc., Confidential
CO MODELOPT  DFAULT  CONC  URBAN
CO AVERTIME  1  ANNUAL
CO POLLUTID  OTHER
CO TERRHGTS  FLAT
CO RUNORNOT  RUN
CO SAVEFILE  I:\8359-111\AIRQUAL\ISCST3\1996X2B.SV1  5  I:\8359-111\AIRQUAL\ISCST3\1996X2B.SV2
CO ERRORFIL  I:\8359-111\AIRQUAL\ISCST3\1996X2B.ERR
CO FINISHED

SO STARTING
SO ELEVUNIT  METERS
SO LOCATION  25RCTF00  VOLUME  1999.0  -500.1  0
SO LOCATION  25RCTF01  VOLUME  1992.0  -500.2  0
SO LOCATION  25RCTF02  VOLUME  1972.1  -503.5  0
SO LOCATION  25RCTF03  VOLUME  1939.1  -507.0  0
SO LOCATION  25RCTF04  VOLUME  1893.2  -512.6  0
SO LOCATION  25RCTF05  VOLUME  1834.3  -520.4  0
SO LOCATION  25RCTF06  VOLUME  1761.4  -528.5  0
SO LOCATION  25RCTF07  VOLUME  1676.5  -539.6  0
SO LOCATION  25RCTF08  VOLUME  1578.7  -551.0  0
SO LOCATION  25RCTF09  VOLUME  1466.9  -564.6  0
SO LOCATION  25RCTF10  VOLUME  1343.1  -580.3  0
SO LOCATION  25RCTF11  VOLUME  1205.3  -597.2  0
SO LOCATION  25RCTF12  VOLUME  1055.5  -615.3  0
SO LOCATION  25RCTF13  VOLUME  891.8  -635.6  0
SO LOCATION  25RCTF14  VOLUME  715.1  -657.1  0
SO LOCATION  25LCTF00  VOLUME  1998.3  -745.2  0
SO LOCATION  25LCTF01  VOLUME  1991.4  -746.3  0
SO LOCATION  25LCTF02  VOLUME  1972.4  -748.5  0
SO LOCATION  25LCTF03  VOLUME  1939.4  -753.0  0
SO LOCATION  25LCTF04  VOLUME  1892.5  -758.6  0
SO LOCATION  25LCTF05  VOLUME  1833.6  -765.5  0
SO LOCATION  25LCTF06  VOLUME  1761.7  -774.5  0
SO LOCATION  25LCTF07  VOLUME  1676.9  -784.7  0
SO LOCATION  25LCTF08  VOLUME  1578.0  -797.0  0
SO LOCATION  25LCTF09  VOLUME  1467.2  -810.6  0
SO LOCATION  25LCTF10  VOLUME  1342.4  -826.3  0
SO LOCATION  25LCTF11  VOLUME  1205.6  -843.2  0
SO LOCATION  25LCTF12  VOLUME  1054.9  -861.3  0
SO LOCATION  25LCTF13  VOLUME  891.1  -881.6  0
SO LOCATION  25LCTF14  VOLUME  715.4  -903.1  0
SO LOCATION  24RCTF00  VOLUME  0.3  903.1  0
SO LOCATION  24RCTF01  VOLUME  -6.7  902.0  0
SO LOCATION  24RCTF02  VOLUME  -26.7  899.7  0
SO LOCATION  24RCTF03  VOLUME  -59.6  895.3  0
SO LOCATION  24RCTF04  VOLUME  -105.6  889.6  0
SO LOCATION  24RCTF05  VOLUME  -164.5  882.8  0
SO LOCATION  24RCTF06  VOLUME  -236.4  873.8  0
SO LOCATION  24RCTF07  VOLUME  -322.2  862.6  0
SO LOCATION  24RCTF08  VOLUME  -420.1  851.2  0
SO LOCATION  24RCTF09  VOLUME  -531.9  837.7  0
SO LOCATION  24RCTF10  VOLUME  -655.7  821.9  0
SO LOCATION  24RCTF11  VOLUME  -793.5  805.0  0
```

```
SO LOCATION  24RCTF12  VOLUME  -943.2  786.9  0
SO LOCATION  24RCTF13  VOLUME  -1107.0  766.6  0
SO LOCATION  24RCTF14  VOLUME  -1283.7  745.2  0
SO LOCATION  24LCTF00  VOLUME  8.0  685.2  0
SO LOCATION  24LCTF01  VOLUME  1.0  684.1  0
SO LOCATION  24LCTF02  VOLUME  -18.9  681.8  0
SO LOCATION  24LCTF03  VOLUME  -51.9  677.3  0
SO LOCATION  24LCTF04  VOLUME  -97.8  671.7  0
SO LOCATION  24LCTF05  VOLUME  -156.7  664.9  0
SO LOCATION  24LCTF06  VOLUME  -229.6  655.9  0
SO LOCATION  24LCTF07  VOLUME  -314.5  645.7  0
SO LOCATION  24LCTF08  VOLUME  -412.3  633.3  0
SO LOCATION  24LCTF09  VOLUME  -524.1  619.7  0
SO LOCATION  24LCTF10  VOLUME  -647.9  604.0  0
SO LOCATION  24LCTF11  VOLUME  -785.7  587.1  0
SO LOCATION  24LCTF12  VOLUME  -935.5  569.0  0
SO LOCATION  24LCTF13  VOLUME  -1099.2  548.7  0
SO LOCATION  24LCTF14  VOLUME  -1275.9  527.2  0
SO LOCATION  TESTCELL  VOLUME  780.2  -1299.2  0
SO LOCATION  TAXIRW24  AREA  -3085.0  130.0  0
** SRCDESCR  Runway 24 Taxi Lane
SO LOCATION  TAXIRN25  AREA  -1000.0  -745.0  0
** SRCDESCR  Runway 25 Taxi Lane
SO LOCATION  LAXTHEME  POINT  0.0  0.0  0
SO LOCATION  TERMNL01  AREA  43.6  195.0  0
SO LOCATION  TERMNL02  AREA  -238.6  164.1  0
SO LOCATION  TERMNL03  AREA  -555.0  155.0  0
SO LOCATION  TERMNL04  AREA  -460.0  -555.0  0
SO LOCATION  TERMNL05  AREA  -235.0  -520.0  0
SO LOCATION  TERMNL06  AREA  -20.0  -495.0  0
SO LOCATION  TERMNL07  AREA  200.0  -450.0  0
SO LOCATION  TERMNL08  AREA  420.0  -410.0  0
SO LOCATION  TBITSO__  AREA  -620.0  -555.0  0
SO LOCATION  TBITNO__  AREA  -686.8  24.2  0
SO LOCATION  REMOTE_E  AREA  -2587.6  -193.4  0
SO LOCATION  REMOTE_M  AREA  -2853.0  -215.0  0
SO LOCATION  REMOTE_W  AREA  -3075.0  -15.0  0
SO SRCPARAM  25RCTF00  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF01  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF02  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF03  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF04  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF05  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF06  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF07  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF08  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF09  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF10  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF11  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF12  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF13  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25RCTF14  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF00  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF01  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF02  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF03  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF04  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF05  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF06  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF07  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF08  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF09  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF10  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF11  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF12  1.000000E+00  1  15.21  6.084
SO SRCPARAM  25LCTF13  1.000000E+00  1  15.21  6.084
```

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SO SRCPARAM  25LCTF14  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF00  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF01  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF02  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF03  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF04  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF05  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF06  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF07  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF08  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF09  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF10  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF11  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF12  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF13  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24RCTF14  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF00  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF01  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF02  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF03  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF04  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF05  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF06  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF07  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF08  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF09  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF10  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF11  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF12  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF13  1.000000E+00  1  15.21  6.084
SO SRCPARAM  24LCTF14  1.000000E+00  1  15.21  6.084
SO SRCPARAM  TESTCELL  1.000000E+00  1  30.42  30.42
SO SRCPARAM  TAXIRW24  1.000000E+00  1  3200  30  -7  1
SO SRCPARAM  TAXIRN25  1.000000E+00  1  1500  30  -7.1  1
SO SRCPARAM  LAXTHEME  0.000000E+00  0  0  0  0
SO SRCPARAM  TERMNL01  1.000000E+00  1  180  220  -7.2  1
SO SRCPARAM  TERMNL02  1.000000E+00  1  190  220  -7.2  1
SO SRCPARAM  TERMNL03  1.000000E+00  1  240  190  -7.2  1
SO SRCPARAM  TERMNL04  1.000000E+00  1  160  300  -7.2  1
SO SRCPARAM  TERMNL05  1.000000E+00  1  160  300  -7.2  1
SO SRCPARAM  TERMNL06  1.000000E+00  1  160  300  -7.2  1
SO SRCPARAM  TERMNL07  1.000000E+00  1  160  300  -7.2  1
SO SRCPARAM  TERMNL08  1.000000E+00  1  65  280  -7.2  1
SO SRCPARAM  TBITSO__  1.000000E+00  1  60  350  -7.2  1
SO SRCPARAM  TBITNO__  1.000000E+00  1  70  300  -7.2  1
SO SRCPARAM  REMOTE_E  1.000000E+00  1  160  300  -7.2  1
SO SRCPARAM  REMOTE_M  1.000000E+00  1  120  300  -7.2  1
SO SRCPARAM  REMOTE_W  1.000000E+00  1  110  90  -7.2  1
SO SRCGROUP  24LTKOFC 24LCTF00 24LCTF01 24LCTF02 24LCTF03 24LCTF04 24LCTF05
SO SRCGROUP  24LTKOFC 24LCTF06 24LCTF07 24LCTF08 24LCTF09 24LCTF10 24LCTF11
SO SRCGROUP  24LTKOFC 24LCTF12 24LCTF13 24LCTF14
SO SRCGROUP  24RTKOFC 24RCTF00 24RCTF01 24RCTF02 24RCTF03 24RCTF04 24RCTF05
SO SRCGROUP  24RTKOFC 24RCTF06 24RCTF07 24RCTF08 24RCTF09 24RCTF10 24RCTF11
SO SRCGROUP  24RTKOFC 24RCTF12 24RCTF13 24RCTF14
SO SRCGROUP  25LTKOFC 25LCTF00 25LCTF01 25LCTF02 25LCTF03 25LCTF04 25LCTF05
SO SRCGROUP  25LTKOFC 25LCTF06 25LCTF07 25LCTF08 25LCTF09 25LCTF10 25LCTF11
SO SRCGROUP  25LTKOFC 25LCTF12 25LCTF13 25LCTF14
SO SRCGROUP  25RTKOFC 25RCTF00 25RCTF01 25RCTF02 25RCTF03 25RCTF04 25RCTF05
SO SRCGROUP  25RTKOFC 25RCTF06 25RCTF07 25RCTF08 25RCTF09 25RCTF10 25RCTF11
SO SRCGROUP  25RTKOFC 25RCTF12 25RCTF13 25RCTF14
SO SRCGROUP  GSEGATES REMOTE_E REMOTE_M REMOTE_W TBITNO__ TBITSO__ TERMNL01
SO SRCGROUP  GSEGATES TERMNL02 TERMNL03 TERMNL04 TERMNL05 TERMNL06 TERMNL07
SO SRCGROUP  GSEGATES TERMNL08
SO SRCGROUP  TAXIIDLE TAXIRN25 TAXIRW24
SO SRCGROUP  TESTCELL TESTCELL
SO FINISHED
```

```
RE STARTING
RE GRIDCART CART1 STA
RE GRIDCART CART1 XYINC  -3000  29  200  -1600  15  200
RE GRIDCART CART1 END
RE DISCCART  -574.6  -1575.6
** RCPDESCR  Imperial School
RE DISCCART  -1137.9  -2111.4
** RCPDESCR  El Segundo High School
RE DISCCART  -1374.2  -2214.3
** RCPDESCR  El Segundo Jr. High
RE DISCCART  -150.0  -2110.0
** RCPDESCR  Center St. School
RE DISCCART  3365.0  -335.0
** RCPDESCR  Felton Ave. School
RE DISCCART  2400.0  450.0
** RCPDESCR  98th Street School
RE DISCCART  -150.0  1415.0
** RCPDESCR  Emerson Manor School
RE DISCCART  -155.0  1635.0
** RCPDESCR  Visitation Catholic School
RE DISCCART  -220.0  2135.0
** RCPDESCR  Kentwood School
RE DISCCART  -135.0  2700.0
** RCPDESCR  Orville Wright Jr. High
RE DISCCART  1230.0  2595.0
** RCPDESCR  Westport Heights School
RE DISCCART  -1620.0  1535.0
** RCPDESCR  Loyola Village School
RE DISCCART  -2400.0  1715.0
** RCPDESCR  Westchester High School
RE DISCCART  -2835.0  1440.0
** RCPDESCR  Paseo del Rey School
RE DISCCART  -2765.0  1100.0
** RCPDESCR  St. Bernard High School
RE DISCCART  -515.0  -2930.0
** RCPDESCR  St. Anthony's School
RE DISCCART  7130.0  960.0
** RCPDESCR  Warren Lane School
RE DISCCART  -3360.0  120.0
** RCPDESCR  West Depostion Monitor Stn
RE DISCCART  2425.0  -420.0
** RCPDESCR  Project Amb. AQ Monitor Stn
** BOUNDARY
RE DISCCART  -2990.0  -1520.0
RE DISCCART  -3624.6  -31.4
RE DISCCART  -3547.0  2.6
RE DISCCART  -3590.0  85.0
RE DISCCART  -3663.1  57.0
RE DISCCART  -4047.1  889.2
RE DISCCART  -3868.4  816.5
RE DISCCART  -3847.2  898.3
RE DISCCART  -3750.3  858.9
RE DISCCART  -3347.5  1095.0
RE DISCCART  -3226.4  1016.3
RE DISCCART  -3074.9  1016.3
RE DISCCART  -3074.9  1058.7
RE DISCCART  -2947.7  1143.5
RE DISCCART  -2867.9  1007.3
RE DISCCART  -2822.5  1028.4
RE DISCCART  -2716.5  1001.2
RE DISCCART  -2661.9  1176.8
RE DISCCART  -2713.4  1331.2
RE DISCCART  -2689.2  1388.7
RE DISCCART  -2583.2  1343.3
RE DISCCART  -2598.3  1300.9
```

RE DISCCART -2192.4 1140.5
RE DISCCART -1795.7 1125.3
RE DISCCART -1447.3 1355.4
RE DISCCART -1389.6 1276.7
RE DISCCART -1268.4 1346.3
RE DISCCART -1023.1 1064.8
RE DISCCART -965.6 1152.6
RE DISCCART -1204.9 1406.9
RE DISCCART -1029.0 1559.5
RE DISCCART -1032.0 1789.6
RE DISCCART -635.3 1789.6
RE DISCCART -635.3 1462.6
RE DISCCART -193.8 1465.4
RE DISCCART -193.1 1365.8
RE DISCCART -105.3 1365.8
RE DISCCART 31.0 1429.3
RE DISCCART 3.7 1489.9
RE DISCCART 385.3 1499.0
RE DISCCART 382.3 1172.0
RE DISCCART 430.7 1060.0
RE DISCCART 564.0 1060.0
RE DISCCART 564.2 987.4
RE DISCCART 642.9 999.5
RE DISCCART 748.9 1202.3
RE DISCCART 857.9 1202.3
RE DISCCART 1309.2 1520.2
RE DISCCART 1412.1 1377.9
RE DISCCART 1481.8 1362.7
RE DISCCART 1478.8 1287.1
RE DISCCART 1545.4 1281.0
RE DISCCART 1518.2 539.3
RE DISCCART 558.2 533.3
RE DISCCART 555.3 463.4
RE DISCCART 282.7 463.4
RE DISCCART 282.7 172.8
RE DISCCART 549.2 175.8
RE DISCCART 597.7 136.4
RE DISCCART 2199.8 145.5
RE DISCCART 2206.0 -315.2
RE DISCCART 2514.9 -318.2
RE DISCCART 2521.0 -384.8
RE DISCCART 2981.3 -390.8
RE DISCCART 2981.3 -1093.4
RE DISCCART 2763.2 -1090.4
RE DISCCART 2763.2 -993.5
RE DISCCART 2675.4 -993.5
RE DISCCART 2672.4 -1093.4
RE DISCCART 2542.2 -1096.5
RE DISCCART 2545.2 -1499.3
RE DISCCART 594.9 -1511.5
RE DISCCART 591.8 -1447.9
RE DISCCART 613.0 -1463.0
RE DISCCART 634.2 -1453.9
RE DISCCART 600.9 -1345.0
RE DISCCART 600.9 -1099.7
RE DISCCART 561.5 -1096.7
RE DISCCART 549.4 -1393.4
RE DISCCART 440.2 -1454.2
RE DISCCART 125.2 -1457.2
RE DISCCART -398.7 -1454.2
RE DISCCART -998.8 -1448.1
RE DISCCART -2201.3 -1460.3
RE DISCCART -2534.5 -1514.7
RE FINISHED

ME STARTING

```
ME INPUTFIL  I:\8359-111\AIRQUAL\ISCST3\LENNOX.ASC
ME ANEMHGHT  10.0  METERS
ME SURFDATA  52118  1981
ME UAIRDATA  91919  1981
ME STARTEND  81  01  01  1  81  12  31  24
ME FINISHED

OU STARTING
OU PLOTFILE  ANNUAL  24LTKOFC  I:\8359-111\AIRQUAL\ISCST3\24LTKOFC.PLT
OU PLOTFILE  ANNUAL  24RTKOFC  I:\8359-111\AIRQUAL\ISCST3\24RTKOFC.PLT
OU PLOTFILE  ANNUAL  25LTKOFC  I:\8359-111\AIRQUAL\ISCST3\25LTKOFC.PLT
OU PLOTFILE  ANNUAL  25RTKOFC  I:\8359-111\AIRQUAL\ISCST3\25RTKOFC.PLT
OU PLOTFILE  ANNUAL  GSEGATES  I:\8359-111\AIRQUAL\ISCST3\GSEGATES.PLT
OU PLOTFILE  ANNUAL  TAXIIDLE  I:\8359-111\AIRQUAL\ISCST3\TAXIIDLE.PLT
OU PLOTFILE  ANNUAL  TESTCELL  I:\8359-111\AIRQUAL\ISCST3\TESTCELL.PLT
OU FINISHED


 *** Message Summary For ISC3 Model Setup ***

 --------- Summary of Total Messages --------

A Total of         0 Fatal Error Message(s)
A Total of         5 Warning Message(s)
A Total of         0 Informational Message(s)


   ******** FATAL ERROR MESSAGES ********
            ***  NONE  ***


   ********   WARNING MESSAGES   ********
SO W320   158 APARM :Source Parameter May Be Out-of-Range for Parameter   XINIT
SO W391   158 APARM :Aspect ratio (L/W) of area source greater than 10  TAXIRW24
SO W391   159 APARM :Aspect ratio (L/W) of area source greater than 10  TAXIRN25
SO W320   160 PPARM :Source Parameter May Be Out-of-Range for Parameter      QS
OU W540   339 OUTQA :No RECTABLE/MAXTABLE/DAYTABLE for Average Period   01-HR

**********************************
*** SETUP Finishes Successfully ***
**********************************
```

**MODELOPTs: CONC URBAN FLAT DFAULT

*** MODEL SETUP OPTIONS SUMMARY ***

- -

**Intermediate Terrain Processing is Selected

**Model Is Setup For Calculation of Average CONCentration Values.

-- SCAVENGING/DEPOSITION LOGIC --
**Model Uses NO DRY DEPLETION. DDPLETE = F
**Model Uses NO WET DEPLETION. WDPLETE = F
**NO WET SCAVENGING Data Provided.
**Model Does NOT Use GRIDDED TERRAIN Data for Depletion Calculations

**Model Uses URBAN Dispersion.

**Model Uses Regulatory DEFAULT Options:
1. Final Plume Rise.
2. Stack-tip Downwash.
3. Buoyancy-induced Dispersion.
4. Use Calms Processing Routine.
5. Not Use Missing Data Processing Routine.
6. Default Wind Profile Exponents.
7. Default Vertical Potential Temperature Gradients.
8. "Upper Bound" Values for Supersquat Buildings.
9. No Exponential Decay for URBAN/Non-SO2

**Model Assumes Receptors on FLAT Terrain.

**Model Assumes No FLAGPOLE Receptor Heights.

**Model Calculates 1 Short Term Average(s) of: 1-HR
and Calculates ANNUAL Averages

**This Run Includes: 77 Source(s); 7 Source Group(s); and 539 Receptor(s)

**The Model Assumes A Pollutant Type of: OTHER

**Model Set To Continue RUNning After the Setup Testing.

**Output Options Selected:
Model Outputs Tables of ANNUAL Averages by Receptor
Model Outputs External File(s) of High Values for Plotting (PLOTFILE Keyword)

**NOTE: The Following Flags May Appear Following CONC Values: c for Calm Hours
m for Missing Hours
b for Both Calm and Missing Hours

**Misc. Inputs: Anem. Hgt. (m) = 10.00 ; Decay Coef. = .0000 ; Rot. Angle = .0
Emission Units = GRAMS/SEC ; Emission Rate Unit Factor = .10000E+07
Output Units = MICROGRAMS/M**3

**Input Runstream File: I:\8359-111\AIRQUAL\ISCST3\1996X2B.DAT ; **Output Print File: I:\8359-111\AIRQUAL\ISCST3\1996X2B.LST
**File for Saving Result Arrays: I:\8359-111\AIRQUAL\ISCST3\1996X2B.SV1
**Detailed Error/Message File: I:\8359-111\AIRQUAL\ISCST3\1996X2B.ERR

**MODELOPTs: CONC URBAN FLAT DFAULT

*** POINT SOURCE DATA ***

SOURCE ID	NUMBER PART. CATS.	EMISSION RATE (GRAMS/SEC)	X (METERS)	Y (METERS)	BASE ELEV. (METERS)	STACK HEIGHT (METERS)	STACK TEMP. (DEG.K)	STACK EXIT VEL. (M/SEC)	STACK DIAMETER (METERS)	BUILDING EXISTS	EMISSION RATE SCALAR VARY BY
LAXTHEME	0	.00000E+00	.0	.0	.0	.00	.00	.00	.00	NO	

**MODELOPTs: CONC URBAN FLAT DFAULT

*** VOLUME SOURCE DATA ***

SOURCE ID	NUMBER PART. CATS.	EMISSION RATE (GRAMS/SEC)	X (METERS)	Y (METERS)	BASE ELEV. (METERS)	RELEASE HEIGHT (METERS)	INIT. SY (METERS)	INIT. SZ (METERS)	EMISSION RATE SCALAR VARY BY
25RCTF00	0	.10000E+01	1999.0	-500.1	.0	1.00	15.21	6.08	
25RCTF01	0	.10000E+01	1992.0	-500.2	.0	1.00	15.21	6.08	
25RCTF02	0	.10000E+01	1972.1	-503.5	.0	1.00	15.21	6.08	
25RCTF03	0	.10000E+01	1939.1	-507.0	.0	1.00	15.21	6.08	
25RCTF04	0	.10000E+01	1893.2	-512.6	.0	1.00	15.21	6.08	
25RCTF05	0	.10000E+01	1834.3	-520.4	.0	1.00	15.21	6.08	
25RCTF06	0	.10000E+01	1761.4	-528.5	.0	1.00	15.21	6.08	
25RCTF07	0	.10000E+01	1676.5	-539.6	.0	1.00	15.21	6.08	
25RCTF08	0	.10000E+01	1578.7	-551.0	.0	1.00	15.21	6.08	
25RCTF09	0	.10000E+01	1466.9	-564.6	.0	1.00	15.21	6.08	
25RCTF10	0	.10000E+01	1343.1	-580.3	.0	1.00	15.21	6.08	
25RCTF11	0	.10000E+01	1205.3	-597.2	.0	1.00	15.21	6.08	
25RCTF12	0	.10000E+01	1055.5	-615.3	.0	1.00	15.21	6.08	
25RCTF13	0	.10000E+01	891.8	-635.6	.0	1.00	15.21	6.08	
25RCTF14	0	.10000E+01	715.1	-657.1	.0	1.00	15.21	6.08	
25LCTF00	0	.10000E+01	1998.3	-745.2	.0	1.00	15.21	6.08	
25LCTF01	0	.10000E+01	1991.4	-746.3	.0	1.00	15.21	6.08	
25LCTF02	0	.10000E+01	1972.4	-748.5	.0	1.00	15.21	6.08	
25LCTF03	0	.10000E+01	1939.4	-753.0	.0	1.00	15.21	6.08	
25LCTF04	0	.10000E+01	1892.5	-758.6	.0	1.00	15.21	6.08	
25LCTF05	0	.10000E+01	1833.6	-765.5	.0	1.00	15.21	6.08	
25LCTF06	0	.10000E+01	1761.7	-774.5	.0	1.00	15.21	6.08	
25LCTF07	0	.10000E+01	1676.9	-784.7	.0	1.00	15.21	6.08	
25LCTF08	0	.10000E+01	1578.0	-797.0	.0	1.00	15.21	6.08	
25LCTF09	0	.10000E+01	1467.2	-810.6	.0	1.00	15.21	6.08	
25LCTF10	0	.10000E+01	1342.4	-826.3	.0	1.00	15.21	6.08	
25LCTF11	0	.10000E+01	1205.6	-843.2	.0	1.00	15.21	6.08	
25LCTF12	0	.10000E+01	1054.9	-861.3	.0	1.00	15.21	6.08	
25LCTF13	0	.10000E+01	891.1	-881.6	.0	1.00	15.21	6.08	
25LCTF14	0	.10000E+01	715.4	-903.1	.0	1.00	15.21	6.08	
24RCTF00	0	.10000E+01	.3	903.1	.0	1.00	15.21	6.08	
24RCTF01	0	.10000E+01	-6.7	902.0	.0	1.00	15.21	6.08	
24RCTF02	0	.10000E+01	-26.7	899.7	.0	1.00	15.21	6.08	
24RCTF03	0	.10000E+01	-59.6	895.3	.0	1.00	15.21	6.08	
24RCTF04	0	.10000E+01	-105.6	889.6	.0	1.00	15.21	6.08	
24RCTF05	0	.10000E+01	-164.5	882.8	.0	1.00	15.21	6.08	
24RCTF06	0	.10000E+01	-236.4	873.8	.0	1.00	15.21	6.08	
24RCTF07	0	.10000E+01	-322.2	862.6	.0	1.00	15.21	6.08	
24RCTF08	0	.10000E+01	-420.1	851.2	.0	1.00	15.21	6.08	
24RCTF09	0	.10000E+01	-531.9	837.7	.0	1.00	15.21	6.08	

**MODELOPTs: CONC URBAN FLAT DFAULT

*** VOLUME SOURCE DATA ***

SOURCE ID	NUMBER PART. CATS.	EMISSION RATE (GRAMS/SEC)	X (METERS)	Y (METERS)	BASE ELEV. (METERS)	RELEASE HEIGHT (METERS)	INIT. SY (METERS)	INIT. SZ (METERS)	EMISSION RATE SCALAR VARY BY
24RCTF10	0	.10000E+01	-655.7	821.9	.0	1.00	15.21	6.08	
24RCTF11	0	.10000E+01	-793.5	805.0	.0	1.00	15.21	6.08	
24RCTF12	0	.10000E+01	-943.2	786.9	.0	1.00	15.21	6.08	
24RCTF13	0	.10000E+01	-1107.0	766.6	.0	1.00	15.21	6.08	
24RCTF14	0	.10000E+01	-1283.7	745.2	.0	1.00	15.21	6.08	
24LCTF00	0	.10000E+01	8.0	685.2	.0	1.00	15.21	6.08	
24LCTF01	0	.10000E+01	1.0	684.1	.0	1.00	15.21	6.08	
24LCTF02	0	.10000E+01	-18.9	681.8	.0	1.00	15.21	6.08	
24LCTF03	0	.10000E+01	-51.9	677.3	.0	1.00	15.21	6.08	
24LCTF04	0	.10000E+01	-97.8	671.7	.0	1.00	15.21	6.08	
24LCTF05	0	.10000E+01	-156.7	664.9	.0	1.00	15.21	6.08	
24LCTF06	0	.10000E+01	-229.6	655.9	.0	1.00	15.21	6.08	
24LCTF07	0	.10000E+01	-314.5	645.7	.0	1.00	15.21	6.08	
24LCTF08	0	.10000E+01	-412.3	633.3	.0	1.00	15.21	6.08	
24LCTF09	0	.10000E+01	-524.1	619.7	.0	1.00	15.21	6.08	
24LCTF10	0	.10000E+01	-647.9	604.0	.0	1.00	15.21	6.08	
24LCTF11	0	.10000E+01	-785.7	587.1	.0	1.00	15.21	6.08	
24LCTF12	0	.10000E+01	-935.5	569.0	.0	1.00	15.21	6.08	
24LCTF13	0	.10000E+01	-1099.2	548.7	.0	1.00	15.21	6.08	
24LCTF14	0	.10000E+01	-1275.9	527.2	.0	1.00	15.21	6.08	
TESTCELL	0	.10000E+01	780.2	-1299.2	.0	1.00	30.42	30.42	

**MODELOPTs: CONC URBAN FLAT DFAULT

*** AREA SOURCE DATA ***

SOURCE ID	NUMBER PART. CATS.	EMISSION RATE (GRAMS/SEC /METER**2)	COORD (SW CORNER) X (METERS)	Y (METERS)	BASE ELEV. (METERS)	RELEASE HEIGHT (METERS)	X-DIM OF AREA (METERS)	Y-DIM OF AREA (METERS)	ORIENT. OF AREA (DEG.)	INIT. SZ (METERS)	EMISSION RATE SCALAR VARY BY
TAXIRW24	0	.10000E+01	-3085.0	130.0	.0	1.00	3200.00	30.00	-7.00	1.00	
TAXIRN25	0	.10000E+01	-1000.0	-745.0	.0	1.00	1500.00	30.00	-7.10	1.00	
TERMNL01	0	.10000E+01	43.6	195.0	.0	1.00	180.00	220.00	-7.20	1.00	
TERMNL02	0	.10000E+01	-238.6	164.1	.0	1.00	190.00	220.00	-7.20	1.00	
TERMNL03	0	.10000E+01	-555.0	155.0	.0	1.00	240.00	190.00	-7.20	1.00	
TERMNL04	0	.10000E+01	-460.0	-555.0	.0	1.00	160.00	300.00	-7.20	1.00	
TERMNL05	0	.10000E+01	-235.0	-520.0	.0	1.00	160.00	300.00	-7.20	1.00	
TERMNL06	0	.10000E+01	-20.0	-495.0	.0	1.00	160.00	300.00	-7.20	1.00	
TERMNL07	0	.10000E+01	200.0	-450.0	.0	1.00	160.00	300.00	-7.20	1.00	
TERMNL08	0	.10000E+01	420.0	-410.0	.0	1.00	65.00	280.00	-7.20	1.00	
TBITSO__	0	.10000E+01	-620.0	-555.0	.0	1.00	60.00	350.00	-7.20	1.00	
TBITNO__	0	.10000E+01	-686.8	24.2	.0	1.00	70.00	300.00	-7.20	1.00	
REMOTE_E	0	.10000E+01	-2587.6	-193.4	.0	1.00	160.00	300.00	-7.20	1.00	
REMOTE_M	0	.10000E+01	-2853.0	-215.0	.0	1.00	120.00	300.00	-7.20	1.00	
REMOTE_W	0	.10000E+01	-3075.0	-15.0	.0	1.00	110.00	90.00	-7.20	1.00	

**MODELOPTs: CONC URBAN FLAT DFAULT

*** SOURCE IDs DEFINING SOURCE GROUPS ***

GROUP ID SOURCE IDs

24LTKOFC 24LCTF00, 24LCTF01, 24LCTF02, 24LCTF03, 24LCTF04, 24LCTF05, 24LCTF06, 24LCTF07, 24LCTF08, 24LCTF09, 24LCTF10, 24LCTF11,

24LCTF12, 24LCTF13, 24LCTF14,

24RTKOFC 24RCTF00, 24RCTF01, 24RCTF02, 24RCTF03, 24RCTF04, 24RCTF05, 24RCTF06, 24RCTF07, 24RCTF08, 24RCTF09, 24RCTF10, 24RCTF11,

24RCTF12, 24RCTF13, 24RCTF14,

25LTKOFC 25LCTF00, 25LCTF01, 25LCTF02, 25LCTF03, 25LCTF04, 25LCTF05, 25LCTF06, 25LCTF07, 25LCTF08, 25LCTF09, 25LCTF10, 25LCTF11,

25LCTF12, 25LCTF13, 25LCTF14,

25RTKOFC 25RCTF00, 25RCTF01, 25RCTF02, 25RCTF03, 25RCTF04, 25RCTF05, 25RCTF06, 25RCTF07, 25RCTF08, 25RCTF09, 25RCTF10, 25RCTF11,

25RCTF12, 25RCTF13, 25RCTF14,

GSEGATES TERMNL01, TERMNL02, TERMNL03, TERMNL04, TERMNL05, TERMNL06, TERMNL07, TERMNL08, TBITSO__, TBITNO__, REMOTE_E, REMOTE_M,

REMOTE_W,

TAXIIDLE TAXIRW24, TAXIRN25,

TESTCELL TESTCELL,

**MODELOPTs: CONC URBAN FLAT DFAULT

*** GRIDDED RECEPTOR NETWORK SUMMARY ***

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

*** X-COORDINATES OF GRID ***
(METERS)

-3000.0, -2800.0, -2600.0, -2400.0, -2200.0, -2000.0, -1800.0, -1600.0, -1400.0, -1200.0,
-1000.0, -800.0, -600.0, -400.0, -200.0, .0, 200.0, 400.0, 600.0, 800.0,
1000.0, 1200.0, 1400.0, 1600.0, 1800.0, 2000.0, 2200.0, 2400.0, 2600.0,

*** Y-COORDINATES OF GRID ***
(METERS)

-1600.0, -1400.0, -1200.0, -1000.0, -800.0, -600.0, -400.0, -200.0, .0, 200.0,
400.0, 600.0, 800.0, 1000.0, 1200.0,

**MODELOPTs: CONC URBAN FLAT DFAULT

*** DISCRETE CARTESIAN RECEPTORS ***
(X-COORD, Y-COORD, ZELEV, ZFLAG)
(METERS)

(	-574.6,	-1575.6,	.0, .0);	(	-1137.9,	-2111.4,	.0, .0);
(	-1374.2,	-2214.3,	.0, .0);	(	-150.0,	-2110.0,	.0, .0);
(	3365.0,	-335.0,	.0, .0);	(	2400.0,	450.0,	.0, .0);
(	-150.0,	1415.0,	.0, .0);	(	-155.0,	1635.0,	.0, .0);
(	-220.0,	2135.0,	.0, .0);	(	-135.0,	2700.0,	.0, .0);
(	1230.0,	2595.0,	.0, .0);	(	-1620.0,	1535.0,	.0, .0);
(	-2400.0,	1715.0,	.0, .0);	(	-2835.0,	1440.0,	.0, .0);
(	-2765.0,	1100.0,	.0, .0);	(	-515.0,	-2930.0,	.0, .0);
(	7130.0,	960.0,	.0, .0);	(	-3360.0,	120.0,	.0, .0);
(	2425.0,	-420.0,	.0, .0);	(	-2990.0,	-1520.0,	.0, .0);
(	-3624.6,	-31.4,	.0, .0);	(	-3547.0,	2.6,	.0, .0);
(	-3590.0,	85.0,	.0, .0);	(	-3663.1,	57.0,	.0, .0);
(	-4047.1,	889.2,	.0, .0);	(	-3868.4,	816.5,	.0, .0);
(	-3847.2,	898.3,	.0, .0);	(	-3750.3,	858.9,	.0, .0);
(	-3347.5,	1095.0,	.0, .0);	(	-3226.4,	1016.3,	.0, .0);
(	-3074.9,	1016.3,	.0, .0);	(	-3074.9,	1058.7,	.0, .0);
(	-2947.7,	1143.5,	.0, .0);	(	-2867.9,	1007.3,	.0, .0);
(	-2822.5,	1028.4,	.0, .0);	(	-2716.5,	1001.2,	.0, .0);
(	-2661.9,	1176.8,	.0, .0);	(	-2713.4,	1331.2,	.0, .0);
(	-2689.2,	1388.7,	.0, .0);	(	-2583.2,	1343.3,	.0, .0);
(	-2598.3,	1300.9,	.0, .0);	(	-2192.4,	1140.5,	.0, .0);
(	-1795.7,	1125.3,	.0, .0);	(	-1447.3,	1355.4,	.0, .0);
(	-1389.6,	1276.7,	.0, .0);	(	-1268.4,	1346.3,	.0, .0);
(	-1023.1,	1064.8,	.0, .0);	(	-965.6,	1152.6,	.0, .0);
(	-1204.9,	1406.9,	.0, .0);	(	-1029.0,	1559.5,	.0, .0);
(	-1032.0,	1789.6,	.0, .0);	(	-635.3,	1789.6,	.0, .0);
(	-635.3,	1462.6,	.0, .0);	(	-193.8,	1465.4,	.0, .0);
(	-193.1,	1365.8,	.0, .0);	(	-105.3,	1365.8,	.0, .0);
(	31.0,	1429.3,	.0, .0);	(	3.7,	1489.9,	.0, .0);
(	385.3,	1499.0,	.0, .0);	(	382.3,	1172.0,	.0, .0);
(	430.7,	1060.0,	.0, .0);	(	564.0,	1060.0,	.0, .0);
(	564.2,	987.4,	.0, .0);	(	642.9,	999.5,	.0, .0);
(	748.9,	1202.3,	.0, .0);	(	857.9,	1202.3,	.0, .0);
(	1309.2,	1520.2,	.0, .0);	(	1412.1,	1377.9,	.0, .0);
(	1481.8,	1362.7,	.0, .0);	(	1478.8,	1287.1,	.0, .0);
(	1545.4,	1281.0,	.0, .0);	(	1518.2,	539.3,	.0, .0);
(	558.2,	533.3,	.0, .0);	(	555.3,	463.4,	.0, .0);
(	282.7,	463.4,	.0, .0);	(	282.7,	172.8,	.0, .0);
(	549.2,	175.8,	.0, .0);	(	597.7,	136.4,	.0, .0);
(	2199.8,	145.5,	.0, .0);	(	2206.0,	-315.2,	.0, .0);
(	2514.9,	-318.2,	.0, .0);	(	2521.0,	-384.8,	.0, .0);
(	2981.3,	-390.8,	.0, .0);	(	2981.3,	-1093.4,	.0, .0);
(	2763.2,	-1090.4,	.0, .0);	(	2763.2,	-993.5,	.0, .0);
(	2675.4,	-993.5,	.0, .0);	(	2672.4,	-1093.4,	.0, .0);
(	2542.2,	-1096.5,	.0, .0);	(	2545.2,	-1499.3,	.0, .0);

**MODELOPTs: CONC URBAN FLAT DFAULT

*** DISCRETE CARTESIAN RECEPTORS ***
(X-COORD, Y-COORD, ZELEV, ZFLAG)
(METERS)

(	594.9,	-1511.5,	.0, .0);	(	591.8,	-1447.9,	.0, .0);
(	613.0,	-1463.0,	.0, .0);	(	634.2,	-1453.9,	.0, .0);
(	600.9,	-1345.0,	.0, .0);	(	600.9,	-1099.7,	.0, .0);
(	561.5,	-1096.7,	.0, .0);	(	549.4,	-1393.4,	.0, .0);
(	440.2,	-1454.2,	.0, .0);	(	125.2,	-1457.2,	.0, .0);
(	-398.7,	-1454.2,	.0, .0);	(	-998.8,	-1448.1,	.0, .0);
(	-2201.3,	-1460.3,	.0, .0);	(	-2534.5,	-1514.7,	.0, .0);

**MODELOPTs: CONC URBAN FLAT DFAULT

* SOURCE-RECEPTOR COMBINATIONS FOR WHICH CALCULATIONS MAY NOT BE PERFORMED *
LESS THAN 1.0 METER OR 3*ZLB IN DISTANCE, OR WITHIN OPEN PIT SOURCE

SOURCE ID	- - RECEPTOR LOCATION - - XR (METERS)	YR (METERS)	DISTANCE (METERS)
25RCTF11	1200.0	-600.0	-26.71
25LCTF08	1600.0	-800.0	-10.50
24RCTF11	-800.0	800.0	-24.50
24LCTF11	-800.0	600.0	-13.44
LAXTHEME	.0	.0	.00

**MODELOPTs: CONC URBAN FLAT DFAULT

*** METEOROLOGICAL DAYS SELECTED FOR PROCESSING ***
(1=YES; 0=NO)

1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
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1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1

METEOROLOGICAL DATA PROCESSED BETWEEN START DATE: 81 1 1 1
AND END DATE: 81 12 31 24

NOTE: METEOROLOGICAL DATA ACTUALLY PROCESSED WILL ALSO DEPEND ON WHAT IS INCLUDED IN THE DATA FILE.

*** UPPER BOUND OF FIRST THROUGH FIFTH WIND SPEED CATEGORIES ***
(METERS/SEC)

1.54, 3.09, 5.14, 8.23, 10.80,

*** WIND PROFILE EXPONENTS ***

STABILITY	WIND SPEED CATEGORY					
CATEGORY	1	2	3	4	5	6
A	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00
B	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00
C	.20000E+00	.20000E+00	.20000E+00	.20000E+00	.20000E+00	.20000E+00
D	.25000E+00	.25000E+00	.25000E+00	.25000E+00	.25000E+00	.25000E+00
E	.30000E+00	.30000E+00	.30000E+00	.30000E+00	.30000E+00	.30000E+00
F	.30000E+00	.30000E+00	.30000E+00	.30000E+00	.30000E+00	.30000E+00

*** VERTICAL POTENTIAL TEMPERATURE GRADIENTS ***
(DEGREES KELVIN PER METER)

STABILITY	WIND SPEED CATEGORY					
CATEGORY	1	2	3	4	5	6
A	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
B	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
C	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
D	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
E	.20000E-01	.20000E-01	.20000E-01	.20000E-01	.20000E-01	.20000E-01
F	.35000E-01	.35000E-01	.35000E-01	.35000E-01	.35000E-01	.35000E-01

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE FIRST 24 HOURS OF METEOROLOGICAL DATA ***

FILE: I:\8359-111\AIRQUAL\ISCST3\LENNOX.ASC FORMAT: (4I2,2F9.4,F6.1,I2,2F7.1,f9.4,f10.1,f8.4,i4,f7.2)
SURFACE STATION NO.: 52118 UPPER AIR STATION NO.: 91919
NAME: UNKNOWN NAME: UNKNOWN
YEAR: 1981 YEAR: 1981

YEAR	MONTH	DAY	HOUR	FLOW VECTOR	SPEED (M/S)	TEMP (K)	STAB CLASS	MIXING HEIGHT (M) RURAL	MIXING HEIGHT (M) URBAN	USTAR	M-O LENGTH (M)	Z-0 (M)	IPCODE	PRATE (mm/HR)
81	1	1	1	134.8	1.00	285.9	7	387.1	152.0	.0000	.0	.0000	0	.00
81	1	1	2	124.9	.00	286.5	7	397.2	152.0	.0000	.0	.0000	0	.00
81	1	1	3	85.0	1.00	285.9	6	407.3	152.0	.0000	.0	.0000	0	.00
81	1	1	4	143.5	1.34	285.9	5	417.4	152.0	.0000	.0	.0000	0	.00
81	1	1	5	151.5	1.00	285.4	4	427.4	427.4	.0000	.0	.0000	0	.00
81	1	1	6	319.5	1.00	283.7	5	437.5	152.0	.0000	.0	.0000	0	.00
81	1	1	7	139.5	1.00	283.7	6	447.5	152.0	.0000	.0	.0000	0	.00
81	1	1	8	314.6	1.00	284.3	5	70.1	201.5	.0000	.0	.0000	0	.00
81	1	1	9	186.5	1.00	287.0	4	144.7	254.3	.0000	.0	.0000	0	.00
81	1	1	10	256.6	1.00	291.5	3	219.4	307.0	.0000	.0	.0000	0	.00
81	1	1	11	44.1	1.34	294.8	2	294.0	359.8	.0000	.0	.0000	0	.00
81	1	1	12	80.6	3.13	290.9	3	368.7	412.5	.0000	.0	.0000	0	.00
81	1	1	13	132.2	3.13	289.8	3	443.3	465.3	.0000	.0	.0000	0	.00
81	1	1	14	124.2	3.13	290.4	3	518.0	518.0	.0000	.0	.0000	0	.00
81	1	1	15	134.8	2.68	290.4	3	518.0	518.0	.0000	.0	.0000	0	.00
81	1	1	16	143.2	2.68	289.3	4	518.0	518.0	.0000	.0	.0000	0	.00
81	1	1	17	132.6	2.68	288.1	5	518.0	511.1	.0000	.0	.0000	0	.00
81	1	1	18	120.1	2.24	287.6	6	518.0	468.5	.0000	.0	.0000	0	.00
81	1	1	19	110.5	1.34	287.0	7	518.0	425.9	.0000	.0	.0000	0	.00
81	1	1	20	145.7	1.00	287.0	7	518.0	383.4	.0000	.0	.0000	0	.00
81	1	1	21	111.1	1.00	287.6	7	518.0	340.8	.0000	.0	.0000	0	.00
81	1	1	22	2.0	1.00	287.0	7	518.0	298.2	.0000	.0	.0000	0	.00
81	1	1	23	.7	.00	286.5	7	518.0	255.6	.0000	.0	.0000	0	.00
81	1	1	24	134.7	1.00	286.5	6	518.0	213.0	.0000	.0	.0000	0	.00

*** NOTES: STABILITY CLASS 1=A, 2=B, 3=C, 4=D, 5=E AND 6=F.
FLOW VECTOR IS DIRECTION TOWARD WHICH WIND IS BLOWING.

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24LTKOFC ***
INCLUDING SOURCE(S): 24LCTF00, 24LCTF01, 24LCTF02, 24LCTF03, 24LCTF04, 24LCTF05, 24LCTF06, 24LCTF07, 24LCTF08, 24LCTF09, 24LCTF10, 24LCTF11, 24LCTF12, 24LCTF13, 24LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-3000.00	-2800.00	-2600.00	-2400.00	-2200.00	-2000.00	-1800.00	-1600.00	-1400.00
1200.00	3.36787	3.53936	3.73019	3.95988	4.26971	4.72273	5.52634	6.64520	8.50846
1000.00	4.64700	5.02674	5.45323	5.93817	6.50269	7.21673	8.25340	10.13883	13.13590
800.00	5.78259	6.50202	7.36596	8.41167	9.69269	11.29720	13.41444	16.77817	23.83297
600.00	6.32237	7.26437	8.48045	10.10302	12.36351	15.70447	21.07331	30.80817	53.29852
400.00	6.26162	7.15120	8.28207	9.76062	11.76580	14.62616	19.04996	27.14623	41.67699
200.00	5.52980	6.14976	6.89688	7.83157	9.07992	10.89184	13.46756	16.22016	19.87476
.00	4.57413	5.03875	5.64479	6.46024	7.52930	8.73147	9.89177	11.20270	12.92511
-200.00	3.98835	4.46259	5.05214	5.72173	6.37142	6.96387	7.54492	8.17448	8.88219
-400.00	3.72462	4.14289	4.56140	4.92919	5.24440	5.51034	5.74164	5.92884	6.27906
-600.00	3.47632	3.73575	3.94666	4.11070	4.21827	4.27125	4.32708	4.44274	4.80460
-800.00	3.11802	3.23609	3.31057	3.33368	3.31389	3.31740	3.39138	3.57373	3.90846
-1000.00	2.70022	2.71923	2.69889	2.65549	2.64153	2.70221	2.82082	3.00891	3.27679
-1200.00	2.26819	2.22880	2.18371	2.16879	2.21604	2.31279	2.42736	2.58732	2.81437
-1400.00	1.87257	1.83453	1.82460	1.86121	1.93951	2.02836	2.12110	2.26419	2.47134
-1600.00	1.56902	1.56518	1.59463	1.65581	1.72964	1.79660	1.87837	2.01805	2.20372

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24LTKOFC ***
INCLUDING SOURCE(S): 24LCTF00, 24LCTF01, 24LCTF02, 24LCTF03, 24LCTF04, 24LCTF05, 24LCTF06, 24LCTF07, 24LCTF08, 24LCTF09, 24LCTF10, 24LCTF11, 24LCTF12, 24LCTF13, 24LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-1200.00	-1000.00	-800.00	-600.00	-400.00	-200.00	.00	200.00	400.00
1200.00	10.82639	12.98565	15.47792	18.58358	22.42739	24.84094	25.09373	18.46488	14.89402
1000.00	17.17470	20.81190	24.84398	29.85943	36.54596	45.77917	48.33771	31.68613	22.07624
800.00	34.19995	44.79480	57.29613	69.51900	89.61023	121.44460	177.46990	74.70691	48.88100
600.00	112.72970	187.71270	159.30590	490.35720	355.28360	385.49670	353.31210	149.62850	75.93875
400.00	65.11195	72.65951	76.91681	77.18101	76.24020	74.26776	74.09171	61.36859	42.28465
200.00	24.74693	29.75377	32.44324	32.86582	33.33769	34.57721	34.68449	29.75888	27.54235
.00	14.90208	16.03178	17.38869	18.70472	19.97578	20.82661	20.92626	18.35277	17.65149
-200.00	9.65730	10.17201	11.09446	12.28372	13.33263	14.16960	14.30046	12.87342	12.16014
-400.00	6.86657	7.38699	7.94706	8.78912	9.48859	10.24758	10.40069	9.62362	9.05949
-600.00	5.31710	5.74616	6.18130	6.63694	7.13952	7.75805	7.88041	7.44383	7.02691
-800.00	4.30597	4.68323	5.01939	5.25162	5.61969	6.08376	6.18249	5.91993	5.59052
-1000.00	3.60668	3.93831	4.16769	4.32132	4.60050	4.90667	4.98430	4.82560	4.55811
-1200.00	3.10300	3.36924	3.52139	3.66026	3.88837	4.06301	4.10774	4.01047	3.79765
-1400.00	2.71383	2.91267	3.02639	3.16791	3.36060	3.45010	3.45576	3.38658	3.21811
-1600.00	2.39449	2.54112	2.64386	2.78775	2.94781	2.99190	2.96796	2.90336	2.76480

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24LTKOFC ***
INCLUDING SOURCE(S): 24LCTF00, 24LCTF01, 24LCTF02, 24LCTF03, 24LCTF04, 24LCTF05, 24LCTF06, 24LCTF07, 24LCTF08, 24LCTF09, 24LCTF10, 24LCTF11, 24LCTF12, 24LCTF13, 24LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	600.00	800.00	1000.00	1200.00	1400.00	1600.00	1800.00	2000.00	2200.00
1200.00	11.93848	8.56504	6.56343	5.74663	5.41636	5.27636	5.20086	5.12753	5.02978
1000.00	14.55686	12.16045	11.30271	10.67506	10.01238	9.30591	8.59562	7.91514	7.28330
800.00	36.74972	28.20346	22.27792	18.09719	15.05803	12.78138	11.02941	9.64948	8.54055
600.00	47.74354	33.41337	25.06540	19.72862	16.07933	13.45493	11.49231	9.97872	8.78158
400.00	28.97499	22.51631	18.60425	15.76976	13.56961	11.81216	10.38587	9.21388	8.24074
200.00	21.50083	16.41624	13.12597	11.17894	9.89497	8.93120	8.14714	7.47970	6.89692
.00	16.26442	13.49679	11.03546	9.07290	7.72912	6.85387	6.24686	5.78621	5.41193
-200.00	12.15492	11.08240	9.54004	8.14529	6.90650	5.91829	5.21363	4.72666	4.37627
-400.00	9.05705	9.02110	8.21732	7.25327	6.37609	5.55798	4.83105	4.25117	3.82421
-600.00	6.97826	7.16433	7.02829	6.43278	5.78389	5.19093	4.62737	4.09296	3.62469
-800.00	5.54824	5.67197	5.85478	5.67077	5.22773	4.76855	4.34639	3.94270	3.54629
-1000.00	4.50924	4.57400	4.77493	4.88671	4.69781	4.36499	4.02874	3.71692	3.41807
-1200.00	3.73534	3.77658	3.90197	4.10454	4.14613	3.97282	3.71975	3.46742	3.23108
-1400.00	3.14569	3.17894	3.24903	3.41385	3.57478	3.56704	3.41583	3.22079	3.02780
-1600.00	2.68612	2.71182	2.76174	2.86455	3.03527	3.14348	3.10645	2.97736	2.82486

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24LTKOFC ***
INCLUDING SOURCE(S): 24LCTF00, 24LCTF01, 24LCTF02, 24LCTF03, 24LCTF04, 24LCTF05, 24LCTF06, 24LCTF07, 24LCTF08, 24LCTF09, 24LCTF10, 24LCTF11, 24LCTF12, 24LCTF13, 24LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS) 2400.00	2600.00
1200.00	4.90162	4.74719
1000.00	6.70759	6.18857
800.00	7.63397	6.88179
600.00	7.81483	7.02050
400.00	7.42463	6.73377
200.00	6.38043	5.91907
.00	5.09384	4.81479
-200.00	4.10698	3.88814
-400.00	3.51632	3.28787
-600.00	3.24987	2.96676
-800.00	3.17433	2.85265
-1000.00	3.11965	2.82627
-1200.00	3.00411	2.77578
-1400.00	2.84528	2.66950
-1600.00	2.67484	2.53178

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24LTKOFC ***
INCLUDING SOURCE(S): 24LCTF00, 24LCTF01, 24LCTF02, 24LCTF03, 24LCTF04, 24LCTF05, 24LCTF06, 24LCTF07, 24LCTF08, 24LCTF09, 24LCTF10, 24LCTF11, 24LCTF12, 24LCTF13, 24LCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
-574.60	-1575.60	2.85211	-1137.90	-2111.40	1.81161
-1374.20	-2214.30	1.61938	-150.00	-2110.00	2.20413
3365.00	-335.00	2.90861	2400.00	450.00	7.58416
-150.00	1415.00	16.22230	-155.00	1635.00	11.19214
-220.00	2135.00	6.13019	-135.00	2700.00	3.75820
1230.00	2595.00	2.51514	-1620.00	1535.00	4.33275
-2400.00	1715.00	2.12900	-2835.00	1440.00	2.35787
-2765.00	1100.00	4.29231	-515.00	-2930.00	1.51281
7130.00	960.00	1.84131	-3360.00	120.00	4.37209
2425.00	-420.00	3.44375	-2990.00	-1520.00	1.67614
-3624.60	-31.40	3.49375	-3547.00	2.60	3.69158
-3590.00	85.00	3.87790	-3663.10	57.00	3.69454
-4047.10	889.20	3.36375	-3868.40	816.50	3.72600
-3847.20	898.30	3.62765	-3750.30	858.90	3.85329
-3347.50	1095.00	3.61790	-3226.40	1016.30	4.17712
-3074.90	1016.30	4.41541	-3074.90	1058.70	4.15359
-2947.70	1143.50	3.76498	-2867.90	1007.30	4.83849
-2822.50	1028.40	4.76035	-2716.50	1001.20	5.18812
-2661.90	1176.80	3.83386	-2713.40	1331.20	2.86322
-2689.20	1388.70	2.63556	-2583.20	1343.30	2.91043
-2598.30	1300.90	3.10496	-2192.40	1140.50	4.81970
-1795.70	1125.30	6.31784	-1447.30	1355.40	6.36453
-1389.60	1276.70	7.63006	-1268.40	1346.30	7.96203
-1023.10	1064.80	17.05635	-965.60	1152.60	14.66658
-1204.90	1406.90	7.87866	-1029.00	1559.50	7.71793
-1032.00	1789.60	6.18995	-635.30	1789.60	7.56216
-635.30	1462.60	11.80530	-193.80	1465.40	14.52874
-193.10	1365.80	17.43299	-105.30	1365.80	18.01455
31.00	1429.30	14.75435	3.70	1489.90	13.52574
385.30	1499.00	9.37668	382.30	1172.00	16.01885
430.70	1060.00	18.77847	564.00	1060.00	14.30862
564.20	987.40	15.81875	642.90	999.50	13.77567
748.90	1202.30	9.34313	857.90	1202.30	7.80436
1309.20	1520.20	4.17120	1412.10	1377.90	3.94500
1481.80	1362.70	3.86304	1478.80	1287.10	4.36668
1545.40	1281.00	4.34805	1518.20	539.30	14.11691
558.20	533.30	43.91803	555.30	463.40	36.01505
282.70	463.40	65.57751	282.70	172.80	27.06049
549.20	175.80	22.18162	597.70	136.40	19.72831
2199.80	145.50	6.46917	2206.00	-315.20	3.99536

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24LTKOFC ***
INCLUDING SOURCE(S): 24LCTF00, 24LCTF01, 24LCTF02, 24LCTF03, 24LCTF04, 24LCTF05, 24LCTF06, 24LCTF07, 24LCTF08, 24LCTF09, 24LCTF10, 24LCTF11, 24LCTF12, 24LCTF13, 24LCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
2514.90	-318.20	3.58270	2521.00	-384.80	3.40507
2981.30	-390.80	2.99805	2981.30	-1093.40	2.33923
2763.20	-1090.40	2.59888	2763.20	-993.50	2.60328
2675.40	-993.50	2.72103	2672.40	-1093.40	2.71502
2542.20	-1096.50	2.88486	2545.20	-1499.30	2.64587
594.90	-1511.50	2.87621	591.80	-1447.90	3.02549
613.00	-1463.00	2.98889	634.20	-1453.90	3.01198
600.90	-1345.00	3.29303	600.90	-1099.70	4.09614
561.50	-1096.70	4.10521	549.40	-1393.40	3.16615
440.20	-1454.20	3.05752	125.20	-1457.20	3.27464
-398.70	-1454.20	3.24027	-998.80	-1448.10	2.81705
-2201.30	-1460.30	1.87101	-2534.50	-1514.70	1.69457

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24RTKOFC ***
INCLUDING SOURCE(S): 24RCTF00, 24RCTF01, 24RCTF02, 24RCTF03, 24RCTF04, 24RCTF05, 24RCTF06,
24RCTF07, 24RCTF08, 24RCTF09, 24RCTF10, 24RCTF11, 24RCTF12, 24RCTF13, 24RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-3000.00	-2800.00	-2600.00	-2400.00	-2200.00	-2000.00	-1800.00	-1600.00	-1400.00
1200.00	4.77835	5.18611	5.64589	6.17116	6.78405	7.55443	8.66665	10.69527	13.97676
1000.00	5.88376	6.63847	7.55319	8.67187	10.05591	11.80373	14.11866	17.78371	25.76148
800.00	6.37440	7.33192	8.57206	10.23476	12.56963	16.06980	21.85906	33.10105	62.97120
600.00	6.25290	7.13179	8.24427	9.69004	11.63455	14.37945	18.59617	26.29807	38.26226
400.00	5.46312	6.06339	6.78869	7.70419	8.94449	10.74848	13.18455	15.69878	19.16607
200.00	4.51860	4.98557	5.59852	6.41961	7.46927	8.59963	9.68833	10.94171	12.57486
.00	3.97273	4.45114	5.03618	5.68078	6.28898	6.84436	7.38926	7.96608	8.62505
-200.00	3.72072	4.12818	4.52462	4.86691	5.15920	5.39939	5.60594	5.77183	6.12858
-400.00	3.45874	3.70165	3.89609	4.04416	4.13353	4.17422	4.22645	4.35399	4.72624
-600.00	3.08703	3.19331	3.25588	3.26703	3.24224	3.25126	3.33338	3.52518	3.85776
-800.00	2.66286	2.67315	2.64604	2.60218	2.59575	2.66429	2.78617	2.97559	3.24064
-1000.00	2.22917	2.18696	2.14391	2.13588	2.19005	2.28845	2.40202	2.56228	2.78949
-1200.00	1.83958	1.80467	1.80034	1.84230	1.92228	2.00912	2.10123	2.24630	2.45374
-1400.00	1.54650	1.54695	1.58023	1.64302	1.71539	1.78047	1.86364	2.00550	2.18973
-1600.00	1.34996	1.37744	1.42608	1.48531	1.53970	1.59302	1.67919	1.81544	1.96905

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24RTKOFC ***
INCLUDING SOURCE(S): 24RCTF00, 24RCTF01, 24RCTF02, 24RCTF03, 24RCTF04, 24RCTF05, 24RCTF06,
24RCTF07, 24RCTF08, 24RCTF09, 24RCTF10, 24RCTF11, 24RCTF12, 24RCTF13, 24RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-1200.00	-1000.00	-800.00	-600.00	-400.00	-200.00	.00	200.00	400.00
1200.00	18.26114	22.13868	26.45845	31.74307	38.87683	49.31126	51.52214	33.25708	22.29949
1000.00	37.22122	49.40211	63.72725	78.27672	103.31950	143.34780	213.80760	83.79720	54.93077
800.00	143.40830	257.47060	170.61380	404.95910	333.33630	301.58220	276.75360	131.34140	70.05455
600.00	57.23160	65.20609	69.72035	70.31042	69.15298	68.28484	67.65754	56.58582	40.11699
400.00	23.60008	28.14090	30.54555	30.97225	31.63168	32.82908	32.76081	28.20029	26.29441
200.00	14.34451	15.32612	16.64871	17.98135	19.25232	20.09012	20.08760	17.64118	17.00507
.00	9.34610	9.86677	10.76609	11.93400	12.92900	13.77093	13.84252	12.46836	11.80814
-200.00	6.71568	7.22564	7.77584	8.58499	9.25603	10.00328	10.11747	9.36006	8.83681
-400.00	5.22863	5.64785	6.07667	6.50362	6.99986	7.59772	7.69563	7.26233	6.87189
-600.00	4.24798	4.62016	4.94345	5.16198	5.53095	5.97230	6.05612	5.79197	5.47832
-800.00	3.56864	3.89245	4.10887	4.26007	4.54011	4.82630	4.89403	4.73239	4.47465
-1000.00	3.07594	3.33220	3.47602	3.61752	3.84392	4.00346	4.04130	3.94055	3.73348
-1200.00	2.69189	2.88182	2.99211	3.13711	3.32570	3.40459	3.40579	3.33300	3.16746
-1400.00	2.37523	2.51583	2.61828	2.76460	2.91929	2.95594	2.92951	2.86170	2.72416
-1600.00	2.10773	2.21866	2.32483	2.46708	2.58977	2.60601	2.56772	2.49612	2.37262

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24RTKOFC ***
INCLUDING SOURCE(S): 24RCTF00, 24RCTF01, 24RCTF02, 24RCTF03, 24RCTF04, 24RCTF05, 24RCTF06,
24RCTF07, 24RCTF08, 24RCTF09, 24RCTF10, 24RCTF11, 24RCTF12, 24RCTF13, 24RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	600.00	800.00	1000.00	1200.00	1400.00	1600.00	1800.00	2000.00	2200.00
1200.00	15.07635	13.00116	12.11304	11.33867	10.51639	9.67554	8.86296	8.10779	7.42244
1000.00	39.29221	29.32978	22.82304	18.38086	15.21345	12.86931	11.07998	9.67839	8.55661
800.00	45.49095	32.37841	24.51710	19.40616	15.87397	13.31588	11.39368	9.90591	8.72618
600.00	27.66077	21.48015	17.82447	15.20113	13.15793	11.51280	10.16556	9.04956	8.11626
400.00	20.76401	15.96578	12.75335	10.83575	9.58834	8.66500	7.91986	7.28812	6.73674
200.00	15.76898	13.16081	10.81586	8.90864	7.57439	6.70100	6.10025	5.64907	5.28549
.00	11.82302	10.83417	9.35787	8.01767	6.81724	5.84197	5.13649	4.64722	4.29693
-200.00	8.84241	8.82983	8.07252	7.14132	6.29283	5.50006	4.78722	4.21011	3.78086
-400.00	6.83244	7.02095	6.90769	6.33920	5.70882	5.13234	4.58517	4.06308	3.60010
-600.00	5.44316	5.56731	5.75471	5.58899	5.16256	4.71493	4.30298	3.91003	3.52332
-800.00	4.43177	4.49699	4.69624	4.81460	4.63910	4.31705	3.98852	3.68349	3.39191
-1000.00	3.67688	3.71884	3.84245	4.04428	4.09267	3.92879	3.68290	3.43602	3.20449
-1200.00	3.10013	3.13453	3.20390	3.36636	3.52818	3.52642	3.38168	3.19157	3.00251
-1400.00	2.64963	2.67677	2.72655	2.82787	2.99687	3.10704	3.07482	2.95020	2.80112
-1600.00	2.29211	2.30790	2.35430	2.41673	2.54428	2.69415	2.75781	2.70949	2.60320

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24RTKOFC ***
INCLUDING SOURCE(S): 24RCTF00, 24RCTF01, 24RCTF02, 24RCTF03, 24RCTF04, 24RCTF05, 24RCTF06,
24RCTF07, 24RCTF08, 24RCTF09, 24RCTF10, 24RCTF11, 24RCTF12, 24RCTF13, 24RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS) 2400.00	2600.00
1200.00	6.80846	6.26205
1000.00	7.64222	6.88512
800.00	7.77157	6.98591
600.00	7.32888	6.65896
400.00	6.24741	5.80885
200.00	4.97826	4.71013
.00	4.02981	3.81408
-200.00	3.47050	3.24101
-400.00	3.22527	2.94008
-600.00	3.15737	2.83752
-800.00	3.10076	2.81301
-1000.00	2.98257	2.75971
-1200.00	2.82356	2.65143
-1400.00	2.65400	2.51362
-1600.00	2.48457	2.36833

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24RTKOFC ***
INCLUDING SOURCE(S): 24RCTF00, 24RCTF01, 24RCTF02, 24RCTF03, 24RCTF04, 24RCTF05, 24RCTF06,
24RCTF07, 24RCTF08, 24RCTF09, 24RCTF10, 24RCTF11, 24RCTF12, 24RCTF13, 24RCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
-574.60	-1575.60	2.51978	-1137.90	-2111.40	1.62201
-1374.20	-2214.30	1.45956	-150.00	-2110.00	1.97050
3365.00	-335.00	2.44994	2400.00	450.00	6.56228
-150.00	1415.00	25.94804	-155.00	1635.00	16.17276
-220.00	2135.00	7.70626	-135.00	2700.00	4.42906
1230.00	2595.00	2.92235	-1620.00	1535.00	5.53334
-2400.00	1715.00	2.57889	-2835.00	1440.00	3.37605
-2765.00	1100.00	6.08067	-515.00	-2930.00	1.39295
7130.00	960.00	1.85418	-3360.00	120.00	3.64332
2425.00	-420.00	3.17247	-2990.00	-1520.00	1.41923
-3624.60	-31.40	2.97389	-3547.00	2.60	3.12406
-3590.00	85.00	3.26586	-3663.10	57.00	3.12020
-4047.10	889.20	3.58509	-3868.40	816.50	3.92987
-3847.20	898.30	3.91669	-3750.30	858.90	4.13113
-3347.50	1095.00	4.58343	-3226.40	1016.30	5.13273
-3074.90	1016.30	5.57436	-3074.90	1058.70	5.39690
-2947.70	1143.50	5.26221	-2867.90	1007.30	6.32844
-2822.50	1028.40	6.38149	-2716.50	1001.20	6.99067
-2661.90	1176.80	5.71278	-2713.40	1331.20	4.26767
-2689.20	1388.70	3.85659	-2583.20	1343.30	4.34302
-2598.30	1300.90	4.69213	-2192.40	1140.50	7.72677
-1795.70	1125.30	10.43326	-1447.30	1355.40	9.00450
-1389.60	1276.70	11.64347	-1268.40	1346.30	11.64424
-1023.10	1064.80	35.60680	-965.60	1152.60	26.83572
-1204.90	1406.90	11.08118	-1029.00	1559.50	10.14783
-1032.00	1789.60	7.64445	-635.30	1789.60	10.17948
-635.30	1462.60	16.64898	-193.80	1465.40	22.40238
-193.10	1365.80	28.66409	-105.30	1365.80	30.20057
31.00	1429.30	23.18166	3.70	1489.90	20.77522
385.30	1499.00	12.97516	382.30	1172.00	24.41413
430.70	1060.00	34.54612	564.00	1060.00	30.30107
564.20	987.40	43.86666	642.90	999.50	36.83205
748.90	1202.30	13.20972	857.90	1202.30	12.58051
1309.20	1520.20	4.55335	1412.10	1377.90	6.03134
1481.80	1362.70	6.25999	1478.80	1287.10	7.91763
1545.40	1281.00	7.95410	1518.20	539.30	11.16097
558.20	533.30	26.63760	555.30	463.40	24.18412
282.70	463.40	33.42742	282.70	172.80	16.25087
549.20	175.80	15.72083	597.70	136.40	14.44446
2199.80	145.50	4.96518	2206.00	-315.20	3.63760

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 24RTKOFC ***
INCLUDING SOURCE(S): 24RCTF00, 24RCTF01, 24RCTF02, 24RCTF03, 24RCTF04, 24RCTF05, 24RCTF06,
24RCTF07, 24RCTF08, 24RCTF09, 24RCTF10, 24RCTF11, 24RCTF12, 24RCTF13, 24RCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
2514.90	-318.20	3.13527	2521.00	-384.80	3.05586
2981.30	-390.80	2.59115	2981.30	-1093.40	2.32963
2763.20	-1090.40	2.55248	2763.20	-993.50	2.57604
2675.40	-993.50	2.67640	2672.40	-1093.40	2.64287
2542.20	-1096.50	2.76986	2545.20	-1499.30	2.47618
594.90	-1511.50	2.44099	591.80	-1447.90	2.55721
613.00	-1463.00	2.52737	634.20	-1453.90	2.54416
600.90	-1345.00	2.76306	600.90	-1099.70	3.37095
561.50	-1096.70	3.37912	549.40	-1393.40	2.66947
440.20	-1454.20	2.59651	125.20	-1457.20	2.78211
-398.70	-1454.20	2.82402	-998.80	-1448.10	2.43922
-2201.30	-1460.30	1.65923	-2534.50	-1514.70	1.50602

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25LTKOFC ***
INCLUDING SOURCE(S): 25LCTF00, 25LCTF01, 25LCTF02, 25LCTF03, 25LCTF04, 25LCTF05, 25LCTF06, 25LCTF07, 25LCTF08, 25LCTF09, 25LCTF10, 25LCTF11, 25LCTF12, 25LCTF13, 25LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-3000.00	-2800.00	-2600.00	-2400.00	-2200.00	-2000.00	-1800.00	-1600.00	-1400.00
1200.00	.66659	.68149	.69763	.71664	.74046	.77113	.81009	.85822	.91771
1000.00	.73225	.75350	.77463	.79641	.82020	.84794	.88208	.92496	.97869
800.00	.80175	.82909	.85734	.88640	.91661	.94885	.98468	1.02640	1.07669
600.00	.90076	.92764	.95796	.99137	1.02770	1.06719	1.11054	1.15905	1.21457
400.00	1.07617	1.09668	1.12175	1.15173	1.18679	1.22716	1.27343	1.32693	1.38978
200.00	1.36171	1.38293	1.40643	1.43336	1.46478	1.50163	1.54472	1.59520	1.65527
.00	1.72824	1.77417	1.81993	1.86608	1.91352	1.96350	2.01741	2.07666	2.14258
-200.00	2.07657	2.17050	2.26800	2.36883	2.47288	2.58021	2.69130	2.80722	2.92961
-400.00	2.31258	2.45396	2.60826	2.77663	2.96033	3.16058	3.37865	3.61583	3.87371
-600.00	2.43345	2.60221	2.79135	3.00439	3.24553	3.51998	3.83406	4.19559	4.61415
-800.00	2.50196	2.68178	2.88500	3.11611	3.38095	3.68689	4.04357	4.46385	4.96512
-1000.00	2.53954	2.72098	2.92581	3.15849	3.42470	3.73172	4.08902	4.50911	5.00898
-1200.00	2.50454	2.67361	2.86254	3.07459	3.31385	3.58524	3.89484	4.25032	4.66123
-1400.00	2.36876	2.51039	2.66560	2.83605	3.02372	3.23088	3.46046	3.71637	4.00445
-1600.00	2.15585	2.26455	2.38156	2.50808	2.64597	2.79810	2.96929	3.16719	3.40377

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25LTKOFC ***
INCLUDING SOURCE(S): 25LCTF00, 25LCTF01, 25LCTF02, 25LCTF03, 25LCTF04, 25LCTF05, 25LCTF06, 25LCTF07, 25LCTF08, 25LCTF09, 25LCTF10, 25LCTF11, 25LCTF12, 25LCTF13, 25LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-1200.00	-1000.00	-800.00	-600.00	-400.00	-200.00	.00	200.00	400.00
1200.00	.99526	1.10052	1.23552	1.38511	1.53154	1.68292	1.86952	2.13658	2.48413
1000.00	1.04747	1.14185	1.27517	1.44589	1.62930	1.81109	2.01118	2.27980	2.67602
800.00	1.13863	1.21909	1.33494	1.50546	1.72200	1.95026	2.18975	2.48038	2.90305
600.00	1.27940	1.35647	1.45494	1.60098	1.82209	2.09692	2.39148	2.73662	3.21648
400.00	1.46456	1.55362	1.65891	1.79045	1.98629	2.28088	2.63264	3.04216	3.61925
200.00	1.72909	1.82274	1.94258	2.09259	2.28471	2.57405	2.98778	3.46356	4.13145
.00	2.21719	2.30520	2.41688	2.56828	2.77577	3.06539	3.53260	4.16062	4.94004
-200.00	3.06044	3.20158	3.35558	3.53061	3.75024	4.05489	4.51161	5.32181	6.38918
-400.00	4.15455	4.46200	4.80170	5.18122	5.61019	6.11269	6.76594	7.74827	9.50136
-600.00	5.10156	5.67223	6.34389	7.13885	8.08735	9.23556	10.66273	12.56415	15.71576
-800.00	5.57152	6.31744	7.25353	8.45724	10.05255	12.24879	15.42206	20.29065	28.31986
-1000.00	5.61203	6.35173	7.27725	8.46420	10.03531	12.20364	15.37719	20.46052	30.13410
-1200.00	5.13981	5.70217	6.37044	7.17813	8.18395	9.50890	11.41925	14.28284	17.55424
-1400.00	4.33436	4.72284	5.19980	5.81584	6.64723	7.76708	9.09358	10.39153	11.84614
-1600.00	3.69639	4.06817	4.54633	5.15215	5.86426	6.58036	7.23683	7.88621	8.60876

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25LTKOFC ***
INCLUDING SOURCE(S): 25LCTF00, 25LCTF01, 25LCTF02, 25LCTF03, 25LCTF04, 25LCTF05, 25LCTF06, 25LCTF07, 25LCTF08, 25LCTF09, 25LCTF10, 25LCTF11, 25LCTF12, 25LCTF13, 25LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	600.00	800.00	1000.00	1200.00	1400.00	1600.00	1800.00	2000.00	2200.00
1200.00	2.79244	2.99358	3.16520	3.39623	3.69389	3.99610	4.00723	3.79319	3.48862
1000.00	3.12088	3.47373	3.68460	3.89043	4.20960	4.60239	4.68913	4.42475	4.02040
800.00	3.44734	4.00414	4.37515	4.57024	4.89222	5.38028	5.58906	5.26064	4.71020
600.00	3.83997	4.56467	5.21883	5.54339	5.84383	6.41593	6.81522	6.41061	5.64477
400.00	4.39261	5.25820	6.17095	6.89172	7.26557	7.87215	8.55715	8.08404	6.97707
200.00	5.14143	6.27283	7.38249	8.59080	9.48934	10.09629	11.18693	10.68982	8.93848
.00	6.23221	7.76734	9.25472	10.88748	12.76268	13.94815	15.48193	15.01033	11.93047
-200.00	8.18873	10.35373	12.39335	14.77546	17.69160	21.11221	23.23624	22.99277	17.11410
-400.00	12.32171	15.93495	19.21444	22.91825	27.64245	33.90184	41.25717	42.16541	28.32659
-600.00	21.96305	30.73722	39.70395	49.49463	58.95297	73.75683	100.55830	129.19710	60.86219
-800.00	47.16338	86.75980	133.99340	243.58980	368.09080	303.61430	656.41030	582.43610	170.42510
-1000.00	52.17175	88.05396	91.83591	93.11026	90.68547	90.69072	86.99809	85.77605	68.98283
-1200.00	21.81479	27.37918	33.24539	36.15541	36.53146	36.79149	38.06639	37.89009	32.52516
-1400.00	13.82255	16.06163	17.51527	18.94772	20.20341	21.43380	22.30853	22.27078	19.45480
-1600.00	9.44217	10.29265	10.80935	11.83135	13.06466	14.15293	14.99576	15.03015	13.44644

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25LTKOFC ***
INCLUDING SOURCE(S): 25LCTF00, 25LCTF01, 25LCTF02, 25LCTF03, 25LCTF04, 25LCTF05, 25LCTF06, 25LCTF07, 25LCTF08, 25LCTF09, 25LCTF10, 25LCTF11, 25LCTF12, 25LCTF13, 25LCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS) 2400.00	2600.00
1200.00	3.07708	2.78922
1000.00	3.51351	3.22053
800.00	4.09392	3.80722
600.00	4.90942	4.60189
400.00	6.08553	5.63541
200.00	7.76247	6.95521
.00	10.16492	8.78814
-200.00	13.95606	11.41461
-400.00	20.56795	13.55566
-600.00	38.70940	31.17769
-800.00	80.20044	48.72557
-1000.00	44.10669	30.54946
-1200.00	29.15224	22.11677
-1400.00	18.81795	16.82704
-1600.00	12.78577	12.69281

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25LTKOFC ***
INCLUDING SOURCE(S): 25LCTF00, 25LCTF01, 25LCTF02, 25LCTF03, 25LCTF04, 25LCTF05, 25LCTF06, 25LCTF07, 25LCTF08, 25LCTF09, 25LCTF10, 25LCTF11, 25LCTF12, 25LCTF13, 25LCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
-574.60	-1575.60	5.29884	-1137.90	-2111.40	3.19488
-1374.20	-2214.30	2.81563	-150.00	-2110.00	3.84109
3365.00	-335.00	7.42121	2400.00	450.00	5.75033
-150.00	1415.00	1.61578	-155.00	1635.00	1.53392
-220.00	2135.00	1.32798	-135.00	2700.00	1.08134
1230.00	2595.00	1.82915	-1620.00	1535.00	.78928
-2400.00	1715.00	.60267	-2835.00	1440.00	.60284
-2765.00	1100.00	.72025	-515.00	-2930.00	1.71769
7130.00	960.00	.79807	-3360.00	120.00	1.45315
2425.00	-420.00	20.09154	-2990.00	-1520.00	2.25309
-3624.60	-31.40	1.62326	-3547.00	2.60	1.59705
-3590.00	85.00	1.47109	-3663.10	57.00	1.49731
-4047.10	889.20	.65477	-3868.40	816.50	.69799
-3847.20	898.30	.66951	-3750.30	858.90	.69233
-3347.50	1095.00	.66884	-3226.40	1016.30	.70299
-3074.90	1016.30	.71906	-3074.90	1058.70	.70574
-2947.70	1143.50	.68943	-2867.90	1007.30	.74375
-2822.50	1028.40	.74088	-2716.50	1001.20	.76185
-2661.90	1176.80	.70081	-2713.40	1331.20	.64410
-2689.20	1388.70	.62883	-2583.20	1343.30	.65157
-2598.30	1300.90	.66333	-2192.40	1140.50	.76279
-1795.70	1125.30	.83422	-1447.30	1355.40	.87058
-1389.60	1276.70	.90484	-1268.40	1346.30	.94009
-1023.10	1064.80	1.11272	-965.60	1152.60	1.12971
-1204.90	1406.90	.96051	-1029.00	1559.50	1.03193
-1032.00	1789.60	.99355	-635.30	1789.60	1.17539
-635.30	1462.60	1.27408	-193.80	1465.40	1.55855
-193.10	1365.80	1.60110	-105.30	1365.80	1.67730
31.00	1429.30	1.79083	3.70	1489.90	1.73559
385.30	1499.00	2.15904	382.30	1172.00	2.47842
430.70	1060.00	2.68270	564.00	1060.00	2.95593
564.20	987.40	3.06309	642.90	999.50	3.20901
748.90	1202.30	2.94497	857.90	1202.30	3.03562
1309.20	1520.20	2.96575	1412.10	1377.90	3.34646
1481.80	1362.70	3.47281	1478.80	1287.10	3.62685
1545.40	1281.00	3.73126	1518.20	539.30	6.52584
558.20	533.30	3.85780	555.30	463.40	4.03021
282.70	463.40	3.13705	282.70	172.80	3.77969
549.20	175.80	4.96525	597.70	136.40	5.42080
2199.80	145.50	9.62733	2206.00	-315.20	22.16301

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25LTKOFC ***
INCLUDING SOURCE(S): 25LCTF00, 25LCTF01, 25LCTF02, 25LCTF03, 25LCTF04, 25LCTF05, 25LCTF06, 25LCTF07, 25LCTF08, 25LCTF09, 25LCTF10, 25LCTF11, 25LCTF12, 25LCTF13, 25LCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
2514.90	-318.20	14.78679	2521.00	-384.80	15.56968
2981.30	-390.80	9.74423	2981.30	-1093.40	16.52072
2763.20	-1090.40	20.53244	2763.20	-993.50	25.26970
2675.40	-993.50	28.04265	2672.40	-1093.40	22.94065
2542.20	-1096.50	28.05990	2545.20	-1499.30	15.07762
594.90	-1511.50	11.11896	591.80	-1447.90	12.50876
613.00	-1463.00	12.36866	634.20	-1453.90	12.81371
600.90	-1345.00	15.45454	600.90	-1099.70	30.52987
561.50	-1096.70	28.95091	549.40	-1393.40	13.37980
440.20	-1454.20	11.13015	125.20	-1457.20	9.17637
-398.70	-1454.20	6.40763	-998.80	-1448.10	4.52445
-2201.30	-1460.30	2.91184	-2534.50	-1514.70	2.55419

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25RTKOFC ***
INCLUDING SOURCE(S): 25RCTF00, 25RCTF01, 25RCTF02, 25RCTF03, 25RCTF04, 25RCTF05, 25RCTF06,
25RCTF07, 25RCTF08, 25RCTF09, 25RCTF10, 25RCTF11, 25RCTF12, 25RCTF13, 25RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-3000.00	-2800.00	-2600.00	-2400.00	-2200.00	-2000.00	-1800.00	-1600.00	-1400.00
1200.00	.74734	.77026	.79308	.81633	.84113	.86921	.90296	.94490	.99736
1000.00	.82028	.84826	.87768	.90829	.94026	.97426	1.01158	1.05426	1.10488
800.00	.93229	.95797	.98760	1.02090	1.05770	1.09816	1.14294	1.19325	1.25085
600.00	1.13147	1.15081	1.17449	1.20311	1.23701	1.27651	1.32223	1.37549	1.43860
400.00	1.44115	1.46548	1.49130	1.51975	1.55205	1.58932	1.63252	1.68277	1.74199
200.00	1.81386	1.86931	1.92475	1.98045	2.03704	2.09556	2.15736	2.22399	2.29682
.00	2.14238	2.24826	2.35960	2.47627	2.59815	2.72521	2.85770	2.99636	3.14266
-200.00	2.34836	2.49799	2.66264	2.84403	3.04413	3.26499	3.50879	3.77789	4.07484
-400.00	2.45151	2.62381	2.81751	3.03649	3.28551	3.57048	3.89886	4.28008	4.72612
-600.00	2.51404	2.69508	2.89975	3.13268	3.39972	3.70843	4.06875	4.49384	5.00171
-800.00	2.53958	2.71963	2.92260	3.15276	3.41559	3.71796	4.06887	4.48001	4.96711
-1000.00	2.48210	2.64598	2.82840	3.03229	3.26115	3.51922	3.81166	4.14473	4.52629
-1200.00	2.32502	2.45911	2.60531	2.76505	2.93997	3.13213	3.34436	3.58079	3.84821
-1400.00	2.10134	2.20311	2.31259	2.43117	2.56111	2.70604	2.87165	3.06689	3.30456
-1600.00	1.86485	1.94518	2.03438	2.13581	2.25421	2.39583	2.56829	2.78004	3.03967

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25RTKOFC ***
INCLUDING SOURCE(S): 25RCTF00, 25RCTF01, 25RCTF02, 25RCTF03, 25RCTF04, 25RCTF05, 25RCTF06,
25RCTF07, 25RCTF08, 25RCTF09, 25RCTF10, 25RCTF11, 25RCTF12, 25RCTF13, 25RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-1200.00	-1000.00	-800.00	-600.00	-400.00	-200.00	.00	200.00	400.00
1200.00	1.06415	1.15522	1.28568	1.45864	1.65074	1.84226	2.04934	2.32007	2.72230
1000.00	1.16639	1.24461	1.35545	1.52185	1.74270	1.98252	2.23394	2.53469	2.96604
800.00	1.31784	1.39667	1.49418	1.63418	1.85078	2.13319	2.44119	2.80146	3.30118
600.00	1.51457	1.60640	1.71600	1.84974	2.04086	2.33501	2.69970	3.12336	3.72355
400.00	1.81415	1.90588	2.02539	2.17902	2.37555	2.66399	3.09315	3.59220	4.27937
200.00	2.37748	2.46948	2.58154	2.73047	2.93746	3.22776	3.69566	4.37246	5.19940
.00	3.29875	3.46699	3.64973	3.85219	4.09299	4.41450	4.88249	5.71317	6.88107
-200.00	4.40268	4.76563	5.17014	5.62604	6.14658	6.75387	7.51871	8.62580	10.64535
-400.00	5.25241	5.87874	6.63057	7.54082	8.65266	10.02670	11.76042	14.05751	17.70858
-600.00	5.61755	6.37754	7.33583	8.57693	10.24071	12.57635	16.07481	21.84889	32.98903
-800.00	5.55158	6.26350	7.14619	8.26440	9.71934	11.67899	14.45061	18.71576	26.52710
-1000.00	4.96635	5.47841	6.08242	6.81209	7.73255	8.97842	10.79158	13.25115	15.79376
-1200.00	4.15815	4.53054	4.99849	5.61250	6.43565	7.49028	8.62932	9.72669	10.99097
-1400.00	3.60186	3.97961	4.45876	5.04556	5.69365	6.30623	6.86585	7.41584	7.99948
-1600.00	3.35454	3.72581	4.13493	4.53379	4.87858	5.17334	5.41642	5.62622	5.79490

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25RTKOFC ***
INCLUDING SOURCE(S): 25RCTF00, 25RCTF01, 25RCTF02, 25RCTF03, 25RCTF04, 25RCTF05, 25RCTF06,
25RCTF07, 25RCTF08, 25RCTF09, 25RCTF10, 25RCTF11, 25RCTF12, 25RCTF13, 25RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	600.00	800.00	1000.00	1200.00	1400.00	1600.00	1800.00	2000.00	2200.00
1200.00	3.19399	3.59209	3.82682	4.02629	4.34828	4.76223	4.87256	4.59494	4.16239
1000.00	3.52688	4.12819	4.55646	4.76302	5.08110	5.58931	5.83534	5.49057	4.89876
800.00	3.94949	4.70429	5.42633	5.81846	6.11766	6.70244	7.15812	6.73653	5.90834
600.00	4.54480	5.45607	6.41208	7.25101	7.69066	8.29272	9.05954	8.57745	7.36139
400.00	5.34753	6.56315	7.73840	9.03802	10.14255	10.78366	11.97906	11.48660	9.51120
200.00	6.57716	8.21883	9.82296	11.59182	13.67951	15.22681	16.83811	16.39209	12.85120
.00	8.85072	11.24485	13.47712	16.06561	19.33040	23.37631	25.94552	25.85040	18.89909
-200.00	13.91514	18.15552	21.99871	26.28963	31.55897	38.66555	48.86131	50.80408	32.87680
-400.00	25.62152	36.92537	48.98595	63.01618	77.20370	101.54180	140.23160	205.88070	81.54204
-600.00	62.13608	139.25810	252.17510	169.79970	412.09880	345.24100	311.25800	285.13950	132.41350
-800.00	38.81554	58.38393	66.22207	70.63150	71.09663	70.01943	69.06152	68.38481	57.12566
-1000.00	19.30380	23.77449	28.38058	30.79477	31.21187	31.86469	33.06665	32.97724	28.39045
-1200.00	12.64408	14.42828	15.43009	16.75691	18.08882	19.35994	20.19797	20.17952	17.71930
-1400.00	8.66806	9.39255	9.91295	10.82088	11.99216	12.99136	13.83337	13.89355	12.50781
-1600.00	6.15409	6.74182	7.25234	7.80584	8.62070	9.29442	10.04283	10.15033	9.38461

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25RTKOFC ***
INCLUDING SOURCE(S): 25RCTF00, 25RCTF01, 25RCTF02, 25RCTF03, 25RCTF04, 25RCTF05, 25RCTF06,
25RCTF07, 25RCTF08, 25RCTF09, 25RCTF10, 25RCTF11, 25RCTF12, 25RCTF13, 25RCTF14,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS) 2400.00	2600.00
1200.00	3.63151	3.33909
1000.00	4.25612	3.96917
800.00	5.14218	4.81662
600.00	6.41971	5.90859
400.00	8.23559	7.31435
200.00	10.86642	9.32974
.00	15.19467	11.95263
-200.00	22.14364	14.94853
-400.00	53.81275	38.79804
-600.00	70.48300	45.61996
-800.00	40.21137	27.73307
-1000.00	26.41267	20.79871
-1200.00	17.09401	15.80899
-1400.00	11.85563	11.86617
-1600.00	8.86499	8.87494

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25RTKOFC ***
INCLUDING SOURCE(S): 25RCTF00, 25RCTF01, 25RCTF02, 25RCTF03, 25RCTF04, 25RCTF05, 25RCTF06,
25RCTF07, 25RCTF08, 25RCTF09, 25RCTF10, 25RCTF11, 25RCTF12, 25RCTF13, 25RCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
-574.60	-1575.60	4.65575	-1137.90	-2111.40	2.80125
-1374.20	-2214.30	2.52334	-150.00	-2110.00	2.86270
3365.00	-335.00	14.42596	2400.00	450.00	7.72064
-150.00	1415.00	1.74352	-155.00	1635.00	1.62244
-220.00	2135.00	1.40951	-135.00	2700.00	1.20479
1230.00	2595.00	1.99849	-1620.00	1535.00	.83167
-2400.00	1715.00	.64054	-2835.00	1440.00	.68081
-2765.00	1100.00	.81231	-515.00	-2930.00	1.50292
7130.00	960.00	1.04282	-3360.00	120.00	1.82648
2425.00	-420.00	58.33244	-2990.00	-1520.00	1.96156
-3624.60	-31.40	1.87047	-3547.00	2.60	1.87856
-3590.00	85.00	1.78384	-3663.10	57.00	1.78310
-4047.10	889.20	.78603	-3868.40	816.50	.85125
-3847.20	898.30	.78946	-3750.30	858.90	.82338
-3347.50	1095.00	.73977	-3226.40	1016.30	.78409
-3074.90	1016.30	.80347	-3074.90	1058.70	.78689
-2947.70	1143.50	.77284	-2867.90	1007.30	.83547
-2822.50	1028.40	.83304	-2716.50	1001.20	.85986
-2661.90	1176.80	.79517	-2713.40	1331.20	.73028
-2689.20	1388.70	.71094	-2583.20	1343.30	.73763
-2598.30	1300.90	.75283	-2192.40	1140.50	.87051
-1795.70	1125.30	.94135	-1447.30	1355.40	.92676
-1389.60	1276.70	.97027	-1268.40	1346.30	.98995
-1023.10	1064.80	1.19844	-965.60	1152.60	1.19003
-1204.90	1406.90	1.00105	-1029.00	1559.50	1.06606
-1032.00	1789.60	1.03244	-635.30	1789.60	1.24702
-635.30	1462.60	1.35338	-193.80	1465.40	1.67649
-193.10	1365.80	1.73771	-105.30	1365.80	1.81548
31.00	1429.30	1.91433	3.70	1489.90	1.84826
385.30	1499.00	2.40789	382.30	1172.00	2.71087
430.70	1060.00	2.96171	564.00	1060.00	3.32142
564.20	987.40	3.44227	642.90	999.50	3.65766
748.90	1202.30	3.49931	857.90	1202.30	3.66969
1309.20	1520.20	3.39450	1412.10	1377.90	3.87526
1481.80	1362.70	4.03961	1478.80	1287.10	4.24349
1545.40	1281.00	4.39108	1518.20	539.30	8.61169
558.20	533.30	4.58836	555.30	463.40	4.83048
282.70	463.40	3.65270	282.70	172.80	4.83067
549.20	175.80	6.36967	597.70	136.40	7.12709
2199.80	145.50	14.12153	2206.00	-315.20	50.57526

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: 25RTKOFC ***
INCLUDING SOURCE(S): 25RCTF00, 25RCTF01, 25RCTF02, 25RCTF03, 25RCTF04, 25RCTF05, 25RCTF06,
25RCTF07, 25RCTF08, 25RCTF09, 25RCTF10, 25RCTF11, 25RCTF12, 25RCTF13, 25RCTF14,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
2514.90	-318.20	27.30282	2521.00	-384.80	40.55175
2981.30	-390.80	22.79433	2981.30	-1093.40	11.90951
2763.20	-1090.40	15.29208	2763.20	-993.50	16.86218
2675.40	-993.50	18.95834	2672.40	-1093.40	16.89349
2542.20	-1096.50	19.33619	2545.20	-1499.30	10.20209
594.90	-1511.50	7.08517	591.80	-1447.90	7.91304
613.00	-1463.00	7.76811	634.20	-1453.90	7.96519
600.90	-1345.00	9.61088	600.90	-1099.70	15.43248
561.50	-1096.70	14.93358	549.40	-1393.40	8.56804
440.20	-1454.20	7.40942	125.20	-1457.20	6.69834
-398.70	-1454.20	5.48646	-998.80	-1448.10	3.90679
-2201.30	-1460.30	2.45514	-2534.50	-1514.70	2.17909

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: GSEGATES ***
INCLUDING SOURCE(S): TERMNL01, TERMNL02, TERMNL03, TERMNL04, TERMNL05, TERMNL06, TERMNL07,
TERMNL08, TBITSO__, TBITNO__, REMOTE_E, REMOTE_M, REMOTE_W,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-3000.00	-2800.00	-2600.00	-2400.00	-2200.00	-2000.00	-1800.00	-1600.00	-1400.00
1200.00	84692.02000	94705.51000	99345.13000	95690.01000	90570.13000	90649.48000	95561.38000	104004.50000	116415.90000
1000.00	107951.10000	119237.40000	126783.50000	120501.10000	111170.50000	110444.50000	113505.10000	121236.10000	133739.50000
800.00	146831.40000	160994.40000	173971.60000	163522.90000	148076.50000	145028.70000	144726.10000	148252.60000	155802.60000
600.00	202164.50000	235856.70000	256750.70000	239260.70000	213975.70000	204370.20000	198592.70000	194614.50000	202082.10000
400.00	296686.70000	393364.40000	428570.30000	375941.70000	314512.90000	280288.00000	265010.90000	277719.40000	317593.60000
200.00	659858.90000	924150.900001088010.00000		824390.70000	503245.90000	408333.00000	384605.80000	400346.50000	451539.40000
.00	8650625.00000**********			9895945.000003489685.000001071407.00000		688543.90000	552080.60000	502895.30000	507008.10000
-200.00	1072526.000009228844.000002948921.000002246860.00000					926655.10000	624075.20000	529851.60000	513115.00000 544641.00000
-400.00	407043.20000	538136.70000	620629.60000	591557.60000	547161.30000	463676.40000	436503.00000	458882.40000	518269.10000
-600.00	249691.20000	303304.70000	330617.30000	364825.60000	367662.50000	378898.90000	371941.60000	379294.30000	411462.90000
-800.00	188901.80000	219196.60000	236668.20000	253691.00000	264450.50000	284187.80000	293917.40000	300587.50000	319301.40000
-1000.00	153094.80000	171194.60000	184423.30000	191350.40000	198827.20000	213562.40000	232585.30000	246489.00000	260374.50000
-1200.00	127999.60000	141076.50000	151201.50000	155467.60000	158803.90000	172231.90000	187750.90000	200318.10000	205394.00000
-1400.00	110736.20000	121169.80000	128858.10000	132221.90000	133846.60000	140856.80000	149334.20000	156406.50000	159285.10000
-1600.00	97737.16000	105984.20000	111675.60000	113758.60000	113380.60000	113674.20000	116889.40000	121115.50000	125665.30000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: GSEGATES ***
INCLUDING SOURCE(S): TERMNL01, TERMNL02, TERMNL03, TERMNL04, TERMNL05, TERMNL06, TERMNL07,
TERMNL08, TBITSO__, TBITNO__, REMOTE_E, REMOTE_M, REMOTE_W,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-1200.00	-1000.00	-800.00	-600.00	-400.00	-200.00	.00	200.00	400.00
1200.00	131189.80000	148686.80000	175266.30000	205368.50000	225260.90000	232608.20000	236422.10000	218850.00000	191543.10000
1000.00	149922.40000	171723.20000	210526.90000	261849.40000	296171.20000	314053.20000	317019.20000	296764.40000	253282.20000
800.00	172841.90000	205505.60000	271597.40000	357898.80000	426265.60000	474377.80000	472443.80000	441602.40000	350409.60000
600.00	228099.30000	282522.10000	391861.30000	580640.70000	749664.40000	856469.20000	864110.90000	781726.00000	535710.10000
400.00	389159.30000	513167.70000	821371.500001571872.000002875160.000004707754.000004342322.000008208451.000001300368.00000						
200.00	562438.90000	821792.400001792066.000005428570.00000**********5029271.000004730745.000001613609.00000							
.00	567318.50000	719818.500001100993.000001979120.000001716864.000001823149.000001794884.000001756987.000001459112.00000							
-200.00	617971.20000	740349.900001046633.000003526080.000002955521.000004778026.00000**********9370309.00000							
-400.00	624436.40000	839524.400001407594.00000**********6024527.00000							
-600.00	482380.10000	618668.10000	891397.400001629346.000002272461.000002192477.000001974240.000001656949.000001307265.00000						
-800.00	356129.10000	409604.10000	477415.40000	588110.90000	696785.80000	760391.80000	786394.60000	755898.80000	684053.30000
-1000.00	274431.30000	286662.90000	306863.30000	348367.50000	389355.80000	418704.70000	451258.40000	456789.20000	437472.80000
-1200.00	207357.00000	210801.20000	222722.50000	246789.80000	271213.10000	284439.90000	297260.80000	306702.80000	302822.60000
-1400.00	159890.10000	164095.80000	175501.80000	193032.40000	209580.00000	217072.30000	219443.50000	222443.10000	221176.30000
-1600.00	129224.10000	134692.50000	144228.60000	157708.20000	170499.00000	175822.50000	174940.70000	172707.00000	170072.20000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: GSEGATES ***
INCLUDING SOURCE(S): TERMNL01, TERMNL02, TERMNL03, TERMNL04, TERMNL05, TERMNL06, TERMNL07,
TERMNL08, TBITSO__, TBITNO__, REMOTE_E, REMOTE_M, REMOTE_W,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	600.00	800.00	1000.00	1200.00	1400.00	1600.00	1800.00	2000.00	2200.00
1200.00	171957.60000	154182.70000	139055.60000	124213.80000	109805.60000	98645.36000	90597.48000	83515.33000	76103.46000
1000.00	218629.30000	187736.50000	162160.80000	139977.20000	124889.10000	114667.10000	105508.50000	96033.05000	87220.79000
800.00	282396.40000	228808.70000	192764.60000	172368.00000	158912.10000	146143.80000	132681.10000	120403.90000	110789.20000
600.00	388708.30000	321147.50000	286645.90000	257772.50000	228275.00000	199125.10000	174537.30000	155907.50000	141916.80000
400.00	797345.80000	587411.60000	457171.80000	362662.80000	292156.00000	243133.20000	210086.20000	186967.40000	169932.60000
200.00	960437.10000	674815.30000	499173.20000	386801.70000	318488.50000	275639.20000	246008.10000	223695.00000	205799.90000
.00	924611.40000	634979.60000	497487.40000	423675.40000	373338.30000	333232.00000	299419.80000	270291.40000	245003.10000
-200.00	1911758.000001046182.00000	738144.60000	568999.60000	461430.00000	387127.50000	332642.40000	290927.40000	257935.60000	
-400.00	2141195.000001138459.00000	776173.70000	582586.10000	460886.70000	378904.80000	321023.10000	278299.70000	245509.00000	
-600.00	992087.00000	735581.40000	555688.40000	445027.10000	371191.70000	317009.30000	275505.30000	243207.50000	217598.40000
-800.00	582856.20000	499489.50000	411734.90000	338275.90000	285350.00000	247939.00000	219988.80000	197937.50000	180138.90000
-1000.00	396978.00000	363691.90000	325081.10000	279338.30000	238553.90000	206136.50000	181570.80000	162951.20000	148393.20000
-1200.00	288343.70000	275227.70000	260917.60000	237737.90000	209583.30000	183592.00000	161531.40000	143558.30000	129294.30000
-1400.00	215062.70000	211902.40000	208712.00000	200885.00000	185300.80000	166542.30000	148774.00000	132990.70000	119360.90000
-1600.00	166434.80000	165880.80000	167057.80000	166679.00000	161362.40000	150300.60000	137235.40000	124593.30000	112951.50000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: GSEGATES ***
INCLUDING SOURCE(S): TERMNL01, TERMNL02, TERMNL03, TERMNL04, TERMNL05, TERMNL06, TERMNL07,
TERMNL08, TBITSO__, TBITNO__, REMOTE_E, REMOTE_M, REMOTE_W,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS) 2400.00	2600.00
1200.00	68767.30000	62545.96000
1000.00	80314.28000	75537.01000
800.00	103650.70000	98107.73000
600.00	130970.60000	122101.10000
400.00	156860.50000	146483.70000
200.00	190735.00000	177607.00000
.00	222981.30000	203771.30000
-200.00	231174.00000	209021.20000
-400.00	219548.60000	198477.60000
-600.00	196810.60000	179570.70000
-800.00	165568.80000	153397.80000
-1000.00	136734.40000	127284.50000
-1200.00	117980.50000	108913.50000
-1400.00	107947.60000	98572.61000
-1600.00	102436.80000	93196.65000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: GSEGATES ***
INCLUDING SOURCE(S): TERMNL01, TERMNL02, TERMNL03, TERMNL04, TERMNL05, TERMNL06, TERMNL07,
TERMNL08, TBITSO__, TBITNO__, REMOTE_E, REMOTE_M, REMOTE_W,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
-574.60	-1575.60	163371.30000	-1137.90	-2111.40	86331.52000
-1374.20	-2214.30	72970.35000	-150.00	-2110.00	115621.00000
3365.00	-335.00	147207.10000	2400.00	450.00	150001.00000
-150.00	1415.00	184462.20000	-155.00	1635.00	152907.10000
-220.00	2135.00	107409.40000	-135.00	2700.00	76741.38000
1230.00	2595.00	52637.01000	-1620.00	1535.00	85543.99000
-2400.00	1715.00	64941.24000	-2835.00	1440.00	75010.69000
-2765.00	1100.00	107602.80000	-515.00	-2930.00	65638.39000
7130.00	960.00	48207.75000	-3360.00	120.00	354343.10000
2425.00	-420.00	214921.10000	-2990.00	-1520.00	103059.20000
-3624.60	-31.40	300014.30000	-3547.00	2.60	334082.90000
-3590.00	85.00	278007.70000	-3663.10	57.00	264387.20000
-4047.10	889.20	62178.07000	-3868.40	816.50	71871.80000
-3847.20	898.30	67607.19000	-3750.30	858.90	74137.00000
-3347.50	1095.00	76479.16000	-3226.40	1016.30	90483.35000
-3074.90	1016.30	100313.10000	-3074.90	1058.70	94890.44000
-2947.70	1143.50	92823.56000	-2867.90	1007.30	114098.50000
-2822.50	1028.40	113496.70000	-2716.50	1001.20	124747.20000
-2661.90	1176.80	102186.10000	-2713.40	1331.20	86718.17000
-2689.20	1388.70	82714.66000	-2583.20	1343.30	85995.66000
-2598.30	1300.90	89657.88000	-2192.40	1140.50	95507.52000
-1795.70	1125.30	101370.30000	-1447.30	1355.40	102622.10000
-1389.60	1276.70	111359.30000	-1268.40	1346.30	113925.50000
-1023.10	1064.80	160994.60000	-965.60	1152.60	157783.10000
-1204.90	1406.90	113074.40000	-1029.00	1559.50	112113.80000
-1032.00	1789.60	95321.03000	-635.30	1789.60	118477.10000
-635.30	1462.60	156691.90000	-193.80	1465.40	175437.90000
-193.10	1365.80	192837.70000	-105.30	1365.80	194542.50000
31.00	1429.30	180376.40000	3.70	1489.90	171156.50000
385.30	1499.00	140715.90000	382.30	1172.00	200817.50000
430.70	1060.00	227117.80000	564.00	1060.00	208488.00000
564.20	987.40	228493.70000	642.90	999.50	211442.50000
748.90	1202.30	158118.00000	857.90	1202.30	149244.10000
1309.20	1520.20	96563.40000	1412.10	1377.90	100273.70000
1481.80	1362.70	97324.05000	1478.80	1287.10	100741.70000
1545.40	1281.00	97372.84000	1518.20	539.30	229317.50000
558.20	533.30	498149.70000	555.30	463.40	669153.50000
282.70	463.40	1269452.00000	282.70	172.80	2257915.00000
549.20	175.80	1042742.00000	597.70	136.40	911628.90000
2199.80	145.50	217458.30000	2206.00	-315.20	251973.60000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: GSEGATES ***
INCLUDING SOURCE(S): TERMNL01, TERMNL02, TERMNL03, TERMNL04, TERMNL05, TERMNL06, TERMNL07,
TERMNL08, TBITSO__, TBITNO__, REMOTE_E, REMOTE_M, REMOTE_W,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
2514.90	-318.20	212742.10000	2521.00	-384.80	207493.10000
2981.30	-390.80	168102.80000	2981.30	-1093.40	104681.90000
2763.20	-1090.40	111730.80000	2763.20	-993.50	121550.20000
2675.40	-993.50	124915.70000	2672.40	-1093.40	114731.40000
2542.20	-1096.50	119714.70000	2545.20	-1499.30	97899.21000
594.90	-1511.50	185641.60000	591.80	-1447.90	201726.10000
613.00	-1463.00	197315.30000	634.20	-1453.90	199336.60000
600.90	-1345.00	232301.80000	600.90	-1099.70	337285.50000
561.50	-1096.70	344090.00000	549.40	-1393.40	218460.60000
440.20	-1454.20	204056.60000	125.20	-1457.20	204889.20000
-398.70	-1454.20	197543.60000	-998.80	-1448.10	155885.90000
-2201.30	-1460.30	127467.70000	-2534.50	-1514.70	119588.20000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TAXIIDLE ***
INCLUDING SOURCE(S): TAXIRW24, TAXIRN25,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-3000.00	-2800.00	-2600.00	-2400.00	-2200.00	-2000.00	-1800.00	-1600.00	-1400.00
1200.00	35475.48000	40441.64000	44540.19000	48591.52000	52509.84000	56292.47000	60075.87000	63688.95000	66998.98000
1000.00	46163.47000	53136.34000	58932.74000	64499.47000	70051.20000	75790.95000	80973.66000	85297.13000	89226.52000
800.00	64501.86000	75624.52000	85365.03000	94579.52000	104022.10000	112505.50000	119788.00000	126515.30000	133430.70000
600.00	99153.16000	120800.70000	140557.10000	159918.50000	176611.90000	193220.70000	211599.20000	232212.80000	256317.30000
400.00	188889.00000	251841.50000	309403.10000	366097.20000	437394.30000	534173.30000	678021.40000	915524.600001388913.00000	
200.00	1224591.000003275321.000008043102.000002917102.000001576961.000001089210.00000	837330.60000	682980.20000	577863.10000					
.00	278892.00000	342980.90000	338395.60000	322250.30000	304614.20000	287259.00000	270990.50000	256714.50000	244996.10000
-200.00	120129.10000	138818.80000	155177.30000	161346.50000	161866.00000	160370.80000	158250.60000	156135.80000	155600.60000
-400.00	82203.70000	90925.07000	100686.00000	109518.70000	115510.00000	119212.20000	121794.20000	124400.40000	128735.90000
-600.00	64613.09000	71123.74000	77197.45000	85175.66000	93296.03000	101798.00000	111752.60000	125205.40000	145739.30000
-800.00	53690.20000	58797.97000	63442.49000	69248.41000	76786.90000	85672.55000	97333.61000	114788.30000	145285.50000
-1000.00	44983.99000	48781.43000	52088.25000	55667.23000	60467.00000	66423.33000	73720.74000	84173.47000	100568.90000
-1200.00	37120.93000	39759.99000	42028.16000	44491.15000	47845.99000	52841.80000	59346.65000	66227.87000	71848.15000
-1400.00	30910.24000	33153.03000	35339.55000	37847.82000	40899.88000	44632.50000	48258.61000	51180.16000	53950.07000
-1600.00	26871.03000	29065.63000	31235.15000	33430.71000	35456.72000	37092.57000	38670.55000	40283.48000	42120.70000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TAXIIDLE ***
INCLUDING SOURCE(S): TAXIRW24, TAXIRN25,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-1200.00	-1000.00	-800.00	-600.00	-400.00	-200.00	.00	200.00	400.00
1200.00	70141.35000	72847.37000	74865.05000	75900.43000	75073.36000	72076.38000	66379.87000	56546.99000	48810.30000
1000.00	93353.13000	97378.43000	101095.60000	103904.30000	104812.20000	101737.30000	93155.16000	75323.76000	62968.58000
800.00	141052.70000	148969.40000	157313.70000	165031.40000	171886.00000	175449.10000	162136.50000	118646.20000	89816.52000
600.00	285711.40000	321081.90000	364800.00000	416849.20000	482118.00000	564204.10000	649931.90000	283781.90000	197782.20000
400.00	2875972.000008135047.000003380800.000001715431.000001150391.00000					835625.10000	589892.80000	354293.20000	215053.30000
200.00	501958.40000	446143.70000	400866.30000	359521.30000	320475.20000	285291.40000	249031.20000	205432.70000	165331.10000
.00	234904.00000	228543.60000	222259.70000	213980.60000	206215.50000	197676.20000	186111.60000	166873.10000	146899.50000
-200.00	158104.10000	166531.00000	176120.20000	182352.70000	187253.00000	190095.30000	188275.00000	179377.60000	160364.60000
-400.00	141137.10000	172214.40000	208464.30000	237166.10000	260217.40000	277357.30000	293540.60000	308894.40000	302129.00000
-600.00	181806.90000	325374.70000	560669.10000	777466.800001157660.000002158785.000007643375.000004417993.000001662338.00000					
-800.00	214494.60000	473420.80000	680188.40000	607189.90000	527835.80000	457558.30000	392318.20000	329329.40000	271174.60000
-1000.00	117266.30000	144074.40000	172637.40000	193134.90000	192930.90000	183083.60000	170596.90000	157100.00000	140508.50000
-1200.00	78216.23000	87971.34000	97131.17000	103817.30000	109109.00000	110056.00000	107186.90000	101496.80000	93618.19000
-1400.00	57300.21000	62146.88000	66803.05000	69222.60000	73158.77000	75849.85000	76260.54000	74293.65000	70230.83000
-1600.00	44093.39000	47212.25000	50553.90000	52471.02000	54284.41000	56523.70000	57477.71000	57195.71000	55343.66000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TAXIIDLE ***
INCLUDING SOURCE(S): TAXIRW24, TAXIRN25,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	600.00	800.00	1000.00	1200.00	1400.00	1600.00	1800.00	2000.00	2200.00
1200.00	42395.95000	36697.27000	31724.93000	28553.82000	26918.72000	26037.50000	25490.53000	25058.96000	24598.61000
1000.00	52532.44000	44095.65000	40168.48000	38543.89000	37559.66000	36700.97000	35700.78000	34459.50000	32906.37000
800.00	73225.70000	67498.91000	63946.64000	60249.61000	56177.87000	52033.37000	47957.91000	43966.48000	40155.72000
600.00	146225.90000	115837.60000	95783.95000	81477.21000	70626.04000	61995.46000	54732.45000	48513.94000	43388.05000
400.00	158191.30000	124730.10000	102370.40000	86227.15000	73944.48000	63958.65000	55720.04000	49285.21000	44354.05000
200.00	129294.10000	106970.50000	91394.96000	79198.63000	68709.59000	59962.95000	53402.44000	48448.79000	44535.32000
.00	119601.90000	98230.29000	82238.96000	69529.33000	60625.89000	54999.04000	51035.75000	47973.83000	45436.61000
-200.00	125313.30000	99635.76000	79417.63000	68520.01000	62716.09000	58777.21000	55495.23000	52408.82000	49433.09000
-400.00	185123.00000	128705.90000	111611.50000	96545.32000	83378.53000	72698.41000	64292.09000	57656.18000	52319.57000
-600.00	466251.20000	229876.60000	152500.30000	114319.80000	91563.37000	76351.51000	65452.98000	57328.55000	51116.13000
-800.00	203611.20000	146264.30000	111932.50000	92340.47000	78566.00000	68025.14000	59668.05000	52928.64000	47467.36000
-1000.00	119167.40000	102322.90000	84291.15000	70664.85000	61261.89000	54586.57000	49371.56000	45007.94000	41265.85000
-1200.00	83338.52000	75435.70000	67210.02000	58390.05000	51010.73000	45148.92000	40804.52000	37474.36000	34762.29000
-1400.00	64444.49000	59676.54000	55523.05000	50249.87000	44850.92000	40030.08000	35908.43000	32628.22000	30071.29000
-1600.00	52205.24000	49302.28000	46826.70000	44006.68000	40323.16000	36586.12000	33127.51000	30046.32000	27440.08000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TAXIIDLE ***
INCLUDING SOURCE(S): TAXIRW24, TAXIRN25,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS) 2400.00	2600.00
1200.00	24003.42000	23230.28000
1000.00	31130.20000	29309.10000
800.00	36719.50000	33789.40000
600.00	39306.61000	36025.95000
400.00	40469.68000	37307.58000
200.00	41341.53000	38704.11000
.00	43207.08000	41152.45000
-200.00	46587.88000	43901.18000
-400.00	47935.76000	44260.07000
-600.00	46253.75000	42340.72000
-800.00	43014.83000	39377.69000
-1000.00	37995.91000	35167.11000
-1200.00	32399.99000	30325.55000
-1400.00	28021.51000	26302.66000
-1600.00	25315.00000	23599.31000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TAXIIDLE ***
INCLUDING SOURCE(S): TAXIRW24, TAXIRN25,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
-574.60	-1575.60	54269.49000	-1137.90	-2111.40	27567.46000
-1374.20	-2214.30	23812.39000	-150.00	-2110.00	32902.30000
3365.00	-335.00	34761.75000	2400.00	450.00	40227.80000
-150.00	1415.00	54698.89000	-155.00	1635.00	44328.40000
-220.00	2135.00	30787.93000	-135.00	2700.00	21202.97000
1230.00	2595.00	15519.79000	-1620.00	1535.00	43515.05000
-2400.00	1715.00	30058.46000	-2835.00	1440.00	31067.56000
-2765.00	1100.00	46791.82000	-515.00	-2930.00	18672.42000
7130.00	960.00	13158.91000	-3360.00	120.00	179897.00000
2425.00	-420.00	47371.12000	-2990.00	-1520.00	28369.78000
-3624.60	-31.40	83125.90000	-3547.00	2.60	96490.22000
-3590.00	85.00	103932.00000	-3663.10	57.00	90524.49000
-4047.10	889.20	25532.24000	-3868.40	816.50	30203.81000
-3847.20	898.30	27361.70000	-3750.30	858.90	30312.26000
-3347.50	1095.00	29854.95000	-3226.40	1016.30	36436.81000
-3074.90	1016.30	42158.56000	-3074.90	1058.70	39782.24000
-2947.70	1143.50	39516.62000	-2867.90	1007.30	50451.95000
-2822.50	1028.40	50233.89000	-2716.50	1001.20	55478.73000
-2661.90	1176.80	44525.29000	-2713.40	1331.20	36612.52000
-2689.20	1388.70	35020.50000	-2583.20	1343.30	38345.39000
-2598.30	1300.90	39784.93000	-2192.40	1140.50	56866.14000
-1795.70	1125.30	66708.50000	-1447.30	1355.40	54981.84000
-1389.60	1276.70	61134.96000	-1268.40	1346.30	58200.19000
-1023.10	1064.80	87424.59000	-965.60	1152.60	77926.12000
-1204.90	1406.90	55313.69000	-1029.00	1559.50	48964.87000
-1032.00	1789.60	39572.39000	-635.30	1789.60	40730.47000
-635.30	1462.60	55617.15000	-193.80	1465.40	52488.02000
-193.10	1365.80	58394.66000	-105.30	1365.80	56863.16000
31.00	1429.30	49577.90000	3.70	1489.90	47420.96000
385.30	1499.00	37438.97000	382.30	1172.00	50971.38000
430.70	1060.00	56200.55000	564.00	1060.00	50510.15000
564.20	987.40	55205.20000	642.90	999.50	50499.50000
748.90	1202.30	38059.08000	857.90	1202.30	35041.77000
1309.20	1520.20	22398.95000	1412.10	1377.90	22589.60000
1481.80	1362.70	22241.51000	1478.80	1287.10	23858.04000
1545.40	1281.00	23658.51000	1518.20	539.30	66869.17000
558.20	533.30	176465.50000	555.30	463.40	178606.70000
282.70	463.40	326952.70000	282.70	172.80	183282.10000
549.20	175.80	135239.60000	597.70	136.40	125447.80000
2199.80	145.50	44574.97000	2206.00	-315.20	51519.56000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TAXIIDLE ***
INCLUDING SOURCE(S): TAXIRW24, TAXIRN25,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
2514.90	-318.20	45853.36000	2521.00	-384.80	45697.03000
2981.30	-390.80	38720.99000	2981.30	-1093.40	29076.34000
2763.20	-1090.40	31194.87000	2763.20	-993.50	33294.34000
2675.40	-993.50	34353.46000	2672.40	-1093.40	32081.38000
2542.20	-1096.50	33475.82000	2545.20	-1499.30	25224.76000
594.90	-1511.50	57224.58000	591.80	-1447.90	61313.00000
613.00	-1463.00	59805.14000	634.20	-1453.90	59884.47000
600.90	-1345.00	68692.27000	600.90	-1099.70	97965.46000
561.50	-1096.70	101163.50000	549.40	-1393.40	66433.09000
440.20	-1454.20	64704.14000	125.20	-1457.20	69344.55000
-398.70	-1454.20	66945.83000	-998.80	-1448.10	57860.79000
-2201.30	-1460.30	39231.13000	-2534.50	-1514.70	33568.01000

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TESTCELL ***
INCLUDING SOURCE(S): TESTCELL,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-3000.00	-2800.00	-2600.00	-2400.00	-2200.00	-2000.00	-1800.00	-1600.00	-1400.00
1200.00	.04021	.04289	.04584	.04890	.05175	.05427	.05732	.06324	.07449
1000.00	.04132	.04391	.04698	.05046	.05419	.05778	.06104	.06496	.07251
800.00	.04355	.04559	.04835	.05183	.05595	.06051	.06508	.06936	.07447
600.00	.04803	.04919	.05108	.05390	.05774	.06257	.06818	.07407	.07976
400.00	.05490	.05574	.05679	.05845	.06115	.06522	.07078	.07767	.08533
200.00	.06240	.06420	.06570	.06704	.06864	.07113	.07519	.08135	.08973
.00	.06962	.07256	.07551	.07825	.08065	.08284	.08539	.08931	.09579
-200.00	.08124	.08365	.08673	.09039	.09445	.09856	.10244	.10612	.11041
-400.00	.10504	.10659	.10850	.11102	.11438	.11876	.12413	.13022	.13657
-600.00	.14038	.14437	.14819	.15187	.15554	.15942	.16391	.16948	.17664
-800.00	.17401	.18370	.19389	.20452	.21551	.22673	.23807	.24945	.26091
-1000.00	.19441	.20894	.22523	.24356	.26423	.28760	.31406	.34404	.37793
-1200.00	.20382	.22052	.23966	.26176	.28750	.31775	.35370	.39694	.44967
-1400.00	.20836	.22561	.24542	.26835	.29513	.32671	.36440	.40997	.46589
-1600.00	.20643	.22265	.24107	.26211	.28629	.31427	.34688	.38519	.43060

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TESTCELL ***
INCLUDING SOURCE(S): TESTCELL,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	-1200.00	-1000.00	-800.00	-600.00	-400.00	-200.00	.00	200.00	400.00
1200.00	.09053	.10559	.11166	.10795	.10466	.10925	.12299	.15306	.18701
1000.00	.08681	.10633	.12217	.12512	.11990	.12078	.13292	.16338	.20574
800.00	.08430	.10277	.12644	.14186	.14078	.13700	.14600	.17569	.22700
600.00	.08656	.09963	.12392	.15231	.16526	.16082	.16399	.19128	.25132
400.00	.09305	.10232	.12018	.15274	.18586	.19373	.19056	.21231	.27958
200.00	.09978	.11048	.12349	.14873	.19326	.22972	.23148	.24293	.31361
.00	.10569	.11890	.13411	.15305	.19024	.25227	.28834	.29181	.35756
-200.00	.11703	.12821	.14535	.16749	.19644	.25436	.34187	.37339	.42169
-400.00	.14304	.15077	.16308	.18463	.21747	.26457	.36183	.48566	.53297
-600.00	.18562	.19621	.20823	.22370	.25025	.29923	.38240	.56419	.74476
-800.00	.27280	.28589	.30141	.32075	.34552	.38220	.45574	.61926	1.02068
-1000.00	.41611	.45880	.50600	.55755	.61348	.67527	.74995	.87482	1.25059
-1200.00	.51503	.59754	.70400	.84491	1.03718	1.30927	1.71126	2.33483	3.35559
-1400.00	.53575	.62484	.74131	.89824	1.11776	1.43997	1.94456	2.81292	4.55164
-1600.00	.48494	.55075	.63177	.73413	.86925	1.06098	1.35674	1.78620	2.06678

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TESTCELL ***
INCLUDING SOURCE(S): TESTCELL,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS)								
	600.00	800.00	1000.00	1200.00	1400.00	1600.00	1800.00	2000.00	2200.00
1200.00	.19586	.19382	.19199	.17468	.14812	.13000	.12524	.12376	.11936
1000.00	.22129	.21941	.21561	.19221	.16123	.14462	.14144	.13784	.13164
800.00	.25248	.25124	.24435	.21270	.17768	.16391	.16058	.15394	.14615
600.00	.29132	.29165	.27986	.23709	.19947	.18895	.18286	.17303	.16351
400.00	.34050	.34425	.32445	.26711	.22960	.22049	.20929	.19634	.18610
200.00	.40402	.41485	.38157	.30617	.27193	.25941	.24194	.22702	.22142
.00	.48794	.51330	.45663	.36113	.33053	.30829	.28560	.27586	.27633
-200.00	.60193	.65751	.55900	.44488	.41005	.37471	.35678	.35280	.32516
-400.00	.76297	.88320	.70779	.57855	.52201	.48636	.47037	.40941	.31888
-600.00	1.00794	1.27088	.95104	.79538	.71771	.66672	.53174	.38981	.30546
-800.00	1.45808	2.04079	1.42930	1.20905	1.03797	.73166	.52881	.44563	.41432
-1000.00	2.50841	4.04382	2.65942	1.92891	1.24104	1.01245	.92976	.86725	.80294
-1200.00	5.53110	15.28694	7.67750	4.71981	3.30867	2.43941	1.87358	1.48878	1.21634
-1400.00	8.35959	14.54627	10.64692	5.65426	3.66527	2.60076	1.95629	1.53540	1.24466
-1600.00	2.23820	3.38532	3.73189	3.08977	2.05541	1.53396	1.26208	1.08124	.94228

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TESTCELL ***
INCLUDING SOURCE(S): TESTCELL,

*** NETWORK ID: CART1 ; NETWORK TYPE: GRIDCART ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

Y-COORD (METERS)	X-COORD (METERS) 2400.00	2600.00
1200.00	.11428	.10960
1000.00	.12565	.12005
800.00	.13897	.13357
600.00	.15619	.15417
400.00	.18276	.18539
200.00	.22353	.21918
.00	.26436	.23104
-200.00	.26963	.21330
-400.00	.24686	.20416
-600.00	.26428	.24427
-800.00	.40175	.39489
-1000.00	.73720	.67335
-1200.00	1.01657	.86562
-1400.00	1.03483	.87799
-1600.00	.82931	.73540

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TESTCELL ***
INCLUDING SOURCE(S): TESTCELL,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
-574.60	-1575.60	.77516	-1137.90	-2111.40	.33671
-1374.20	-2214.30	.28256	-150.00	-2110.00	.46003
3365.00	-335.00	.14269	2400.00	450.00	.17485
-150.00	1415.00	.10250	-155.00	1635.00	.09487
-220.00	2135.00	.07927	-135.00	2700.00	.07702
1230.00	2595.00	.09962	-1620.00	1535.00	.06566
-2400.00	1715.00	.04248	-2835.00	1440.00	.04125
-2765.00	1100.00	.04388	-515.00	-2930.00	.16396
7130.00	960.00	.03684	-3360.00	120.00	.06072
2425.00	-420.00	.23918	-2990.00	-1520.00	.20909
-3624.60	-31.40	.06420	-3547.00	2.60	.06305
-3590.00	85.00	.05925	-3663.10	57.00	.05961
-4047.10	889.20	.03810	-3868.40	816.50	.04003
-3847.20	898.30	.03816	-3750.30	858.90	.03920
-3347.50	1095.00	.03712	-3226.40	1016.30	.03888
-3074.90	1016.30	.04036	-3074.90	1058.70	.04006
-2947.70	1143.50	.04114	-2867.90	1007.30	.04293
-2822.50	1028.40	.04343	-2716.50	1001.20	.04513
-2661.90	1176.80	.04503	-2713.40	1331.20	.04343
-2689.20	1388.70	.04340	-2583.20	1343.30	.04511
-2598.30	1300.90	.04522	-2192.40	1140.50	.05268
-1795.70	1125.30	.05862	-1447.30	1355.40	.07384
-1389.60	1276.70	.07660	-1268.40	1346.30	.08692
-1023.10	1064.80	.10463	-965.60	1152.60	.10849
-1204.90	1406.90	.09139	-1029.00	1559.50	.09246
-1032.00	1789.60	.08119	-635.30	1789.60	.07593
-635.30	1462.60	.09063	-193.80	1465.40	.09828
-193.10	1365.80	.10214	-105.30	1365.80	.10722
31.00	1429.30	.11785	3.70	1489.90	.11273
385.30	1499.00	.16201	382.30	1172.00	.18717
430.70	1060.00	.20415	564.00	1060.00	.21295
564.20	987.40	.22266	642.90	999.50	.22107
748.90	1202.30	.19364	857.90	1202.30	.19373
1309.20	1520.20	.14087	1412.10	1377.90	.13692
1481.80	1362.70	.13029	1478.80	1287.10	.13443
1545.40	1281.00	.12883	1518.20	539.30	.20003
558.20	533.30	.30194	555.30	463.40	.31746
282.70	463.40	.22708	282.70	172.80	.26800
549.20	175.80	.39598	597.70	136.40	.42745
2199.80	145.50	.23458	2206.00	-315.20	.32629

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE ANNUAL (8760 HRS) AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: TESTCELL ***
INCLUDING SOURCE(S): TESTCELL,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	X-COORD (M)	Y-COORD (M)	CONC
2514.90	-318.20	.22277	2521.00	-384.80	.21809
2981.30	-390.80	.16686	2981.30	-1093.40	.62446
2763.20	-1090.40	.70785	2763.20	-993.50	.61734
2675.40	-993.50	.64227	2672.40	-1093.40	.75115
2542.20	-1096.50	.81992	2545.20	-1499.30	.85759
594.90	-1511.50	3.49310	591.80	-1447.90	5.58672
613.00	-1463.00	5.02801	634.20	-1453.90	5.51031
600.90	-1345.00	12.05382	600.90	-1099.70	3.31086
561.50	-1096.70	2.71505	549.40	-1393.40	7.46082
440.20	-1454.20	4.12985	125.20	-1457.20	2.15325
-398.70	-1454.20	1.06947	-998.80	-1448.10	.61645
-2201.30	-1460.30	.29440	-2534.50	-1514.70	.25151

**MODELOPTs: CONC URBAN FLAT DFAULT

*** THE SUMMARY OF MAXIMUM PERIOD (8760 HRS) RESULTS ***

** CONC OF OTHER IN MICROGRAMS/M**3 **

GROUP ID	AVERAGE CONC	NETWORK RECEPTOR (XR, YR, ZELEV, ZFLAG)	OF TYPE	GRID-ID
24LTKOFC	1ST HIGHEST VALUE IS	490.35720 AT (-600.00, 600.00, .00, .00)	GC	CART1
	2ND HIGHEST VALUE IS	385.49670 AT (-200.00, 600.00, .00, .00)	GC	CART1
24RTKOFC	1ST HIGHEST VALUE IS	404.95910 AT (-600.00, 800.00, .00, .00)	GC	CART1
	2ND HIGHEST VALUE IS	333.33630 AT (-400.00, 800.00, .00, .00)	GC	CART1
25LTKOFC	1ST HIGHEST VALUE IS	656.41030 AT (1800.00, -800.00, .00, .00)	GC	CART1
	2ND HIGHEST VALUE IS	582.43610 AT (2000.00, -800.00, .00, .00)	GC	CART1
25RTKOFC	1ST HIGHEST VALUE IS	412.09880 AT (1400.00, -600.00, .00, .00)	GC	CART1
	2ND HIGHEST VALUE IS	345.24100 AT (1600.00, -600.00, .00, .00)	GC	CART1
GSEGATES	1ST HIGHEST VALUE IS	14709740.00000 AT (-200.00, -400.00, .00, .00)	GC	CART1
	2ND HIGHEST VALUE IS	14466260.00000 AT (-400.00, -400.00, .00, .00)	GC	CART1
TAXIIDLE	1ST HIGHEST VALUE IS	8135047.00000 AT (-1000.00, 400.00, .00, .00)	GC	CART1
	2ND HIGHEST VALUE IS	8043102.00000 AT (-2600.00, 200.00, .00, .00)	GC	CART1
TESTCELL	1ST HIGHEST VALUE IS	15.28694 AT (800.00, -1200.00, .00, .00)	GC	CART1
	2ND HIGHEST VALUE IS	14.54627 AT (800.00, -1400.00, .00, .00)	GC	CART1

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLR
DC = DISCCART
DP = DISCPOLR
BD = BOUNDARY

**MODELOPTs: CONC URBAN FLAT DFAULT

*** Message Summary : ISCST3 Model Execution ***

--------- Summary of Total Messages --------

A Total of 0 Fatal Error Message(s)
A Total of 5 Warning Message(s)
A Total of 1120 Informational Message(s)

A Total of 1120 Calm Hours Identified

******** FATAL ERROR MESSAGES ********
*** NONE ***

******** WARNING MESSAGES ********
SO W320 158 APARM :Source Parameter May Be Out-of-Range for Parameter XINIT
SO W391 158 APARM :Aspect ratio (L/W) of area source greater than 10 TAXIRW24
SO W391 159 APARM :Aspect ratio (L/W) of area source greater than 10 TAXIRN25
SO W320 160 PPARM :Source Parameter May Be Out-of-Range for Parameter QS
OU W540 339 OUTQA :No RECTABLE/MAXTABLE/DAYTABLE for Average Period 01-HR

****
*** ISCST3 Finishes Successfully ***
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Attachment B5
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Attachment C
Toxicity Profiles

1,3-BUTADIENE[1]

Introduction

1,3-Butadiene is a colorless gas with a gasoline-like odor at room temperature. It is usually produced as a byproduct of ethylene production. 1,3-Butadiene is used in the production of rubber and plastics. The majority of 1,3-butadiene produced is used in the manufacture of styrene-butadiene rubber copolymers. There are many other polymer products that use butadiene as a starting point including polybutadiene, hexamethylene diamine, chloroprene, and nitrile rubbers. Butadiene is also used as a chemical intermediate in the manufacture of a number of commercial chemical products. Additionally, butadiene is found in automobile exhaust, gasoline vapor, fossil fuel incineration products, and cigarette smoke.

Potential for Human Exposure

Releases to the Environment

1,3-Butadiene may be released to the environment as intentional or fugitive emissions during production, use, storage, transport, or disposal. The majority of 1,3-butadiene is released to air. The Toxic Release Inventory (TRI) estimates that of the 9.6 million pounds released to air, soil, water, public treatment works, and offsite areas in 1987 from manufacturing and processing facilities in the United States, about 9 million pounds were released to air. TRI release estimates should not be considered total releases, as not all facilities are required to report releases and 1987 data represent first-time reporting by these facilities.

1,3-Butadiene is also released to air in motor vehicle exhaust, volatilization from gasoline, cigarette smoke, brush fire smoke, and thermal breakdown or burning of plastics.

Environmental Fate

1,3-Butadiene is highly volatile; therefore, it is expected to partition primarily to air. In air, 1,3-butadiene is removed rapidly (half-life of about 6 hours) by reaction with photochemically produced hydroxyl radicals. 1,3-Butadiene is also removed by the gas-phase reaction with ozone and by reaction at night with nitrate radicals in urban areas. In soil and water, 1,3-butadiene is primarily removed via rapid volatilization to air. Microbial degradation may also occur. 1,3-Butadiene is not expected to adsorb significantly to soil or sediment.

Environmental Levels

Air: Although atmospheric 1,3-butadiene undergoes rapid destruction, 1,3-butadiene is almost always present in urban and suburban air at low concentrations due to constant releases from vehicle exhaust. Median concentrations of 1,3-butadiene are 0.32 parts per billion (ppb) in suburban areas, 0.29 ppb in urban areas, and 0.10 ppb in rural areas.

Water: Data on the occurrence of 1,3-butadiene in water are limited. 1,3-Butadiene was detected in 1 of 2,045 water samples taken in 1975-1976 from surface waters near known industrialized areas across the United States. The single positive sample was obtained in the Carquinez Strait, Posta Corta, California, at a concentration of about 2 ppb.

Soil and Sediment: No data are available describing concentrations of 1,3-butadiene in soil or sediment.

Other Environmental Media: 1,3-Butadiene is used to manufacture synthetic rubber and plastics that are frequently used for food packaging. However, migration of the 1,3-butadiene monomer from packaging food is unlikely to occur. 1,3-Butadiene occurs in cigarette smoke; concentrations are not available. 1,3-Butadiene occurs in gasoline vapor at a concentration of 4.4 ppb.

Toxicokinetics

No studies were located regarding absorption of 1,3-butadiene in humans after inhalation exposure. Animal studies indicate that pulmonary absorption following inhalation exposure is rapid. No studies were located regarding absorption in humans or animals after oral or dermal exposure to 1,3-butadiene.

[1] Information pertaining to 1,3-Butadiene is derived from ATSDR, Toxicological Profile of 1,3-Butadiene, July 1992, as well as other sources, as noted.

No studies were located regarding distribution in humans after inhalation exposure to 1,3-butadiene. The distribution of 1,3-butadiene in several tissues in rats was measured following a 1-hour inhalation exposure to 129,000 parts per million (ppm). There was a high concentration of 1,3-butadiene in perinephric fat with low levels in the brain, liver, and kidney. These levels decreased with time; at 90 minutes following inhalation exposure, only trace levels of 1,3-butadiene could be found. No studies could be found regarding distribution following exposure via oral or dermal routes in humans or animals.

Butadiene is metabolized extensively in humans as well as other animals. One of the major metabolite of 1,3-butadiene is 1,2-epoxybutene-3. The amount of 1,2-epoxybutene-3 formed by metabolism in human liver was comparatively lower than the amount formed from livers of rats and mice. These species differences in the metabolism of 1,3-butadiene to the epoxide suggest differences between humans and rodents in the expression of 1,3-butadiene toxicity.

1,2-Epoxybutene-3 is transformed into 3-butene-1, 2-diol by microsomal epoxide hydrolase. In the metabolism of 1,2-epoxybutene-3 in microsomes, two stereoisomers of DL-diepoxybutane, and two stereoisomers of 3,4-epoxy-1,2-butanediol were detected as further metabolites.

No studies were located regarding the excretion of 1,3-butadiene in humans following inhalation exposure to 1,3-butadiene. Animal studies indicate that metabolites of 1,3-butadiene are exhaled rapidly, with half times of between 2 and 10 hours.

About 2 percent of the total inhaled amount of 1,3-butadiene was excreted as its metabolites in Cynomolgus monkeys. Carbon dioxide was the major exhalatory product at low exposure levels, while epoxy-metabolites were exhaled at higher levels. Urinary excretion of total metabolites was not influenced by exposure levels. In *Macaca fascicularis* monkeys, about 39 percent of metabolites was examined in the urine, 0.8 percent in feces, and 56 percent was exhaled as carbon dioxide during the first 70 hours of postexposure. No studies were located regarding excretion in humans or animals after oral or dermal exposure to 1,3-butadiene.

Qualitative Description of Health Effects

Narcosis and death from respiratory paralysis may occur in humans and animals after inhalation exposure to very high concentrations of 1,3-butadiene. 1,3-Butadiene concentrations resulting in death in humans from acute exposure were not reported; acute inhalation exposure of rabbits to 250,000 ppm 1,3-butadiene resulted in the death of the majority of animals within an average of 23 minutes.

An early occupational study reported complaints of irritation of the eyes, nasal passages, throat, and lungs in rubber manufacturing workers following acute exposures to unknown levels of 1,3-butadiene. Additional symptoms included coughing, fatigue, and drowsiness. However, all symptoms abated upon removal from the exposure. Epidemiological studies suggest a possible risk of harmful effects associated with exposure to 1,3-butadiene as evidenced by a higher incidence of cardiovascular and hamatopoietic diseases, respiratory diseases, and cancer among exposed workers; however, exposures were not to 1,3-butadiene exclusively. In animals, effects include increased mortality, anemia, respiratory lesions, liver necrosis, nephrosis, and cancer. Fetotoxic and reproductive effects have been observed in mice after exposure to 1,3-butadiene.

EPA has classified 1,3-butadiene as a B2, probably human carcinogen, based on rodent studies in which exposure to airborne concentrations of 1,3-butadiene caused multiple tumors and tumor types. Compounds related to 1,3-butadiene are carcinogenic and mutagenic.[2]

Quantitative Description of Health Effects

EPA has assigned 1,3-butadiene classification B2; probable human carcinogen. This classification was based on inadequate human data and sufficient rodent (rat and mouse) studies in which exposure to airborne concentrations of 1,3-butadiene caused multiple tumors and tumor types. The EPA has assigned a unit risk factor of 2.8×10^{-4} (micrograms per cubic meter [$\mu g/m^3$])$^{-1}$. The inhalation slope factor is 0.98 (milligrams per kilogram per day [mg/kg-day])$^{-1}$.[3] CalEPA has assigned an inhalation and oral cancer

[2] U.S. Environmental Protection Agency, Integrated Risk Information Database (IRIS), 1,3-Butadiene, IRSN 136, February 2000.

[3] U.S. Environmental Protection Agency, Integrated Risk Information Database (IRIS), 1,3-Butadiene, IRSN 136, February 2000.

potency factor of 0.6 x 10^0 $(mg/kg\text{-}day)^{-1}$.[4] Data used by EPA[5] to develop the oral unit risk are described below.

There are minimal human epidemiological data available. Of the three studies on workers specifically identified as being exposed to 1,3-butadiene, two were cohort studies while one was a cross-sectional study designed to look at certain hematologic parameters. One of the cohort studies was a mortality study of 14,000 workers at eight plants, and it found none of the Standard Mortality Ratios (SMR) for cancer to be significantly elevated.[6] The second cohort study found an increase of borderline significance in the SMR for lymphatic and hematopoietic cancer in a subpopulation.[7] The cross-sectional study found no evidence of hematologic effects.[8] Two studies found an association between employment in the synthetic rubber industry and an elevated risk of cancer. Synthetic rubber is manufactured from styrene and butadiene. In one case-control study, synthetic rubber plan workers were found to have an increased risk ratio for deaths from lymphatic and hematopoietic cancer.[9] The second, a cohort mortality study, found excess lung cancer deaths among workers in a synthetic rubber area of a plant. This latter finding was based on three deaths with no control for smoking.[10] Given the inconsistency of results, the methodological limitations of the different studies, and the confounding effects of exposure to various solvents, styrene, and possibly other chemicals, the epidemiologic evidence is considered inadequate.

Animal carcinogenicity data are sufficient to determine the carcinogenic potential of 1,3-butadiene. Two lifetime inhalation studies of 1,3-butadiene in rodents were initiated. B6C3F1 mice (50/sex/group) were exposed to 625 or 1,250 ppm for 6 hours per day, 5 days per week. Exposure began at 8 to 9 weeks of age, and all mice were killed after weeks 60 to 61 because of excessive deaths among treated mice. Increases were observed in the number of mice with primary tumors and in the number of mice with multiple primary tumors. Tumors occurring through the body included hemangiosarcomas of the heart, lymphomas, and alveolar/bronchiolar adenomas/ carcinomas.[11]

Charles River CD rats (110/sex/group) were exposed to 1,000 or 8,000 ppm 1,3-butadiene for 6 hours per day, 5 days per week for 111 weeks (males) or 105 weeks (females). There was a treatment-related increase in mortality, some of which was attributed to nephropathies in males. Significant increases occurred in incidence in both common and uncommon tumors including mammary gland tumors, thyroid follicular adenomas and carcinomas, and Leydig cell adenomas and carcinomas.[12] Because of problems with reporting of this study and because pharmacokinetic analysis indicated that the effective doses were the same for both treatment groups, this study was not considered adequate for the estimation of risk.

Additionally, three studies have shown 1,3-butadiene to be mutagenic for Salmonella typhimurium upon addition of mammalian hepatic homogenates for metabolism.[13] Pharmacokinetic and various types of toxicity studies indicate that the carcinogenic effect of 1,3-butadiene can be attributed to the metabolites 3,4-epoxybutane and/or 1,2,3,4-diepoxybutane. These metabolites, which are potent alkylating agents,

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4 California Environmental Protection Agency, California Cancer Potency Factors, Standards and Criteria Workgroup, November 1994.

5 U.S. Environmental Protection Agency, Integrated Risk Information Database (IRIS), 1,3-Butadiene, IRSN 136, February 2000.

6 Matanoski, G.M., L. Schwartz, J. Sperraza, and J. Tonascia, Mortality of Workers in the Styrene-Butadiene Rubber Polymer Manufacturing Industry, 1982.

7 Meinhardt, T.J., R.A. Lemen, M.S. Crandall, and R.J. Young, Environmental Epidemiologic Investigation of the Styrene-Buadiene Rubber Industry, Scand. J. Work Environ. Health, 1982

8 Checkoway, H. and T.M. Williams, A Hematology Survey of Workers at a Styrene-Butadiene Synthetic Rubber Manufacturing Plant. Am. Ind. Hyg. Assoc, 1982.

9 McMichael, A.J., R. Spritas, J.F. Gamble, and P.M. Tousey, Mortality among Rubber Workers: Relationship to Specific Jobs. J. Occup. Med., 1976.

10 Andjelkovich, D.J. Taulbee, M. Symons, and T. Williams, Mortality of Rubber Workers with Reference to Work Experience. J. Occup. Med. 1977

11 National Toxicology Program, Toxicology and Cardcinogenesis Studies of 1,3-Butadiene (CAS 106-99-0) in B6C3F1 Mice (Inhalation Studies), 1984

12 Hazelton Laboratories Europe, Ltd., The Toxicity and Cardinogenicity of Butadiene Gas Administered to Rats by Inhalation for Approximately 24 Months, Unpublished 1981

13 de Meester, C., F. Poncelet, F. Roberfroid, and M. Mercier, The Mutagenicity of Butadiene towards Salmonella Typhimurium. Toxicol. Lett, 1980

have been shown to be mutagenic and carcinogenic.[14, 15, 16, 17, 18, 19, 20, 21] 1,3-Butadiene is structurally related to known carcinogens.

In development of the inhalation unit risk, animals dying before onset of first tumor (20 weeks) were eliminated. An adjustment was made for early sacrifice in the calculation. The concentration in ppm is assumed to be equivalent for the experimental animals and humans. In determining the animal-to-equivalent-human dose, an adjustment was made to account for the lack of proportionality to external concentration at high levels. The function for the incremental cancer risk to the animals was based on a calculated internal dose (in mg/kg) and converted back to risk for low-dose ppm equivalents in the animal. Animal upper-limit slope factors of 6.1E-1 per (mg/kg)/day for males, and 3.0E-1 per (mg/kg)/day for females were reconverted to air concentration units of 9.2E-1 per ppm and 4.5E-1 per ppm by assuming a 20 percent absorption rate at low exposures. Data from Bond[22] support the assumption that in mice and rats exposed to 13 µg 1,3-butadiene/liter air or less, absorption will be 20 percent.

The quantitative estimates of 1.8E+0 per (mg/kg)/day or 6.4E-1 per ppm is a geometric mean of slope factors derived form the male and female mouse data sets. This is a correction from[23] in which preliminary data was used to calculate the unit risk[24].

The unit risk should not be used if the air concentration exceeds 16 µg/m^3, since above this concentration the unit risk may not be appropriate.

No oral or inhalation reference doses are currently available for 1,3-butadiene. EPA has not developed any drinking water criteria for 1,3-butadiene.

Summary of 1,3-Butadiene Criteria

Criteria	Value	Source
EPA Carcinogenic Classification	B2	EPA 2000
Inhalation Slope Factor	0.98×10^{0} $(mg/kg/day)^{-1}$	EPA 2000
Inhalation Unit Risk Factor	2.8×10^{-4} $(\mu g/m^3)^{-1}$	EPA 2000
CalEPA Inhalation Potency Factor	0.6×10^{0} $(mg/kg/day)^{-1}$	CalEPA 1994
CalEPA Oral Potency Factor	0.6×10^{0} $(mg/kg/day)^{-1}$	CalEPA 1994
Cal	0.6×10^{0} $(mg/kg/day)^{-1}$	
Cal Permissible Exposure Limits, PEL	2.2 mg/m^3	CCR, Title 8, 2000[1]
Cal Permissible Exposure Limits, STEL	11 mg/m^3	CCR, Title 8, 2000[1]

[1] California Code of Regulations, Title 8, Section 5155, February 16, 2000.

[14] Lawley, P.D. and P. Brookes, Interstrand Cross-Linking of DNA by Difunctional Alkylating Agents. J. Mol. Biol., 1967

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[16] Dean, B.M. and G. Hodson-Walker, An in Vitro Chromosome Assay Using Cultured Rat-Liver Cells. Mutat. Res, 1979.

[17] Perry, P. and H. J. Evans, Cytological Detection of Mutagen-Carcinogen Exposure by Sister Chromatid Exchange, 1975.

[18] Wade, M.J., J. W. Moyer, and C.H. Hine, Mutgenic Action of a Series of Epoxides. Mutat. Res. 66, 1979

[19] Voogd, C.E., J.J. van de Stel, and J.A. Jacobs, The Mutagenic Action of Aliphatic Epoxides. Mutat Res., 1981

[20] Conner, M., J. Lou, and O. Gutierrez de Gotera, Induction and Rapid Repair of Sister-Chromatid Exchanges in Multiple Murine Tissues in Vitro by Diepoxybutane. Mutat. Res., 1983

[21] U.S. Environmental Protection Agency, Mutagenicity and Carcinogenicity Assessment Document for 1,3-Butadiene, 1985

[22] Bond, J.A., A.R. Dahl, R.J. Henderson, G.S. Dutcher, J.L. Mauderly, and L.S. Birnbaum, Species Differences in the Dispostion of Inhaled Butadiene, July.

[23] U.S. Environmental Protection Agency, Mutagenicity and Cardinogenicity Assesment Document for 1,3-Butadiene, 1985.

[24] Cote, I.L. and S.P. Bayard, Cancer Risk Assessment of 1,3-Butadiene. Environ. Health Perspec., 1990.

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ACETALDEHYDE

Introduction

Acetaldehyde is a colorless, volatile liquid with a characteristic sweet, pungent odor. It is a highly flammable and reactive compound that is miscible in water and most common solvents.[1] Acetaldehyde is used primarily as a chemical intermediate in the production of acetic acid. The second most significant use is the production of esters, primarily ethyl acetate and isobutyl acetate.[2] It is also used in the production of pyridine, pentaerythritol, and peracetic acid and in silvering mirrors, hardening gelatin fibers, denaturing alcohol, and in the manufacture of disinfectants, dyes, explosives, flavorings, rubber accelerators, and varnishes.[3, 4]

Acetaldehyde occurs naturally in certain foods, such as ripe fruits and coffee, and is found in cigarette smoke.[5] Acetaldehyde is a metabolic intermediate in humans and higher plants and is a product of alcohol fermentation.[6]

Potential for Human Exposure

Releases to the Environment

Acetaldehyde is released into air or wastewater from facilities producing or using this chemical. In 1992, the Toxic Chemical Release Inventory (TRI) reported that certain types of U.S. industries released approximately 8.4 million pounds of acetaldehyde to environmental media. Not all industries report releases to TRI, therefore, this value should not be regarded as total releases. Of reported releases, 6.42 million pounds were to the air, 1.9 million pounds were to underground injection sites, 77,188 pounds were discharged to surface water, and 289 pounds were to land.[7]

Acetaldehyde is also released to the environment in vehicle exhaust and as a product of open burning of gas, fuel oil, and coal.[8] Degradation of hydrocarbons, sewage, and solid biological wastes produces acetaldehyde. Environmental exposure can also occur when people eat fruit, drink coffee or alcohol, or smoke cigarettes.[9, 10]

Environmental Fate

The majority of direct releases of acetaldehyde are to air and underground sites. In air, acetaldehyde reacts with other chemicals in air or undergoes photolysis; products of acetaldehyde reaction with other

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1 World Health Organization (WHO). Acetaldehyde. Report No. 167. 1995.

2 U.S. Environmental Protection Agency. Chemical Summary for Acetaldehyde. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003a. 1994.

3 U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

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8 Verschueren, K. Handbook of Environmental Data on Organic Chemicals. Second Edition. New York: Van Nostrand Reinhold Company. 1993.

9 U.S. Environmental Protection Agency. Chemical Summary for Acetaldehyde. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003a. 1994.

10 World Health Organization (WHO). Acetaldehyde. Report No. 167. 1995.

chemicals in air include peroxyacetylnitrate, methyl nitrate, methyl nitrite, and nitric acid.[11] Acetaldehyde can contribute to the formation of photochemical smog when it reacts with other volatile organic carbon substances in air.[12]

Acetaldehyde is highly volatile. The majority of acetaldehyde released to surface soil and water will volatilize to air. Acetaldehyde in soil and water can also undergo microbial degradation. Acetaldehyde does not bind well to soil; released to soil it may leach into the ground and can enter groundwater. Bioaccumulation and bioconcentration of acetaldehyde in plants and animals is unlikely to occur.[13, 14]

Environmental Levels

Air: Concentrations of acetaldehyde in air vary depending on several conditions, such as weather (i.e., smog). Acetaldehyde concentrations of 590 parts per billion (ppb) were detected in clouds over California. Acetaldehyde concentrations of 35 ppb were measured in the air of Claremont, California; severe smog conditions were reported at the time.[15]

Acetaldehyde concentrations in air were characterized for Pico Rivera, a Los Angeles suburb, for the summer of 1994 in the 1995 National Air Quality and Emissions Trends Report.[16] The mean concentration of acetaldehyde in air in Pico Rivera in the summer of 1994 was 7.0 ppb (494 observations). The maximum concentration of acetaldehyde detected in air of Pico Rivera in the summer of 1994 was 22.6 ppb.

Water: Trace amounts of acetaldehyde have been detected in the drinking water of several U.S. cities.[17] Concentrations in water are generally less than 0.1 micrograms per liter (µg/L).[18]

Soil and Sediment: No information was available describing acetaldehyde concentrations in soil or sediments. Due to volatilization, microbial degradation, and leaching, it is unlikely acetaldehyde would be present in soil or sediments in significant concentrations.

Other Environmental Media: The general population may be exposed to acetaldehyde through metabolism of alcohol, inhalation of cigarette smoke, ingestion of food and beverages containing acetaldehyde, and inhalation of contaminated air. The contribution from drinking water is negligible.[19]

Toxicokinetics

Studies of the kinetics of acetaldehyde are limited. Toxicity studies indicate it is rapidly absorbed through inhalation and ingestion. Absorption through the skin is probable. Experimental studies indicate that sufficient first-pass metabolism occurs in the liver and respiratory tract following ingestion and inhalation exposure to limit acetaldehyde access to systemic circulation.[20] However, acetaldehyde was detected in

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[11] U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

[12] U.S. Environmental Protection Agency. Chemical Summary for Acetaldehyde. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003a. 1994.

[13] U.S. Environmental Protection Agency. Chemical Summary for Acetaldehyde. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003a. 1994.

[14] U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

[15] U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

[16] U.S. Environmental Protection Agency. National Air Quality and Emissions Trends Report, 1995. Office of Air Quality Planning and Standards. Washington, D.C. 1995.

[17] U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

[18] World Health Organization (WHO). Acetaldehyde. Report No. 167. 1995.

[19] World Health Organization (WHO). Acetaldehyde. Report No. 167. 1995.

[20] U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003.

the blood, liver, kidney, spleen, heart, and other muscle tissues following inhalation exposure. Low levels of acetaldehyde were detected in embryos following maternal intraperitoneal injection (pregnant mice) and following maternal exposure to ethanol (pregnant mice and rats).[21]

Acetaldehyde is metabolized to acetic acid. The rate of metabolism to acetic acid varies, but is generally considered to be rapid. Saturation kinetics are not apparent even following exposure to large doses. Acetic acid enters into intermediary metabolism and is used in the production of carbon dioxide and water or in cellular synthesis of cholesterol, fatty acids, and other tissue components.[22] The liver is the primary metabolic site for acetaldehyde, although some metabolism of acetaldehyde occurs in human renal tubules.[23]

Following uptake, virtually no acetaldehyde is excreted in the urine.[24] Acetaldehyde has been detected in expired air (usually no more than 5% of that inhaled) but only its metabolites have been detected in urine.[25]

Qualitative Description of Health Effects

Acute dermal exposure to acetaldehyde may cause a rash or burning feeling.[26] Humans exposed acutely to moderate concentrations of acetaldehyde through inhalation experience irritation and inflammation of the eyes, nose, throat, and respiratory tract. Acute irritation from exposure to acetaldehyde is characterized by the following: eye irritation in sensitive individuals at 25 parts per million (ppm) for 15 minutes; eye irritation at 50 ppm for 15 minutes; irritation of the respiratory tract at 134 ppm for 30 minutes (2.15 milligrams per kilogram [mg/kg] over 30 minutes); irritation of the nose and throat at 200 ppm for 15 minutes.[27]

Chronic dermal exposure to acetaldehyde can cause skin burns and a skin allergy. Chronic inhalation exposure at high concentrations causes adverse respiratory tract effects in animals.[28] Carcinogenicity studies in rats have shown that acetaldehyde causes respiratory tract tumors.[29] No carcinogenicity studies are available on orally administered acetaldehyde.

Quantitative Description of Health Effects

An inhalation reference concentration (RfC) of 0.009 milligrams per cubic meter (mg/m^3) has been established by EPA[30] based on the studies of Appleman, et al.[31, 32] In these studies, rats were exposed to concentrations up to 5,000 ppm, 6 hours/day for four weeks. Extensive pathologic examinations were

1994.

21 World Health Organization (WHO). Acetaldehyde. Report No. 167. 1995.

22 U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

23 World Health Organization (WHO). Acetaldehyde. Report No. 167. 1995.

24 World Health Organization (WHO). Acetaldehyde. Report No. 167. 1995.

25 U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

26 U.S. Environmental Protection Agency (EPA). Integrated Risk Information System (IRIS). Acetaldehyde. April. 1998.

27 U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

28 U.S. Environmental Protection Agency. Chemicals in the Environment: Acetaldehyde (CAS No. 75-07-0). Office of Pollution Prevention and Toxics Chemical Fact Sheet. Prepared by the Office of Pollution Prevention and Toxics. EPA 749-F-94-003. 1994.

29 U.S. Environmental Protection Agency (EPA). Integrated Risk Information System (IRIS). Acetaldehyde. April. 1998.

30 U.S. Environmental Protection Agency (EPA). Integrated Risk Information System (IRIS). Acetaldehyde. April. 1998.

31 Appleman, L.M., R.A. Woutersen, V.J. Feron, R.N. Hooftman, and W.R.F. Notten. Effect of Variable Versus Fixed Exposure Levels on the Toxicity of Acetaldehyde in Rats. J. Appl. Toxicol. 6(5):331-336. 1986.

32 Appleman, L.M., R.A. Woutersen, and V.J. Feron. Inhalation Toxicity of Acetaldehyde in Rats. I. Acute and subacute studies. Toxicology. 23:293-297. 1982.

performed on tissues of the respiratory tract. In these tissues, cell density and viability were significantly decreased in the group exposed to 500 ppm. Degeneration of the cells lining nasal passages was also observed at this exposure level. The lower dose of 150 ppm (49 mg/m^3) was identified as the no observed adverse effect level (NOAEL). The NOAEL health effects concentration for a gas respiratory effect in the extrathoracic region (8.7 mg/m^3) was calculated from this dose. An uncertainty factor of 1,000 was applied: 10 to account for sensitive human populations; 10 for both uncertainty in the interspecies extrapolation using dosimetric adjustments and to account for the incompleteness of the database; and 10 to account for extrapolation from subchronic to chronic.[33] Confidence in the study is medium, confidence in the database and the RfC is low.

EPA has classified acetaldehyde as a probable human carcinogen (B2) based on increased incidence of nasal tumors in male and female rates and laryngeal tumors in male and female hamsters exposed via inhalation. The inhalation slope factor is based on studies in rats that showed that the compound causes respiratory tumors.[34, 35] In the primary study, rats were exposed to 0, 750, 1,500, or 3,000 ppm acetaldehyde 6 hours/day for 27 months. Interim sacrifices were performed and the first tumor was observed at 52 weeks.

Results of the study indicated a dose-related increase in the incidence of cancer in both male and female rats. In addition, there were exposure-related increases in the incidences of multiple respiratory tract tumors. Based on this study, an inhalation slope factor of 2.2×10^{-6} per $\mu g/m^3$ was developed. In a related study, exposure was terminated at 52 weeks. Findings from this study indicate that after exposure for this duration, lesions produced by unnatural cell growth of the nasal cell lining may develop into tumors even without continued exposure.[36] No studies of carcinogenicity via the oral route are available.

The California Environmental Protection Agency (Cal EPA) has developed its own set of cancer potency factors for use in risk assessments required by regulatory programs in California. Cal EPA has developed an inhalation cancer potency factor of $0.01\ (mg/kg\text{-}day)^{-1}$ for acetaldehyde based on data provided in Feron et al.[37] and Woutersen et al.[38]

Summary of Criteria

Criterion	Value	Source
EPA Carcinogen Classification	B2	EPA 1998
Cancer Slope Factor/Unit Risk Inhalation	$2.2 \times 10^{-6}\ (:/m^3)^{-1}$	EPA 1998
Cal EPA Cancer Potency Factor	$0.01\ (mg/kg\text{-}day)^{-1}$	Cal EPA 1995
Inhalation Reference Concentration	$9 \times 10^{-3}\ mg/m^3$	EPA 1998
Cal Permissible Exposure Limits, PEL	$180\ mg/m^3$	CCR, Title 8, 2000[1]
Cal Permissible Exposure Limits, STEL	$270\ mg/m^3$	CCR, Title 8, 2000[1]

[1] California Code of Regulations, Title 8, Section 5155, February 16, 2000.

[33] U.S. Environmental Protection Agency (EPA). Integrated Risk Information System (IRIS). Acetaldehyde. April. 1998.

[34] Woutersen, R.A. and L.M. Appleman. Lifespan Inhalation Carcinogenicity Study of Acetaldehyde in Rats. III. Recovery after 52 Weeks of Exposure. Report No. V84.288/190172. The Netherlands: CIVO-Institutes TNO. 1984.

[35] Woutersen, R., A. Van Garderen-Hoetmer and L.M. Appleman. Lifespan (27 months) Inhalation Carcinogenicity Study of Acetaldehyde in Rats. Report No. V85.145/190172. The Netherlands: CIVO-Institutes TNO. 1985.

[36] Woutersen, R., A. Van Garderen-Hoetmer and L.M. Appleman. Lifespan (27 months) Inhalation Carcinogenicity Study of Acetaldehyde in Rats. Report No. V85.145/190172. The Netherlands: CIVO-Institutes TNO. 1985.

[37] Feron, V.J., A. Kruysse, and R.A. Woutersen. Respiratory Tract Tumors in Hamsters Exposed to Acetaldehyde Vapor Alone or Simultaneously to Benzo(a)Pyrene or Diethylnitrosamine. Eur. J. Cancer Clin. Oncol. 18:13-31. 1982.

[38] Woutersen, R.A., L.M. Appleman, A. Van Garderen-Hoetmer, and V.J. Feron. Inhalation Toxicology of Acetaldehyde in Rats. III. Carcinogenicity Study. Toxicology. 41:213-232. 1986.

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ACROLEIN[1]

Introduction

Acrolein is a clear or yellow liquid with a disagreeable, sharp odor. It burns easily and is easily volatilized. Acrolein is used as a chemical intermediate in the production of acrylic acid; acrolein is also used as a biocide in liquid petrochemical fuels and oil wells; as a herbicide and algaecide in irrigation waters and drainage ditches; as a slimicide in the paper industry; in the control of algae, weeds, and mollusks in recirculating process water systems; and is found in some livestock feeds and pesticides. Small amounts of acrolein can be formed and can enter the air when organic matter such as trees and other plants, including tobacco, are burned and also when fuels such as gasoline and oil are burned.

Potential for Human Exposure

Releases to the Environment

Acrolein may be released to the environment in emissions and effluents from its manufacturing and use facilities, in emissions from combustion processes such as combustion of petrochemical fuels, as a photooxidation product of various hydrocarbon pollutants found in air (including propylene and 1,3-butadiene), from direct application to water and wastewater as a slimicide and herbicide, and from land disposal of some organic waste materials.

Environmental Fate

Acrolein is an unstable compound and is removed from air primarily by reaction with photochemically generated hydroxyl radicals; it has a half-life of 15 - 20 hours in air. Reaction products include carbon monoxide, formaldehyde, and glycolaldehyde. Small amounts of acrolein may be removed from the atmosphere in precipitation. Acrolein has a half-life of 1 - 3 days in surface water and may be removed by volatilization, aerobic biodegradation, or reversible hydration to B-hydroxypropionaldehyde, which subsequently biodegrades. Acrolein in soil is subject to the same removal processes as in water. Acrolein is highly mobile in soil; however, volatilization and degradation processes reduce movement through soil.

Environmental Levels

Air: No current information is available describing acrolein concentrations in ambient air. Acrolein concentrations in air samples collected in Los Angeles, California during 1960-1961 averaged between 5 and 8 parts per billion (ppb). Air samples collected in the Los Angeles basin over a 12-week period during 1968 contained acrolein concentrations ranging from non-detect to 18 ppb; most values ranged between 0.9 and 9 ppb. These data are too old to provide insight regarding acrolein concentrations in urban air.

Water: Acrolein rarely occurs in wastewater streams, surface water, and groundwater in the United States. Acrolein has not been found as a drinking water contaminant. Acrolein, in combination with acetone, was detected in rainwater collected in Los Angeles, California, at a concentration of 0.05 parts per trillion. These compounds were not detected in rainwater samples from less densely populated areas of California.

Soil and Sediment: Acrolein was detected in soil at 1 of 357 hazardous waste sites in the United States at a mean concentration of 6.5 ppb. Concentrations in soil from non-hazardous waste sites was not located; due to its volatile and mobile nature, it is unlikely that acrolein is present in soil in significant concentrations.

Other Environmental Media: Acrolein is a gaseous constituent of tobacco smoke; the level of acrolein in sidestream smoke is 12 times higher than in mainstream smoke. Smoke from various types of cigarettes has been found to contain acrolein at concentrations ranging from 3 to 220 micrograms (Fg) per cigarette. Trace concentrations of acrolein have been detected in alcohol; acrolein has also been detected in food, however, data were not sufficient to determine concentrations typically encountered.

[1] Information pertaining to Acrolien is derived from Agency for Toxic Substances and Disease Registry. Toxicological Profile of Acrolein, December 1990, as well as other sources, as noted.

Toxicokinetics

Acrolein can be absorbed through the respiratory tract, and to a lesser extent through oral, and dermal routes. No studies were located which indicate the amount of absorption in humans through oral, dermal or respiratory routes. Dermal absorption appears to be influenced by the carriers present. Only limited information is available on human metabolism of acrolein. In rat liver and lung preparations free acrolein was shown to interact with proteins and nucleic acids and thiol groups such as glutathione. Acrolein also could be transformed into acrylic acid by liver cytosol or microsomes, or it can be oxidized to glycidaldehyde by lung or liver microsomes. In rats, a single dose was administered and urine collected for three days. S-carboxyethylmercapturic acid was detected, but S-hydroxypropyplmercapturic acid (which should have been formed if acrolein had reacted with glutathione) was not. Therefore, Draminski et al. proposed an alternative metabolic pathway in which acrolein is first metabolized to acrylic acid, with subsequent formation of S-carbxoyethylmercapturic acid methyl ester.[2] No information was found relating to the excretion rate or biological half life of acrolein.

Qualitative Description of Health Effects

The only known effects of acrolein exposure in humans are general respiratory congestion and eye nose and throat irritation. Studies in humans have shown that eye irritation occurs with concentrations slightly lower than those that produced either nose or throat irritation.

The clinical signs common to humans and animals following acute inhalation exposure to acrolein (e.g., upper respiratory tract irritation and congestion, airway occlusion, and death by asphyxiation) point to the respiratory system as the major target of toxicity. Even if death is prevented, some respiratory effects may persist for months. No other systems or organs have yet been identified as targets for acrolein, although nonspecific effects have been identified in the liver, kidney, and brain of animals.

Quantitative Description of Health Effects

Information regarding the toxicity of acrolein to humans is scarce. Acrolein acts primarily as an irritant to the eyes and respiratory tract. An oral reference dose (RfD) has not been approved by EPA work groups; however, an oral RfD for acrolein of 2×10^{-2} milligrams per kilogram per day (mg/kg-day) is presented in EPA's Health Effects Assessment Summary Tables (HEAST).[3] EPA has established an inhalation reference concentration (RfC) of 2×10^{-5} milligrams per cubic meter (mg/m^3), based on information presented in Kutzman[4] and Feron et al.[5]

Kutzman[6] exposed male and female F344 rats to concentrations of 0, 0.4, 1.4 and 4.0 parts per million (ppm) (0, 0.917, 3.21 and 11.23 mg/m^3, respectively) 6 hours/day, 5 days/week for 62 days. Duration-adjusted concentrations are 0, 0.164, 0.573 and 2.0 mg/m^3, respectively. Results of this study support a lowest-observed-adverse-effects-level (LOAEL) for respiratory effects in the nasal cavity at 0.4 ppm. The LOAEL (Human Equivalent Concentration [HEC]) for F344 male rats is 0.02 mg/m^3; the critical effect observed was squamous metaplasia and neutrophilic infiltration of nasal epithelium.

Feron et al. [7] exposed Syrian Golden hamsters (10/sex/concentration), Wistar rats (6/sex/concentration) and Dutch rabbits (2/sex/concentration) at 0, 0.4, 1.4, and 4.9 ppm (0, 0.917, 3.21, and 11.23 mg/m^3, respectively) to acrolein 6 hours/day, 5 days/week for 13 weeks. Duration-adjusted concentrations are 0, 0.164, 0.573, and 2.0 mg/m^3, respectively. Rats appeared to be the most sensitive of the three species tested. Results of this study support a LOAEL for effects in the nasal cavity of 0.4 ppm based on the

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2 Draminski, W., E. Eder, and Henschler. A New Pathway of Acrolein Metabolism in Rats. Arch. Toxicol. 52:243-247. 1983.

3 U.S. Environmental Protection Agency. 1997 Health Effects Assessment Summary Tables. Office of Research and Development. Office of Solid Waste and Emergency Response. Washington DC. July.

4 Kutzman, R.S. A subchronic inhalation study of Fischer 344 rats exposed to 0, 0.4, 1.4, or 4.0 ppm acrolein. Brookhaven National Laboratory, Upton, NY. National Toxicology Program: Interagency Agreement No. 222-Y01-ES-9-0043. 1981.

5 Feron, V.J., A. Kruysse, H.P. Til and H.R. Immel. Repeated exposure to acrolein vapour: Subacute studies in hamsters, rats and rabbits. Toxicology. 9: 47-57. 1978.

6 Kutzman, R.S. A subchronic inhalation study of Fischer 344 rats exposed to 0, 0.4, 1.4, or 4.0 ppm acrolein. Brookhaven National Laboratory, Upton, NY. National Toxicology Program: Interagency Agreement No. 222-Y01-ES-9-0043. 1981.

7 Feron, V.J., A. Kruysse, H.P. Til and H.R. Immel. Repeated exposure to acrolein vapour: Subacute studies in hamsters, rats and rabbits. Toxicology. 9: 47-57. 1978.

slightly affected metaplastic and inflammatory changes. The LOAEL (HEC), using the ventilation rate for male Wistar rats, is 0.03 mg/m^3. The LOAEL (HEC) for F344 male rats is used as the operational basis for the RfC since it is more conservative than the HEC based on Wistar rats.

To derive the RfC, the LOAEL (HEC) was divided by an uncertainty factor of 1,000; 10 to account for sensitive human populations, 10 for both interspecies extrapolation, due to use of the dosimetric adjustment based on respiratory surface areas, and to account for the mild nature of the LOAEL, and 10 to account for the lack of chronic studies. This is also sufficient for the lack of reproductive and developmental studies in the database.

Several additional animal studies (Kane et al.,[8] Buckley et al.,[9] Astry and Jakab[10] Leach et al.,[11] Feron and Kruysse,[12] Lyon et al.,[13] Bouley et al.,[14] and Lam et al.,[15]) are available describing adverse impacts associated with acute and subchronic inhalation exposure to acrolein; generally, the results confirm that acrolein is a highly selective respiratory toxicant.

The principal studies used to develop the RfC were given high confidence because adequate numbers of animals were used, careful attention was paid to experimental protocol and together they demonstrated a consistent profile of histopathological changes in the respiratory system. The data base was given low to medium confidence due to the lack of chronic data and adequately conducted reproductive or developmental studies. Medium confidence in the derived RfC follows.

EPA has assigned a carcinogen classification of C, possible human carcinogen to acrolein. The basis for classification is increased incidence of adrenal cortical adenomas to female rats and carcinogenic potential of an acrolein metabolite. Acrolein is mutagenic in bacteria and is structurally related to probable or known human carcinogens. Oral and inhalation cancer slope factors are not available from EPA for acrolein.[16]

The California Environmental Protection Agency (Cal EPA) has not developed cancer potency factors for acrolein.[17] EPA has not developed a maximum contaminant level (MCL) or maximum contaminant level goal (MCLG) for acrolein in drinking water.[18]

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8 Kane, L.E., C.S. Barrow and Y. Alarie. A short-term test to predict acceptable levels of exposure to airborne sensory irritants. J. Am. Hygiene Assoc. 40: 207-229. 1979.

9 Buckley, L.A., X.Z. Jiang, R.A. James, K.T. Morgan and C.S. Barrow. Respiratory tract lesions induced by sensory irritants at the RD50 concentration. Toxicol. Appl. Pharmacol. 74: 417-429. 1984.

10 Astry, C.L. and G.J. Jakab. The effects of acrolein exposure on pulmonary antibacterial defenses. Toxicol. Appl. Pharmacol. 67: 49-54. 1983.

11 Leach, C.L., N.S. Hatoum, H.V. Ratajczak and J.M. Gerhart. The pathologic and immunologic effects of inhaled acrolein in rats. Toxicol. Lett. 39: 189-198. 1987.

12 Feron, V.J. and A. Kruysse. Effects of exposure to acrolein vapor in hamsters simultaneously treated with benzo(a)pyrene or diethylnitrosamine. J. Toxicol. Environ. Health. 3: 379-394. 1977.

13 Lyon, J.P., L.J. Jenkins, Jr., R.A. Jones, R.A. Coon and J. Siegel. Repeated and continuous exposure of laboratory animals to acrolein. Toxicol. Appl. Pharmacol. 17: 726-732. 1970.

14 Bouley, G., A. Dubreuil, J. Godin, M. Boisset and C. Boudene. Phenomena of adaptation in rats continuously exposed to low concentrations of acrolein. Ann. Occup. Hyg. 19: 27-32. 1976.

15 Lam, C-W. M. Casanova and H. d'A. Heck. Depletion of nasal mucosal glutathione by acrolein and enhancement of formaldehyde-induced DNA-protein cross-linking by simultaneous exposure to acrolein. Arch. Toxicol. 58:67-71. 1985.

16 U.S. Environmental Protection Agency. Integrated Risk Information System. Acrolein. 1998.

17 Cal EPA (California Environmental Protection Agency). (April 10, 1995, April 1, 1996) California Cancer Potency Factors Update. Standards and Criteria Workgroup, Cal EPA. November. 1994

18 U.S. Environmental Protection Agency. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-R-96-001. February. 1996.

Summary of Acrolein Criteria

Criteria	Value	Source
EPA carcinogen classification	C - possible human carcinogen	IRIS 2000
RfC (EPA)	2×10^{-5} mg/m^3	IRIS 2000
Oral Chronic RfD (EPA)	2×10^{-2} mg/kg/day	HEAST 1997
Cal Permissible Exposure Limits, PEL	0.25 mg/m^3	CCR, Title 8, 2000*
Cal Permissible Exposure Limits, STEL	0.8 mg/m^3	CCR, Title 8, 2000*

* California Code of Regulations, Title 8, Section 5155, February 16, 2000.

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ARSENIC[1]

Introduction

Arsenic (As) is a naturally occurring metalloid, which can be present in a number of different valence states and as a constituent in both inorganic and organic compounds. Elemental arsenic is used as an alloying agent for heavy metals and in special solders. Arsenic trioxide is the arsenic compound of chief commercial importance. The principal use of arsenic, as arsenic trioxide, is in products used for wood preservation. Organic and inorganic arsenic is also used in insecticides, herbicides, algicides, and growth stimulants for plants and animals. Gallium arsenide (GaAs) has widespread use in the microelectronics industry. Some organic arsenic compounds are used medicinally in the treatment of syphilis, yaws, amoebic dysentery, and trypanosomiasis.

Potential for Human Exposure

Releases to the Environment

Arsenic occurs naturally in a variety of sulfidic ores and can be released to the environment from natural sources, such as volcanoes and erosion from mineral deposits. Human activities, such as metal smelting, chemical production and use, coal combustion, and waste disposal, release considerable amounts of arsenic to the environment. Most human releases are to land, but substantial amounts are also released to air and water.

About 17 million pounds per year of arsenic may be released to the air from natural sources, such as volcanic eruptions and forest fires. Globally, this is probably greater than the amount released to air by human activities; however, industrial activities are the primary localized sources of arsenic released to air. In the late 1970's, arsenic releases from industrial sources ranged from 13 to 19 million pounds per year; regulations on industrial emissions have likely resulted in decreases in air releases from these sources. The Toxics Release Inventory (TRI) database reported industrial releases to air of 270,000 pounds for 1988. Not all industries are required to report to TRI, therefore, these releases should not be regarded as total arsenic releases to air.

Arsenic may be released to water by natural weathering processes, discharge from industry, leaching from soil or landfills, and urban runoff. TRI reports that industrial arsenic discharges to surface water and public sewage treatment works for 1988 were 7,500 and 5,100 pounds, respectively. Underground injection, which can lead to groundwater contamination, totaled 27,400 pounds in 1988.

Most arsenic released to the environment from human activities is released to soil. Major sources include application of pesticides, disposal of solid wastes from fossil fuel combustion and industrial processes, and land application of sewage sludge. TRI reports about 5.6 million pounds of arsenic was released to land in 1988, accounting for nearly 95% of reported environmental releases.

Environmental Fate

Arsenic is released to air as particulate matter, primarily as arsenic trioxide; some of this arsenic undergoes oxidation to the pentavalent state, resulting in a mixture of trivalent and pentavalent forms of arsenic in air. Arsenic particles in air are deposited to soil or surface water through wet or dry deposition. Typical residence time of particulate-bound arsenic is about 9 days.

Arsenic released to surface water or deposited to surface water from air can undergo transformations, including oxidation-reduction reactions, ligand exchange, and biotransformation. Factors influencing fate processes include oxidation-reduction potential (Eh), pH, metal sulfide and sulfide ion concentrations, iron concentrations, water temperature, salinity, and distribution and composition of biota. Arsenic exists in surface water primarily as arsenate, but microorganisms may reduce arsenate to arsenite and methylated arsenicals. Arsenate generally predominates in groundwater, although arsenite may be an important component.

Arsenic in soil undergoes transformations similar to those occurring in aquatic systems. Arsenic (V) predominates in aerobic soils, arsenic (III) in slightly reduced soils (i.e., temporarily flooded), and arsine,

[1] Information pertaining to Arsenic is derived from Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. April, 1993, as well as other sources as noted.

methylated arsenic, and elemental arsenic in very reduced conditions, such as swamps and bogs. Organoarsenical pesticides (e.g., monomethyl arsonic acid [MMA] and dimethyl arsinic acid [DMA]) applied to soil are metabolized by soil bacteria to alkylarsines, arsenate, and MMA.

Environmental Levels

AIR: Arsenic in air is usually a mixture of arsenite and arsenate; organic species of arsenic may be present in areas of methylated arsenic pesticide application. Arsenic concentrations in air are generally greater in urban areas than in rural areas. Mean concentrations in air in the United States range from <1 to 3 nanograms per cubic meter (ng/m^3) in remote areas and from 20 to 30 ng/m^3 in urban areas. Large cities generally have greater arsenic air concentrations than smaller cities due to emissions from coal-fired power plants, but maximum 24-hour concentrations are generally less than 100 ng/m^3.

Water: Arsenic is found in surface water, groundwater, and drinking water throughout the United States. Arsenic concentrations in lakes and rivers are typically less than 10 parts per billion (ppb). Groundwater arsenic concentrations average between 1 and 2 ppb, except in some western states with mineral deposits high in arsenic; groundwater arsenic concentrations up to 3,400 ppb have been observed in these areas. More than 99% of public water supplies have arsenic concentrations less than the EPA maximum contaminant level (MCL).

Soil and Sediment: Background arsenic concentrations in soil range from 1 to 40 parts per million (ppm). Soil overlying arsenic-rich deposits may have arsenic concentrations two orders of magnitude higher. Industrial wastes and pesticide application may increase arsenic concentrations in soil.

Sediments in aquatic systems act as a sink for arsenic; sediment arsenic concentrations reported for rivers, lakes, and streams in the United States range from 0.1 to 4,000 ppm. Much higher levels may occur in areas of contamination.

Other Environmental Media: Arsenic is found in food, with the highest levels detected in seafood, meats, and grains. Arsenic concentrations in grains and cereals average about 0.02 ppm; meat, fish, and poultry contain average arsenic concentrations of 0.14 ppm. Shellfish and other marine foods typically contain the greatest arsenic concentrations; mean levels in seafood are usually 4 to 5 ppm, but may be as high as 170 ppm. Much of the arsenic in fish and shellfish is in an organic form that is essentially nontoxic.

Arsenic is found in tobacco and cigarettes. Cigarettes contain an average of 1.5 micrograms of arsenic.

Toxicokinetics

Absorption of arsenic from the gastrointestinal tract is dependent on the solubility of the arsenic compound. Soluble forms of both As(III) and As(V) are completely absorbed in laboratory animals[2] and humans.[3] Insoluble forms may not be available for absorption in humans as indicated by the lack of increase in urinary excretion of arsenic in human volunteers administered arsenic selenide orally.[4]

Following inhalation, arsenic absorption is dependent on particle size, with larger particles being quickly cleared from the lungs with little absorption. In one study, Holland et al.[5, 6] examined the absorption and deposition of arsenic in lung cancer patients exposed to arsenic in arsenite-containing cigarette smoke and arsenic-containing aerosols. In the patients, approximately 40% of arsenic particulates were deposited in the lungs and approximately 75-85% of the deposited arsenic was absorbed by the lungs. Smaller particles penetrate into alveolar spaces and may remain there for extended periods, increasing

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2 Vahter, M. Biotransformation of Trivalent and Pentavalent Inorganic Arsenic in Mice and Rats. Environ. Res. 25:286-293. 1981.

3 U.S. Environmental Protection Agency. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, DC. EPA 600/8083-021F. March, 1984.

4 Mappes, R. (Experiments on Excretion of Arsenic in Urine.) Versuche zur Ausscheidung von Arsen in Urin. Int. Arch. Occup. Environ. Health. 40:267-272. 1977.

5 Holland, R. H., M. S. McCall, H. C. Lanz. A study of inhaled arsenic-74 in man. Cancer Res. 19:1154-1156. 1959.

6 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. April, 1993.

the chances for inhaled arsenic to be absorbed.[7] Absorption from the lung may be rapid for soluble arsenic forms, but is much slower for more insoluble forms.

No studies are available regarding the absorption of arsenic in humans following dermal exposure. Animal studies indicate that arsenic may bind to the skin following dermal exposure, and be slowly absorbed even after exposure ends. In one study in which the tails of rats were immersed in sodium arsenate for 1 hour, arsenic uptake was not detected for up to 24 hours after exposure; however, over the next five days arsenic concentrations rose in the blood, liver and spleen. The rate of uptake was estimated to be 1 to 33 micrograms per squared centimeter per hour ($\mu g/cm^2/hr$).

Following absorption, arsenic is distributed throughout the body. Analysis of autopsy tissues collected from humans exposed to background levels of arsenic in food show that arsenic was present in all tissues of the body. Similarly, elevated levels of arsenic were noted in all tissues of mice and hamsters given oral doses of arsenate or arsenite. There is little tendency for arsenic to accumulate preferentially in any internal organs. Arsenic can cross the placental barrier as evidenced by elevated levels of arsenic in the placenta and fetus of pregnant females.

Metabolism of inorganic arsenic takes place via two major processes: (1) oxidation/reduction reactions that interconvert arsenate and arsenite, and (2) methylation reactions that convert arsenite to MMA and DMA. These processes appear to be used for metabolism regardless of the route of exposure.

Arsenic is efficiently metabolized to methylated forms (MMA and DMA) in the liver in both animals and humans.[8] Because acute toxicity of these methylated forms is much less than for inorganic arsenic, methylation is considered detoxification. At high arsenic doses, methylation pathways may become saturated.[9, 10] This may result in a "threshold" determined by the ability to metabolize arsenic, where low doses are relatively nontoxic due to conversion to methylated forms, and higher doses are more toxic since greater amounts of inorganic arsenic will be available for distribution to target tissues. This is especially important for carcinogenesis following oral exposure, where small daily intakes could be much less effective in inducing cancer than higher doses that saturate metabolism. Unfortunately, available information is insufficient to determine the saturation point in humans.

Arsenic is primarily excreted in the urine in both animals and humans in the form of metabolic products, including As (+3), As(+5), DMA, and MMA.[11] This is true for both inorganic and methylated forms. Biliary excretion has been noted to be highly variable in animals, but due to reabsorption in the intestines, does not contribute significantly to overall excretion.[12] Vahter et al.[13] reported that urinary arsenic levels in smelter workers rose within hours of starting work on a Monday and then fell over the weekend. This indicates that excretion is rapid, an observation supported by experimental studies in animals.[14, 15] Human oral exposure to known amounts of arsenite or arsenate indicate that very little is excreted in the feces,[16, 17] while 45-85% is excreted in the urine between 1-3 days.[18, 19, 20, 21] Small amounts of arsenic may remain bound to tissues, depending inversely on the rate and extent of methylation.

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7 U.S. Environmental Protection Agency. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, DC. EPA 600/8083-021F. March, 1984.

8 Buchet, J.P., R. Lauwerys, and H. Roels. Comparison of the Urinary Excretion of Arsenic Metabolites after a Single Oral Dose of Sodium Arsenite, Monomethyl Arsonate, or Dimethyl Arsinate in Man. Int. Arch. Occup. Environ. Health. 48:71-79. 1981.

9 Lovell, M.A. and J.G. Farmer. Arsenic Speciation in Urine from Humans Intoxicated by Inorganic Arsenic Compounds. Hum. Toxicol. 4:203-214. 1985.

10 Buchet, J.P., R. Lauwerys, and H. Roels. Comparison of the Urinary Excretion of Arsenic Metabolites After a Single Oral Dose of Sodium Arsenite, Monomethy, Arsonate, or Dimethyl Arsinate in Man. Int. Arch. Occup. Environ. Health. 48:71-79. 1981.

11 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. April, 1993.

12 Klassen, C. Biliary Excretion of Arsenic in Rats, Rabbits, and Dogs. Toxicol. Appl. Pharmacol. 29:447-457. 1974.

13 Vahter, M., L. Friberg, B. Rahnster, et al. Airborne arsenic and urinary excretion of metabolites of inorganic arsenic among smelter workers. Int. Arch. Occup. Environ. Health. 57:79-91. 1986.

14 Rhoads, K., C. L. Sanders. Lung clearance, translocation, and acute toxicity of arsenic, beryllium, cadmium, cobalt, lead, selenium, vanadium, and ytterbium oxides following deposition in rat lung. Environ. Res. 36:359-378. 1985.

15 Marafante, E., M. Vahter. Solubility, retention, and metabolism of intratracheally and orally administered inorganic arsenic compounds in the hamster. Environ. Res. 42:72-82. 1987.

16 Bettley, F. R., J. A. O'Shea. The absorption of arsenic and its relation to carcinoma. Br. J. Dermatol. 92:563-568. 1975.

17 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. April, 1993.

Qualitative Description of Health Effects

Toxicological information on arsenic has been reviewed by EPA in its ambient water quality criteria document[22] and health assessment document[23] and more recently by EPA's Risk Assessment Forum[24] and ATSDR. Acute exposure to ingested arsenic may result in death. Although the information on lethal doses to humans is sparse, Armstrong et al.[25] reported that two people in a family of eight died after ingesting 110 ppm of arsenic in water. Acute poisoning of humans with arsenic may result in gastrointestinal effects, hemolysis, and neuropathy. Skin contact may result in burning, itching, and a rash. Eye contact can cause red, watery eyes and irritation.[26]

Chronic exposure is associated with characteristic toxic effects on the peripheral nervous system, such as "pins and needles", burning, numbness, and weakness of arms and legs. In children, arsenic may have toxic effects on the central nervous system. In humans, vascular damage, keratosis, hyperpigmentation, precancerous dermal lesions, and cardiovascular injury frequently follow chronic exposure to arsenic. An example of vascular damage is "blackfoot disease," a disease characterized by loss of circulation in hands and feet, which leads to necrosis and gangrene. The disease was endemic in an area of Taiwan where the population was exposed to arsenic ranging from 0.17 to 0.8 ppm in well water. Chronic exposure can cause an ulcer or hole in the septum dividing the inner nose.[27] Arsenic has been found to be embryotoxic, fetotoxic, and teratogenic in several animal species at high doses. One report suggests that children of women working in a Swedish copper smelter had lower birth weights than expected.[28] Though arsenic exposure was involved, women were also exposed to a variety of heavy metals and sulfur dioxide. Thus, it is not possible to link fetal effects with arsenic exposure.

Arsenic induces chromosome aberrations and impairs DNA repair but has not been shown to cause point mutations. There is convincing evidence from a large number of studies that ingestion of arsenic increases the risk of skin cancer. EPA[29] has classified arsenic via oral exposure in Group A - Human Carcinogen. Squamous cell carcinomas are the most common types of skin cancer and appear to develop from hyperkeratinized corns. Basal cell carcinomas also occur. In a key study by Tseng et al.,[30] ingestion of contaminated drinking water from wells in Taiwan was correlated with an increased skin cancer rate. Based on an examination of over 40,000 people in Taiwan, the skin cancer rate was 10.6/1,000. There is also mounting evidence that ingestion of arsenic may increase the risks of internal cancers. These include tumors of the bladder, kidney, liver, and lung.[31]

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[18] Buchet, J.P., R. Lauwerys, and H. Roels. Comparison of the Urinary Excretion of Arsenic Metabolites after a Single Oral Dose of Sodium Arsenite, Monomethyl Arsonate, or Dimethyl Arsinate in Man. Int. Arch. Occup. Environ. Health. 48:71-79. 1981.

[19] Crecelius, E. A. Changes in the chemical speciation of arsenic following ingestion by man. Environ. Health Perspect. 19:147-150. 1977.

[20] Mappes, R. (Experiments on Excretion of Arsenic in Urine.) Versuche zur Ausscheidung von Arsen in Urin. Int. Arch. Occup. Environ. Health. 40:267-272. 1977.

[21] Tam, G. K., S. M. Charbonneau, G. Lacroix, et al. Confirmation of inorganic arsenic and dimethylarsenic acid in urine and plasma of dog by ion-exchange and TLC. Bull. Environ. Contam. Toxicol. 21:371-374. 1979.

[22] U.S. Environmental Protection Agency. Ambient Water Quality Criteria for Arsenic. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-80-021. October, 1980.

[23] U.S. Environmental Protection Agency. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, DC. EPA 600/8083-021F. March, 1984.

[24] U.S. Environmental Protection Agency. Ambient Water Quality Criteria for Arsenic-1984. Office of Water Regulations and Standards. PB85-227445. Washington, DC. EPA 440/5-84-003. January, 1985.

[25] Armstrong, C. W., R. B. Stroube, T. Rubio, et al. Outbreak of fatal arsenic poisoning caused by contaminated drinking water. Arch. Environ. Health. 39:276-279. 1986.

[26] U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Arsenic. 1986.

[27] U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Arsenic. 1986.

[28] Nordström, S., L. Beckman, and I. Nordenson. Occupational and Environmental Risks in and around a Smelter in Southern Sweden. I. Variations in Birth Weight. Hereditas. 88:43-46. 1978.

[29] U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

[30] Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin, and S. Yeh. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463. 1968.

[31] U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

Epidemiological studies have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma, while ingestion has been linked to a form of skin cancer and more recently to bladder, liver, and lung cancer.[32, 33] Although arsenic's potential as a human carcinogen has long been recognized, reliable induction of cancer in animal models has not yet been achieved. Arsenic exposure has been reported to increase the neurotoxic effects of lead in children as measured by aggressive behavior.[34] Arsenic and aluminum may interact in similar fashion, promoting aggressive behavior. Arsenic and cigarette smoke are reported to have multiplicative effects on lung cancer mortality in smelter workers.[35] Arsenic and cadmium together had a greater effect on reduced weight gain in rats than expected from the simple sum of their individual effects.[36]

Chronic inhalation of arsenic compounds may lead to an increased risk of mortality from cardiovascular disease, but this effect has not been observed in all studies. An increased incidence of Raynaud's disease (cyanosis of the digits due to arterial and arteriolar contraction) and increased constriction of blood vessels in response to cold, suggests that long-term inhalation exposure to arsenic compounds (0.05-0.5 mg As/m^3) may injure blood vessels and/or the heart.

Quantitative Description of Health Effects

EPA[37] has classified arsenic as a Group A - Human Carcinogen. This category applies to chemical agents for which there is sufficient evidence of carcinogenicity in humans.

Oral Toxicity

Tseng observed a population in Taiwan where well water contaminated with arsenic was used for 60 years. The study found significantly elevated standard mortality ratios for cancer of the bladder, lung, liver, kidney, skin, and colon. The study was extensive, but did not define a control population. Concentrations of arsenic in the water ranged from 0.01 to 1.82 mg/L. The overall prevalence rate for skin cancer was 10.6 per 1,000 and for peripheral vascular disorder of the extremities was 8.9 per 1,000. Three dose groups were designated as "low" (below 0.3 mg/L), "mid" (0.3-0.6 mg/L), and "high" (above 0.6 mg/L). Tseng[38] reported a dose-response relationship between concentrations of arsenic in the water and skin cancer. Based on this study, the oral CSF is 1.50 $((mg/kg)/day)^{-1}$.[39] EPA assumed 100% absorption of arsenic following oral exposure from water.[40]

EPA has developed an oral reference dose (RfD) for arsenic of 3×10^{-4}.[41] The RfD is based on data for chronic oral exposure to arsenic in humans.[42, 43] The data reported in Tseng[44] show an increased incidence of blackfoot disease for humans exposed to arsenic in well water. The incidences of blackfoot

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32 Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin, and S. Yeh. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463. 1968.

33 Chen, C., Y. Chuang, S. You, T. Lin, and H. Wu. A Retrospective Study on Malignant Neoplasms of Bladder, Lung and Liver in Blackfoot Disease Endemic Area in Taiwan. Brit. J. Cancer. 53:399-405. 1986.

34 Marlowe M., J. Stellern, C. Moon, and J. Errera. Main and Interaction Effects of Metallic Toxins on Aggressive Classroom Behavior. Aggressive Behav. 11:41-48. 1985.

35 Pershagen, G., G. Nordberg, and N.E. Bjorklud. Carcinomas of the Respiratory Tract in Hamsters Given Arsenic Trioxide and/or Benzo(a)pyrene by the Pulmonary Route. Environ. Res. (In Press). (As cited in EPA 1984). 1983.

36 Mahaffey, K.R. and B.A. Fowler. Environ. Health Perspect. 19, 165-171. 1977.

37 U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

38 Tseng, W.P. Effects and Dose-Response Relationships of Skin Cancer and Blackfoot Disease with Arsenic. Environ. Health Perspect. 19:109-119. 1977.

39 U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

40 U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

41 U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

42 Tseng, W.P. Effects and Dose-Response Relationships of Skin Cancer and Blackfoot Disease with Arsenic. Environ. Health Perspect. 19:109-119. 1977.

43 Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin, and S. Yeh. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463. 1968.

44 Tseng, W.P. Effects and Dose-Response Relationships of Skin Cancer and Blackfoot Disease with Arsenic. Environ. Health Perspect. 19:109-119. 1977.

disease increased with age and dose. The data in Tseng, et al.[45] also show increased incidences of hyperpigmentation and keratosis with age. A no-observable-adverse effects level (NOAEL) was identified from the Tseng, et al.[46] study based on the absence of the critical effect (hyperpigmentation, keratosis, and possible vascular complications). The NOAEL corresponded to a dose of 8×10^{-4} mg/kg-day. The lowest-observed-adverse-effect-level (LOAEL) corresponded to a dose of 1.4×10^{-2} mg/kg-day. An uncertainty factor of three was applied to the NOAEL to account for the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals. The resulting oral Rfd for arsenic was rounded to 3×10^{-4} mg/kg-day. The studies on which the RfD are based have been given a medium level of confidence, based on the presence of other contaminants and poor characterization of the exposure doses. The supporting human toxicity database is extensive but lacking in some important areas. However, it does support the choice of NOAEL and is given a medium degree of confidence. Therefore, medium confidence is placed in the oral RfD.[47]

EPA interim primary drinking water standard for arsenic is 50 :g/L.[48] This value was established as a maximum allowable level for arsenic in drinking water by the U.S. Public Health Service in 1942, and it continues to be used in the current EPA regulations.[49] EPA's Office of Drinking Water is considering maintaining the present maximum contaminant level (MCL) of 50 :g/L for arsenic in municipal drinking water supplies.[50]

Inhalation Toxicity

Health risks posed by airborne arsenic compounds have been reviewed in considerable detail by EPA,[51] and studies on the carcinogenicity of arsenic compounds were reviewed by the International Agency for Research on Cancer (IARC) in 1980. Risk assessments for exposure to airborne arsenic are presented by OSHA[52] and EPA.[53] The following summary is based on these reviews and risk assessments and on review of the primary literature.

It is well established that inhalation of certain arsenic compounds can cause cancer in humans. Several studies of workers in smelters and plants that manufacture arsenical pesticides have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma.[54]

EPA has derived an inhalation cancer slope factor of 15 $(mg/kg\text{-}day)^{-1}$ based on six occupational exposure studies of two different exposed populations.[55, 56, 57, 58, 59, 60,] These studies have reported an association

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[45] Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin, and S. Yeh. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463. 1968.

[46] Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin, and S. Yeh. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463. 1968.

[47] U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

[48] U.S. Environmental Protection Agency. National Primary Drinking Water Regulations. 140 CFR 141.11. 1992.

[49] U.S. Environmental Protection Agency. Ambient Water Quality Criteria for Arsenic-1984. Office of Water Regulations and Standards. PB85-227445. Washington, DC. EPA 440/5-84-003. January, 1985.

[50] U.S. Environmental Protection Agency. Drinking Water Criteria Document for Arsenic. Office of Water, Washington, D.C. Draft. 1993.

[51] U.S. Environmental Protection Agency. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, DC. EPA 600/8083-021F. March, 1984.

[52] Occupational Safety and Health Administration. Occupational Exposure to Inorganic Arsenic. Fed. Reg. 48:1,864-1,903. 1983.

[53] U.S. Environmental Protection Agency. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, DC. EPA 600/8083-021F. March, 1984.

[54] U.S. Environmental Protection Agency. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, DC. EPA 600/8083-021F. March, 1984.

[55] Brown, C.C. and K.C. Chu. A New Method for the Analyses of Cohort Studies: Implications of the Multistage Theory of Carcinogenesis Applied to Occupational Arsenic Exposure. Environ. Health Perspect. 5:293-308. 1983.

[56] Brown, C.C. and K.C. Chu. Implications of the Multistage Theory of Carcinogenesis Applied to Occupational Arsenic Exposure. JNCI. 70:455-463. 1983.

[57] Brown, C.C. and K.C. Chu. Approaches to Epidemiologic Analysis of Prospective and Retrospective Studies: Example of Lung Cancer and Exposure to Arsenic. In: Environmental Epidemiology: Risk Assessment. Prentic, R.L. and A.S. Whittemore, eds. SIAM, Philadelphia. 1983.

between occupational exposure to arsenic and lung cancer mortality. To derive the inhalation cancer slope factor, the geometric mean was taken within each of the exposed populations and the final inhalation cancer slope factor was the geometric mean of the two exposed populations. Supporting evidence of the carcinogenicity of arsenic has also been found in residents drinking arsenic-containing water and residents living near a pesticide manufacturing plant. EPA assumed 30% absorption of arsenic following inhalation exposure.[61]

The California Environmental Protection Agency (Cal EPA) has developed its own cancer potency factor for inhalation exposure to arsenic.[62] The Cal EPA cancer potency factor of 12 $(mg/kg\text{-}day)^{-1}$ was developed through evaluation of data provided in Welch et al.,[63] Higgins et al.,[64] Lee-Feldstein et al.,[65] Enterline et al.,[66] and CDHS (California Department of Health Services).[67], [68]

No reference concentration is available for inorganic arsenic. Extrapolation from the oral value is deemed inappropriate based on the following considerations. First, the relative sensitivity of various tissues to arsenic exposure via oral and inhalation routes is not clear. Certainly, the skin is the critical target for carcinogenic response following oral exposure, while the lung is the target after inhalation. Since it cannot be determined if the target organ is the same for the two exposures, route-to-route extrapolation is not appropriate. Further, metabolism may influence relative doses by the two routes. Inorganic arsenic is methylated in vivo by a saturable process in the liver. Because of first pass effects, and differences in the rate and extent of absorption following exposure by the two routes, the concentrations of inorganic arsenic which reach critical targets may differ. Again, this suggests that route-to-route extrapolation is inappropriate. Lack of an RfC requires that inhalation exposures to arsenic be assessed qualitatively for systemic effects.

The American Conference of Governmental Industrial Hygienists[69] recommends a time-weighted average Threshold Limit Value (TLV) of 0.2 mg/m^3 for arsenic and soluble compounds of arsenic.

Uncertainties Associated with Estimates of Arsenic Toxicity

There are specific uncertainties regarding the toxicity criteria derived by EPA for arsenic. For example, there continues to be discussion of the oral cancer slope factor for arsenic. Recent reviews and letters[70, 71] present one view of evidence that the oral cancer slope factor for arsenic is too high. Several

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58 Lee-Feldstein, A. Arsenic and Respiratory Cancer in Man: Follow-up of an Occupational Study. In: Arsenic: Industrial, Biomedical and Environmental Perspectives. W. Lederer and R. Fensterheim, eds. Van Nostrand Reinhold, New York. 1983.

59 Higgins, I.T., K. Welch, M. Oh, K. Kryston, C. Burchfield, and N. Wilkinson. Arsenic Exposure and Respiratory Cancer in a Cohort of 8044 Anaconda Smelter Workers. A 43-Year Follow-Up Study. Unpublished Report Submitted to Chemical Manufacturer's Association and Smelters Environmental Research Association. 1985.

60 Enterline, P., V. Henderson, and G. Marsh. Exposure to Arsenic and Respiratory Cancer: A Reanalysis. Am. J. Epidemiol. 125: 929-938. 1987.

61 U.S. Environmental Protection Agency. Integrated Risk Information System. Arsenic. 2000.

62 California Environmental Protection Agency. California Environmental Protection Agency Criteria for Carcinogens. 1995.

63 Welch, K., I. Higgins, M. Oh, and C. Burchfield. Arsenic Exposure, Smoking, and Respiratory Cancer in Copper Smelter Workers. Arch. Environ. Health. 37: 325-335. 1982.

64 Higgins, I.T., K. Welch, M. Oh, K. Kryston, C. Burchfield, and N. Wilkinson. Arsenic Exposure and Respiratory Cancer in a Cohort of 8044 Anaconda Smelter Workers. A 43-Year Follow-Up Study. Unpublished Report Submitted to Chemical Manufacturer's Association and Smelters Environmental Research Association. 1985.

65 Lee-Feldstein, A. Cumulative Exposure to Arsenic and It's Relationship to Respiratory Cancer Among Copper Smelter Employees. J. Occup. Med. 28: 296-302. 1986.

66 Enterline, P., V. Henderson, and G. Marsh. Exposure to Arsenic and Respiratory Cancer: A Reanalysis. Am. J. Epidemiol. 125: 929-938. 1987.

67 California Department of Health Services. Risk-Specific Intake Level for Inhaled Arsenic. Reproductive and Cancer Hazard Assessment Section. Berkeley, CA. 1990.

68 California Department of Health Services. Report to the Air Resources Board on Inorganic Arsenic. Part B. Health Effects of Inorganic Arsenic Compounds. Air Toxicology and Epidemiology Section, Berkeley, CA. 1990.

69 American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio. 1986.

70 Carlson-Lynch, H., B.D. Beck, and P.D. Boardman. Arsenic Risk Assessment. Environ. Health Perspect. 102:354-356. 1994.

71 Beck, B.D., P.D. Boardman, G.C. Hook, R.A. Rudel, T.M. Slayton, and H. Carlson-Lynch. Response to Smith, et al. (letter). Environ. Health Perspect. 103:15-17. 1995.

lines of evidence are advanced to support this conclusion, and all are based on criticisms of the studies of the Taiwanese population from which toxicity criteria for ingested arsenic are derived.

First, a recent study[72] suggests that estimates of inorganic arsenic in the diet of the Taiwanese population may have been underestimated in the past, resulting in an exaggerated estimate of cancer potency. The study measured inorganic arsenic in rice and sweet potatoes, two staples in the Taiwanese diet, and results were interpreted to indicate that inorganic arsenic in these foodstuffs was much greater than previously assumed.

Second, several studies in both humans and laboratory animals were interpreted to indicate that arsenic metabolism is saturable, and that saturation occurs at exposures less than those received by the Taiwanese population. This, in turn, would suggest that the apparent potency of inorganic arsenic as a carcinogen is exaggerated at high doses by reduction in detoxification. At lower doses, efficient metabolism to organic forms would reduce the effectiveness of a given exposure to inorganic arsenic in producing cancer.

Third, dietary methionine, an essential amino acid, may be inadequate in the Taiwanese diet to support both basic metabolic needs and the metabolic demands caused by the ingestion of large amounts of inorganic arsenic. Methionine is likely to be a methyl donor in the conversion of inorganic arsenic to methylated forms, and lack of sufficient methionine in the diet could limit the capacity for arsenic metabolism in the body. This would result in a higher apparent potency of arsenic, since less metabolic detoxification could take place.

Finally, the presence of humic acids in the water supply for the Taiwanese population is suggested as causative or interactive in the production of human cancer. If humic acids do play such a role, exposure to arsenic in the absence of humic acids may not have the same high potential to cause cancer as that seen in the study population.

Though the above studies seem, on the surface, to make a reasonable case for lowering the arsenic oral cancer slope factor, objective examination of all the evidence demonstrates significant flaws in all of the above arguments. An appropriate cancer slope factor can only be developed if the limitations of all information are understood and factored into the analysis. On more thorough examination, it does not appear that sufficient information is currently available on which to base a reevaluation of the arsenic cancer slope factor.

Data presented by Yost et al.[73] are dramatically counter to other measurements of inorganic arsenic in rice and potatoes grown in soils treated with inorganic arsenic. This discrepancy is unexplained, but could be due to strong acid treatment used to extract arsenic in the Yost study. This could have resulted in the artifactual production of inorganic arsenic.[74] The forms of organic arsenic in plants are poorly known, and it is not clear how easily inorganic arsenic can be produced from these forms, nor how this may vary among different plant species. Until such problems are resolved, it will not be possible to revise the cancer slope factor based on the single least conservative study.

Information available on biotransformation in humans is generally weak and difficult to interpret. Moreover, there are conflicting reports which variously suggest that the saturation point for human methylation of inorganic arsenic falls above or below the exposures received by the Taiwanese population.[75] That some reports suggest the former is an important observation. For example, similar percentages for inorganic arsenic, monomethyl arsenic, and dimethyl arsenic were found in urine of subjects in Nevada, exposed on average to levels of arsenic similar to those for the "high dose" group in Taiwan, and in subjects in a control group. The results did not support saturation of metabolism and, in fact, indicated that organic arsenic made up 78 percent of total arsenic in exposed subjects and

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[72] Yost, L., R.A. Schoof, H.R. Guo, P.A. Valberg, B.D. Beck, E. Crecelius, E., and Green, P. Bergstrom. Recalculation of the Oral Toxicity Values for Arsenic Correcting for Dietary Arsenic Intake. Presented at the Society for Environmental Geochemistry and Health Rocky Mountain Conference, Salt Lake City, Utah. July 18-19. 1994.

[73] Yost, L., R.A. Schoof, H.R. Guo, P.A. Valberg, B.D. Beck, E. Crecelius, E., and Green, P. Bergstrom. Recalculation of the Oral Toxicity Values for Arsenic Correcting for Dietary Arsenic Intake. Presented at the Society for Environmental Geochemistry and Health Rocky Mountain Conference, Salt Lake City, Utah. July 18-19. 1994.

[74] Mushak, Paul and A.F. Crocetti. Risk and Revisionism in Arsenic Cancer Risk Assessment (commentary). Environ. Health Perspect. 103:684-689. 1995.

[75] Mushak, Paul and A.F. Crocetti. Risk and Revisionism in Arsenic Cancer Risk Assessment (commentary). Environ. Health Perspect. 103:684-689. 1995.

86 percent in controls.[76] Such a small difference is probably not statistically or biologically significant and is not consistent with a low threshold for saturation of arsenic metabolism.

Similarly, in the study by Buchet et al.,[77] which is often cited in support of a relatively low metabolic threshold, data seem to indicate significant metabolic capability at all doses. Individuals receiving 1,000 :g of inorganic arsenic per day, for example, formed nearly the same proportion of total methyl metabolites as did individuals receiving only 125 µg (74 versus 84 percent, respectively). Such differences are small enough to be due to sampling errors and individual variation. On the basis of metabolite formation, it is difficult to conclude that metabolism has reached saturation.

The key to resolving the issue of metabolism in arsenic would seem to be characteristic of mechanisms of methylation and the study of these biochemical pathways in human systems. In addition, empirical studies should focus on the kinetics of the inorganic arsenic rather than on metabolite formation and metabolite ratios. The latter are indirect measures of the amount of the ultimate carcinogen (assumed to be inorganic arsenic) which reaches target tissues. Moreover, metabolite ratios especially are difficult to interpret and have no demonstrated connection with the amounts of inorganic arsenic that reaches target tissues.

The nutritional status of the Taiwanese appears to be sufficient for normal metabolic processes.[78] In addition, a simple calculation[79] suggests that the amount of methionine that might be necessary to support metabolism of ingested arsenic is at best a small fraction of total daily intake, on the average of less than 1 percent. It seems likely that the "problem" related to nutritional status is really a "red herring." Until such time as new data become available which challenge the above conclusions, it seems safe to dismiss the argument for nutritional deficits as a factor influencing cancer potency in the Taiwanese populations.

Finally, the presence of humic acids in water consumed by the Taiwanese seems unlikely to be a causative factor in cancer. It appears that arsenic, not humic acids, is the constant in the various stages of both Blackfoot disease and precancerous skin lesions.[80] Moreover, both skin cancer and internal cancers are found in patients treated with Fowler's solution where humic acids were not a factor. Thus, it has been reasonably concluded that humic acids are not necessary for the carcinogenic activity of arsenic. It is possible that humic acids could alter the carcinogenic response in humans through some as yet unknown mechanism. Available data are, however, apparently not sufficient to establish this as a possibility, much less quantify such an effect. Until substantial additional data are available, it will not be possible to assess the contribution, if any, of humic acids to carcinogenesis in the Taiwanese population.

It is assumed that uncertainties in the arsenic oral cancer slope factor are best taken into account in the risk management process.

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[76] Warner, M.L., L.E. Moore, M.T. Smith, D.A. Kalman, E. Fanning, and A.H. Smith. Increased Micronuclei in Exfoliated Bladder Cells of Individuals Who Chronically Ingest Arsenic-Contaminated Water in Nevada. Cancer Epidemiol Biomarkers Prev. 3:483-590. 1994.

[77] Buchet, J.P., R. Lauwerys, and H. Roels. Comparison of the Urinary Excretion of Arsenic Metabolites after a Single Oral Dose of Sodium Arsenite, Monomethyl Arsonate, or Dimethyl Arsinate in Man. Int. Arch. Occup. Environ. Health. 48:71-79. 1981.

[78] Engel, R.R. and O. Receveur. Arsenic Ingestion and Internal Cancers: A Review (letter). Am J Epidemiol. 138:896-897. 1993.

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[80] Mushak, Paul and A.F. Crocetti. Risk and Revisionism in Arsenic Cancer Risk Assessment (commentary). Environ. Health *Perspect*. 103:684-689. 1995.

Summary of Arsenic Criteria

Criterion	Value	Source
EPA carcinogen classification	Group A	EPA 2000
Oral slope factor	1.5×10^{0} (mg/kg-day)$^{-1}$	EPA 2000
Inhalation slope factor	1.5×10^{1} (mg/kg-day)$^{-1}$	EPA 2000
RfD	3×10^{-4} mg/kg-day	EPA 2000
Cal EPA inhalation cancer potency factor	1.2×10^{1} (mg/kg-day)$^{-1}$	Cal EPA 1995
Maximum Contaminant Level (MCL)	0.05 mg/L	EPA 1996
Cal Permissible Exposure Limits, PEL	0.01 mg/M^{3} *	CCR, Title 8, 2000**

* Inorganics
** California Code of Regulations, Title 8, Section 5155, February 16, 2000.

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BENZENE[1]

Introduction

Benzene is a volatile, colorless, flammable liquid aromatic hydrocarbon that has a characteristic odor. It is a chemical intermediate in the synthesis of compounds such as styrene, synthetic rubber, and phenol, and it is used as an additive to gasoline to increase the octane.

Potential for Human Exposure

Releases to the Environment

Benzene is released to the environment by both natural and man-made sources; however, natural sources account for only a very small part of benzene releases. Major sources of atmospheric releases include vehicle exhaust emissions, evaporative gasoline fumes, emissions from vehicle refueling (i.e., service stations), and industrial emissions. In 1984, motor vehicle exhaust accounted for almost 80% of total emissions in California. Other sources of atmospheric benzene include cigarette smoke and the exhaled breath of smokers, landfill emissions, off-gassing from particle board, and emissions from structural fires. Benzene is released to soils and water from industrial discharges, landfill leachate, and gasoline leaks from underground storage tanks.

Environmental Fate

Benzene is water-soluble and highly volatile. Atmospheric benzene is removed primarily through chemical degradation. Due to its water-solubility, some benzene is removed from the atmosphere in rainwater. Benzene in soil and water is removed through volatilization, photooxidation, and biodegradation.

Environmental Levels

Air: Benzene is ubiquitous in the atmosphere. It has been detected in outdoor air samples from rural and urban areas and in indoor air. Results of the U.S. Environmental Protection Agency (EPA) Total Exposure Assessment Methodology (TEAM) studies in the 1980s indicated that exposure to benzene in water, food, and beverages is insignificant; more than 99% of total personal exposure to benzene was through inhalation of benzene in the air.[2] Wilson et al.[3] measured indoor and outdoor 48-hour average benzene concentrations at 161 homes throughout much of California. Indoor mean concentrations were 8.3 micrograms per meter cubed (:g/m^3) compared to 6.1 :g/m^3 outdoors.[4]

Twenty-four hour average benzene levels have been measured every twelfth day at about 20 sites throughout California since 1986 by the California Air Resources Board (CARB).[5] From 1986 to 1992, statewide annual average benzene concentrations ranged from 9 to 6 :g/m^3. For the years 1989 to 1992, the average concentration was 7 :g/m^3. In 1993 and 1994, the statewide annual average values dropped to 4 :g/m^3. The decline appears to be due to one or more of several factors: a) the 50% reduction in hydrocarbon emissions mandated for new cars; b) the Stage II vapor recovery controls recently in effect; and c) a reduction in benzene content in gasoline down to the 1% mandated in the 1990 Clean Air Act Amendments. Analysis of the California database indicates seasonal variation in benzene concentrations, with winter values about twice summer values. This may be due to changes in the blend of gasoline or to increased likelihood of inversions during the winter.

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1 Information pertaining to benzene is derived from Agency for Toxic Substances and Disease Registry. Toxicological Profile for Benzene. Prepared by Clement International Corporation for U.S. Department of Health and Human Services, Public Health Service, ATSDR. 1995, as well as other sources, as noted.

2 Wallace, L. Environmental Exposure to Benzene: An Update. Environmental Health Perspectives 104(6): 1129-1136. 1996.

3 Wilson, A.L., S.D. Colome, and Y. Tian. California Residential Indoor Air Quality Study. Volume 1: Methodology and Descriptive Statistics. Irvine, CA: Integrated Environmental Services. 1993.

4 Wilson, A.L., S.D. Colome, and Y. Tian. California Residential Indoor Air Quality Study. Volume 1: Methodology and Descriptive Statistics. Irvine, CA: Integrated Environmental Services. 1993.

5 Wallace, L. Environmental Exposure to Benzene: An Update. Environmental Health Perspectives 104(6): 1129-1136. 1996.

The South Coast Air Quality Management District (SCAQMD)[6] characterized in-vehicle benzene exposure for Los Angeles commuters in summer and winter seasons. In-vehicle benzene exposure averaged 40 :g/m^3 for commuters during rush hour, approximately 5 times greater than concentrations at a fixed outdoor site. Benzene concentration in the gasoline used was not measured; benzene content in gasoline has been reduced from 2 or 3% to 1% since this study was conducted. Smaller studies conducted more recently in North Caroline and New Jersey-New York have also shown increased benzene concentrations while driving.[7] These later studies showed lower in-vehicle exposures, but outdoor concentrations were also less, so the ratio of personal exposure to outdoor concentration continued to range from 5 to 10. Decreased concentrations could be due to the difference in location or could reflect reductions of benzene in gasoline.[8]

The primary source of benzene exposure for cigarette smokers is mainstream cigarette smoke.[9] A typical smoker takes in roughly 2 milligrams (mg) of benzene per day; about 1.8 mg is delivered by mainstream smoke.[10] A typical nonsmoker inhales about 0.2 mg benzene per day. The majority of benzene exposure for nonsmokers is from automotive exhaust or gasoline vapor emissions. This includes most outdoor air benzene exposure, indoor exposures due to intrusion of evaporative gasoline fumes from attached garages, and personal activities such as driving. About 10% of nonsmoker exposure comes from environmental tobacco smoke exposures at home or work. Smokers have an average benzene body burden of about 6 to 10 times that of nonsmokers.[11]

Water: A number of studies have reported finding benzene at concentrations of 5 nanograms per liter (ng/L) in surface water and well water.[12] Assuming ingestion of 2 liters of water daily, this corresponds to a daily intake of 10 ng benzene. This is only 5% of the average daily intake for nonsmokers of 200 ng from air. Studies of benzene exposure while showering in water contaminated by a gasoline spill indicated that 20-minute exposure to volatilized benzene while showering was on the same order of magnitude as a full days exposure to benzene for a typical nonsmoker.[13,] A smoker would still get the majority of exposure through smoking.

Soil and Sediment: Benzene levels ranging from <2 to 191 parts per billion (ppb) were recorded in the vicinity of five industrial facilities using or producing benzene. Data from EPA's Storage and Retrieval (STORET) database (1980 - 1982) showed that benzene had been positively detected in sediment samples taken at 9% of 355 observation stations with a median level of < 5 ppb.

Other Environmental Media: The TEAM study concluded that food and beverages contained minimal concentrations of benzene. Recent studies have confirmed the results of the TEAM study. A U.S. Food and Drug Administration (FDA) study analyzed more than 50 foods for benzene. Most foods contained less than 2 nanograms per gram (ng/g) parts per billion by weight (ppbw).[14] Exceptions included strawberry preserves (38 ng/g), taco sauces (9 and 22 ng/g), duck sauce (7 ng/g), and barbecue sauce (5 ng/g).

Toxicokinetics

Benzene is readily absorbed into the body via ingestion and inhalation. Dermal absorption is somewhat slower. It is stored in the bone marrow, liver, kidney, and body fat. The body metabolizes benzene through several pathways; some of the metabolites formed (i.e., hydroquinone, phenol, and muconic

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6 South Coast Air Quality Management District. In-vehicle Characterization Study in the South Coast Air Basin, Los Angeles. 1989.

7 Wallace, L. Environmental Exposure to Benzene: An Update. Environmental Health Perspectives 104(6): 1129-1136. 1996.

8 Wallace, L. Environmental Exposure to Benzene: An Update. Environmental Health Perspectives 104(6): 1129-1136. 1996.

9 Wallace, L., E. Pellizzari, T. Hartwell, K. Perritt, and R. Ziegenfus. Exposures to Benzene and Other Volatile Organic Compounds from Active and Passive Smoking. Arch Environ. Health 42: 272-279. 1987.

10 South Coast Air Quality Management District. In-vehicle Characterization Study in the South Coast Air Basin, Los Angeles. 1989.

11 South Coast Air Quality Management District. In-vehicle Characterization Study in the South Coast Air Basin, Los Angeles. 1989.

12 Wallace, L. Environmental Exposure to Benzene: An Update. Environmental Health Perspectives 104(6): 1129-1136. 1996.

13 Wallace, L. Environmental Exposure to Benzene: An Update. Environmental Health Perspectives 104(6): 1129-1136. 1996.

14 Wallace, L. Environmental Exposure to Benzene: An Update. Environmental Health Perspectives 104(6): 1129-1136. 1996.

dialdehyde) can produce hematotoxic effects. Following inhalation exposure to benzene, the majority of the compound is excreted unchanged in exhaled air. Absorbed benzene is excreted primarily in the urine following metabolism; some benzene may be accumulated in the body.

Qualitative Description of Health Effects

Carcinogenicity

Many case studies have described a causal relationship between exposure to benzene (concentrations unspecified) by inhalation (either alone or in combination with other chemicals) and leukemia in humans[15] Most cases were acute myelogenous leukemia, although some were monocytic, erythroblastic, or lymphocytic. Various hematological disorders other than leukemia have also been reported; these include pancytopenia (reduction in the number of red blood cells, white blood cells, and platelets) and aplastic anemia (cessation of bone marrow function).

A series of epidemiological studies, both cohort and case-control, showed statistically significant associations between leukemia and occupational exposure (concentration unspecified) to benzene.[16,17,18,19] These results have been replicated in a number of countries and in different industries.[20]

The carcinogenicity of benzene has been evaluated in rats and mice by various routes of exposure (inhalation, oral, dermal, subcutaneous). Oral exposure to benzene has been associated with increased incidences of zymbal gland and mammary gland carcinomas, oral cavity carcinomas, and lymphomas.[21,22,] Inhalation exposure to benzene has been associated with thymic and nonthymic lymphoma, hematopoietic neoplasms, zymbal gland carcinomas, carcinomas of the oral and nasal cavities, and other malignant tumors.[23, 24] Leukemia has been observed in studies in which benzene was administered by subcutaneous injection; however, these studies were limited by lack of controls and high incidences of leukemia in untreated controls.[25]

Mutagenicity

Benzene does not induce gene mutations in bacterial systems and has not been found to be a point mutagen in mammalian cells. However, benzene did induce cytogenetic abnormalities in mammalian cells in vitro (chromosomal aberrations and sister-chromatid exchanges). Several studies demonstrate

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15 International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. 1982.

16 Aksoy, M. Malignancies Due to Occupational Exposure to Benzene. Am. J. Ind. Med. 7:395-402. 1985.

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24 Snyder, C.A., B.D Goldstein, A.R. Sellakumar, I. Bromberg, S. Laskin, and R.E. Albert. The Inhalation Toxicology of Benzene: Incidence of Hematopoietic Neoplasms and Hematotoxicity in AKR/J and C57BL/6J Mice. Toxicol. Appl. Pharmacol. 54:323-331. 1980.

25 International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. 1982.

that benzene exposure of laboratory animals in vivo leads to chromosomal aberrations in bone marrow cells. There is a clear correlation between exposure to benzene and the appearance of chromosomal aberrations in the bone marrow and in peripheral lymphocytes of individuals exposed to high levels of benzene (more than 100 parts per million [ppm]).[26] Examination of workers occupationally exposed to benzene shows increased incidence of lymphocytes with unstable chromosomal aberrations. Additional case studies also support the chromosomal damaging effects of benzene.

Teratogenicity/Reproductive Effects

Data suggest that occupational exposure to benzene may impair reproduction in women, however, findings are inconclusive because the studies are limited. Inhalation experiments conducted in rats, mice, guinea pigs, and rabbits suggest that benzene is not teratogenic at doses that are fetotoxic and embryolethal.[27] Studies with pregnant animals indicate that inhalation exposure to benzene may have adverse effects on the developing fetus, including low birth weight, delayed bone formation, and bone marrow damage. Animal experiments in rats, guinea pigs, and rabbits suggest that exposure to benzene vapors may damage the testicles and ovaries.

Acute/Chronic Effects

The toxic effects of benzene vapors in humans exposed occupationally and in experimental animals include central nervous system effects, hematological effects, and effects on the immune system.[28]

In humans, acute inhalation of benzene concentrations ranging from 300 to 3,000 ppm produces central nervous system effects that include dizziness, drowsiness, headache, vertigo, tremor, delirium, and coma. Acute exposure (5 to 10 minutes) to higher concentrations of benzene vapor (10,000 to 20,000 ppm) can result in death. In cases not resulting in death, individuals exhibited symptoms similar to those reported for lower exposures, such as headaches, nausea, staggering, paralysis, convulsions, and coma. Death is usually the result of respiratory or cardiac failure.[29] In laboratory animals, acute exposures to high concentrations of benzene vapors cause depression of the central nervous system.[30]

Chronic human exposure to benzene vapors can cause a continuum of changes in the circulatory blood elements and bone marrow precursors.[31] Leukopenia, thrombocytopenia, anemia, or combinations of these all occur. In early stages of such blood dyscrasias, effects appear to be reversible. Exposure for longer periods of time may lead to pancytopenia or aplastic anemia, which are irreversible.[32]

Leukopenia is the most commonly observed effect of chronic benzene exposure in laboratory animals. Longer exposure periods may lead to pancytopenia and general bone marrow depression.[33]

Immune system depression by benzene is well known. Depression of serum antibodies (IgG and IgA) in workers exposed occupationally to benzene (exposure concentration unspecified) has been reported.[34]

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[26] International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. 1982.

[27] International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. 1982.

[28] U.S. Environmental Protection Agency. National Primary Drinking Water Regulations, Volatile Synthetic Organic Chemicals, Proposed Rulemaking. Fed. Reg. 50:46,901-46,933. November 13, 1985.

[29] National Academy of Science. Health Effects of Benzene: A Review Committee on Toxicology, Assembly of Life Sciences. National Research Council, Washington, DC. 1976.

[30] U.S. Environmental Protection Agency. Draft Health Advisory for Benzene. Office of Drinking Water, Washington, DC. September 30, 1985.

[31] U.S. Environmental Protection Agency. Draft Health Advisory for Benzene. Office of Drinking Water, Washington, DC. September 30, 1985.

[32] International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. 1982.

[33] U.S. Environmental Protection Agency. National Primary Drinking Water Regulations; Volatile Synthetic Organic Chemicals, Final Rule. Fed. Reg. 50:46,880-46,901. November 13, 1985.

[34] U.S. Environmental Protection Agency. National Primary Drinking Water Regulations; Volatile Synthetic Organic Chemicals, Final Rule. Fed. Reg. 50:46,880-46,901. November 13, 1985.

However, the workers were exposed to multiple solvents making it difficult to conclude that benzene exposure alone was responsible for the adverse effects noted. Cellular immunity is also impacted by benzene exposure; workers exposed chronically to benzene vapors had reduced leukocytes and lymphocytes. It has been demonstrated that administration of benzene to mice inhibits the function of B- and T-lymphocytes tested in vitro.[35] These observations, as well as the well-known ability of benzene to depress leukocytes, may explain why benzene-exposed individuals readily succumb to infection and the terminal event in severe benzene toxicity is often overwhelming infection.[36]

Quantitative Description of Health Effects

Applying EPA's criteria for evaluating the overall weight of evidence of carcinogenicity to humans[37], benzene has been classified in Group A-Human Carcinogen.[38] Epidemiological studies indicating increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and supporting data form the basis for this classification.[39]

The EPA Carcinogen Assessment Group (CAG) calculated an oral cancer slope factor for benzene derived from human epidemiological studies[40,41,42] in which significantly increased incidences of leukemia were observed for workers exposed to benzene principally by inhalation.[43,44] EPA proposed a "single best judgment" estimate of 2.9×10^{-2} $(mg/kg\text{-}day)^{-1}$ [45] A drinking water ingestion unit risk estimate of 8.3×10^{-7} $(:g/L)^{-1}$ was derived by EPA based upon human occupational exposure.[46,47] The concentration in water corresponding to a 10^{-6} excess lifetime cancer risk is 1 :g/L (EPA 2000). Risk estimates based on animal gavage studies are about 5 times higher than those derived from human data. Pharmacokinetic data that could impact the risk assessment are currently being evaluated.

EPA derived an inhalation unit risk of 8.3×10^{-6} $(:g/m^3)^{-1}$ based on the human epidemiological studies used to calculate an oral cancer slope factor (Ott, et al. 1978; Rinsky, et al. 1981; and Wong, et al. 1983). EPA provided an inhalation cancer slope factor of 2.9×10^{-2} $(mg/kg\text{-}day)^{-1}$ in its Integrated Risk Information System (IRIS) database (EPA 2000).

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35 International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. 1982.

36 International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. 1982.

37 U.S. Environmental Protection Agency. Evaluation of the Potential Carcinogenicity of Benzene (71-43-2). Prepared by Carcinogen Assessment Group for the Office of Response. Washington, DC. OHEA-C-073-29. 1986.

38 U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). Benzene. Online; Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. Cincinnati, Ohio. 2000.

39 EPA (U.S. Environmental Protection Agency). Integrated Risk Information System (IRIS). Benzene. Online; Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. Cincinnati, Ohio. 2000.

40 Ott, M.G., J.C. Townsend, W.A. Fishbeck, and R.A. Langner. Mortality Among Individuals Occupationally Exposed to Benzene. Arch. Environ. Health. 33:3-10. 1978.

41 Rinsky, R.A., A.B Smith, R. Hornung, T.G. Filloon, R.J. Young, A.H. Okun, and P.J. Longdrigan. Benzene and Leukemia: An Epidemiologic Risk Assessment. N. Eng. J. Med. 316:1,044-1,050. 1987.

42 Wong, O. An Industry-Wide Mortality Study of Chemical Workers Occupationally Exposed to Benzene. Prepared for the Chemical Manufacturers Association by Environmental Health Associates, Oakland, California. 1983.

43 U.S. Environmental Protection Agency. Evaluation of the Potential Carcinogenicity of Benzene (71-43-2). Prepared by Carcinogen Assessment Group for the Office of Response. Washington, DC. OHEA-C-073-29. 1986.

44 U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). Benzene. Online; Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. Cincinnati, Ohio. 2000.

45 U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). Benzene. Online; Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. Cincinnati, Ohio. 2000.

46 U.S. Environmental Protection Agency. Ambient Water Quality Criteria for Benzene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 40/5-80-0018. NTIS PB 81-117293. 1980.

47 U.S. Environmental Protection Agency. Health Effects Assessment for Benzene. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-037. September, 1984.

California Environmental Protection Agency (Cal EPA) has developed an oral and inhalation cancer potency factor for benzene of 1.0 x 10^{-2} (mg/kg-day)$^{-1}$. Cal EPA has also developed an inhalation unit risk value of 2.9 x 10^{-5} (:g/m^3)$^{-1}$.

EPA (1985d, 1996) promulgated a final drinking water maximum contaminant level goal (MCLG) of zero because benzene is a human carcinogen. A drinking water maximum contaminant level (MCL) of 5 :g/L was finalized in 1987. MCLGs consider only health effects whereas MCLs consider analytical limitations, treatability, occurrence and cost, as well as health effects.

The EPA Office of Drinking Water developed a 1-day and 10-day health advisory (HA) of 200 :g/L for children (EPA 1996). These HAs were based on an inhalation study in which 103 mg/m^3 caused depressed white blood cell counts within two weeks. A dose of 96 mg/m^3 had no effects after two weeks (EPA 1985b). Health advisories for longer exposure periods were not developed because of the potent carcinogenic response of benzene (EPA 1985b).

Neither a reference dose (RfD) nor concentration (RfC) are available for benzene.

The American Conference of Governmental Industrial Hygienists (ACGIH 1995) has recommended an 8-hour time-weighted average threshold limit value of 10 ppm (32 mg/m^3) for occupational exposure to benzene. It was also specified that benzene should not be employed when substitute materials are available. The OSHA national regulation for occupational exposure is an 8-hour time weighted average of 1 ppm.

Summary of Benzene Criteria

Criterion	Value	Source
EPA carcinogen classification	Group A	EPA 2000
Oral cancer slope factor	2.9 x 10^{-2} (mg/kg-day)$^{-1}$	EPA 2000
Inhalation unit risk	8.3 x 10^{-6} (:g/m^3)$^{-1}$	EPA 2000
Inhalation cancer slope factor	2.9 x 10^{-2} (mg/kg-day)$^{-1}$	EPA 2000
Cal EPA Oral cancer potency factor	1.0 x 10^{-2} (mg/kg-day)$^{-1}$	Cal EPA 1995
Cal EPA Inhalation cancer potency factor	1.0 x 10^{-2} (mg/kg-day)$^{-1}$	Cal EPA 1995
Cal EPA Inhalation unit risk value	2.9 x 10^{-5} (:g/m^3)$^{-1}$	Cal EPA 1995
Final MCLG	0	EPA 1996
Final MCL	5 :g/L	EPA 1996
1-day and 10-day HA	200 :g/L	EPA 1996
Ambient Water Quality Criteria (Water and Fish Consumption)	0.66 :g/L	EPA 1986b
Cal Permissible Exposure Limits, PEL	1 ppm	CCR, Title 8, 2000*
Cal Permissible Exposure Limits, STEL	5 ppm	CCR, Title 8, 2000*

* California Code of Regulations, Title 8, Section 5155, February 16, 2000.

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BERYLLIUM

Introduction

Beryllium (Be) is a dark gray metal of the alkaline earth family and is moderately rare in its natural form. Beryllium is used industrially to harden copper, for the manufacture of nonsparking alloys for tools, in the manufacture of lightweight alloys and ceramics, and in the construction of nuclear reactors. However, most beryllium in the environment is released through coal burning operations.

Toxicokinetics

Inhalation is generally the route of exposure of greatest concern for beryllium. Inhaled beryllium is deposited directly into a major target organ for toxic effects (lung) and remains at the site of deposition for extended periods. Once in the lungs, beryllium only slowly mobilizes to the blood. Upon reaching the bloodstream it is rapidly distributed to various tissues and stored, chiefly in pulmonary lymph nodes and bone, for long periods of time. The ultimate storage site is the skeletal tissue.[1] Ingested beryllium is poorly absorbed (<1 percent) in the intestine and therefore quickly passes out of the body in the feces. Distribution in the body is similar for ingested and inhaled beryllium once the metal reaches the bloodstream.

Qualitative Description of Health Effects

Beryllium has been shown to be a carcinogen when injected or inhaled at sufficient levels. When inhaled, beryllium compounds have produced lung cancers in rats and monkeys.[2, 3, 4]

Several epidemeological studies have also correlated beryllium exposure with increase incidence of lung cancer.[5] However, other studies found no such link, and all of the positive studies have been criticized for various methodological flaws.[6] Thus, EPA has classified beryllium as a B2 carcinogen reflecting the relatively strong evidence from animal studies and the inconclusive epidemiologic information.

No data were found concerning human mutagenicity or teratogenic effects by beryllium compounds. However, beryllium does have inhibitory and teratogenic effects on amphibian embryogenesis. For example, limb regeneration in salamander larvae can be inhibited by topical application of beryllium. Also, normal embryonic development is retarded by beryllium (Be^{2+}) treatment of frog and snail eggs.[7]

Inhalation Toxicity

Data on human toxicity from beryllium are only available following inhalation exposures. The lung is the major target organ following inhalation of beryllium in a variety of forms. High levels of beryllium in air can cause an acute pneumonitis (acute beryllium disease) characterized by edema and inflammation. Extreme cases can be fatal. Chronic exposure to low levels of beryllium in air may lead to chronic beryllium disease (berylliosis). This condition results from the formation of granulomatous tissue identical to that seen in sarcoidosis. The main clinical symptom is shortness of breath. Until recently mortality from berylliosis was estimated to be about 30 percent.

Dose-response relationships for chronic beryllium disease have not been established. This is due in part to difficulties in estimating exposures in exposed human populations. However, in some instances,

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1 U.S. Environmental Protection Agency. Reviews of the Environmental Effects of Pollutants: VI. Beryllium. EPA, Cincinnati, Ohio. EPA 600/11-78-028. 1978.

2 Vorwald, A.J. and A.L. Reeves. Pathologic Changes Induced by Beryllium Compounds. AMA Arch. Ind. Health. 19:190. 1959.

3 Reeves, A.L. and A.J. Vorwald. Beryllium Carcinogenesis. II. Pulmonary Deposition and Clearance of Inhaled Beryllium Sulfate in the Rat. Cancer Res. 27:446-451. 1967.

4 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Beryllium. 1988.

5 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Beryllium. 1988.

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7 U.S. Environmental Protection Agency. Reviews of the Environmental Effects of Pollutants: VI. Beryllium. EPA, Cincinnati, Ohio. EPA 600/11-78-028. 1978.

workers from the cleanest plants using beryllium have shown the most severe clinical manifestations. An explanation for these findings could be that the etiology of berylliosis involves a hypersensitivity (allergic) response. This is consistent with the findings that most individuals with berylliosis are sensitive to beryllium. A lymphocyte transformation test can be used in diagnosis of the disease, and corticosteroid treatment can be effective in slowing or stopping progression of the lesions. Moreover, the onset of the disease can occur up to 25 years following exposure and may be triggered by stress or trauma. Currently, data are insufficient to warrant treatment of berylliosis as the most sensitive toxic endpoint. However, for sensitive individuals, it may occur even at very low levels of exposure and, hence, demands consideration in any risk assessment where beryllium is a chemical of concern. To this end, it is important to note that EPA[8] suggests that berylliosis cases are more frequent when air concentrations in the workplace have exceeded the current OSHA standard of 2 $\mu g/m^3$. However, Cullen, et al.[9] reported five cases of berylliosis where workplace air concentrations have been consistently less than $<2\mu g/m^3$. Thus though it appears that the OSHA standard is effective in preventing many berylliosis cases, it may not be completely protective.

Chronic beryllium disease (CBD) is a chronic inflammatory lung lesion that can result from inhalation exposure to beryllium. It is characterized by the formation of granulomas (pathologic clusters of immune cells) and involves a beryllium-specific immune response.

Quantitative Description of Health Effects

Beryllium is classified by EPA a B1 (probable) human carcinogen based on the limited evidence of carcinogenicity in humans exposed to airborne beryllium (lung cancer) and sufficient evidence of carcinogenicity in animals (lung cancer in rats and monkeys inhaling beryllium, lung tumors in rats exposed to beryllium via intratracheal instillation, and osteosarcomas in rabbits and possibly mice receiving intravenous or intramedullary injection).

Studies regarding the potential carcinogenicity of ingested beryllium to humans are not available. EPA[10] describes several cohort mortality studies showing increases in lung cancer in beryllium processing workers[11,12,13,14] and in studies of entrants on the BCR,[15,16] no increases in other types of cancer were observed in any of these studies, but increases in deaths from nonmalignant respiratory disease were observed. The existing unit risk factor for beryllium is 8.4 $(mg/kg\text{-}day)^{-1}$, based on the data that featured poorly defined exposure estimates.[17]

Chronic oral studies of the potential carcinogenicity of beryllium in animals were conducted at dose levels below the MTD, and therefore are inadequate for the assessment of carcinogenicity. Beryllium has been shown to induce lung cancer in rats exposed to beryllium by both inhalation and intratracheal instillation and in monkeys by inhalation. Osteosarcomas have been produced in rabbits and possibly in mice by

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intravenous and intramedullary injection using a variety of beryllium compounds and beryllium metal. No tumors were produced by intracutaneous or percutaneous injections of beryllium compounds.[18]

The majority of studies do not induce gene mutations in bacterial assays with or without metabolic activation. Gene mutations have been observed in mammalian cells cultured with beryllium chloride. Culturing mammalian cells with beryllium chloride, beryllium sulfate, or beryllium nitrate has resulted in clastogenic alterations.[19]

EPA[20] has derived a reference concentration (RfC) for inhalation of beryllium of 2.2×10^{-5} mg/m^3. The RfC is based on beryllium sensitization and progression to chronic beryllium disease (CBD) identified in studies by Kreiss et al. [21] and Eisenbud et al.[22] The Kreiss et al.[23] occupational exposure study identified a LOAEL for beryllium sensitization in workers exposed to 0.55 µg/m^3, while the Eisenbud et al.[24] study suggests a NOAEL of 0.01-0.1 µ/m^3 in community residents living near a beryllium plant. The LOAEL from the Kreiss et al.[25] study was used for the operational derivation of the RfC because the screening method used in the Eisenbud et al.[26] study was less sensitive than the method used in the Kreiss et al.[27] study.

The Kreiss et al. study was conducted of 136/139 of the then-current beryllium workers in a plant that made beryllia ceramics from beryllium oxide powder.[28] Measurements from 1981 and later were reviewed and included area samples, process breathing-zone samples, and personal lapel samples (the last year only). Quarterly daily-weighted average (DWA) exposures were calculated using a formula based on all of these measurements for each job title. General area and breathing zone samples were not recorded until the last quarter of 1985, soon after machining production was transferred to that plant, even though a limited amount of machining had been conducted since 1982.

Beryllium lymphocyte transformation tests were performed by two different laboratories on blood samples collected from 136 employees. Positive results from one or both laboratories were confirmed by analyzing a subsequent blood sample. Of 136 tested employees, 5 had consistently abnormal blood BeLT results from the two laboratories and were diagnosed with CBD based on observation of granulomas in lung biopsy samples. An additional two employees had abnormal blood results from one of the two laboratories and had no granulomas in lung biopsy samples. Both employees developed abnormal blood results in the other laboratory within 2 years. One of these two employees also developed symptoms of CBD. The other employee declined clinical follow-up. An additional case of CBD was found during the study in an employee hired in 1991, who had a nonhealing granulomatous response to a beryllium-contaminated skin wound. Of the eight sensitized workers, seven had worked in machining at some point, while one case had never worked in a production job. The beryllium sensitization rate was 14.3% among the machinists, compared to 1.2% among all other employees.

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[28] Kreiss, K; Mroz, MM; Newman, LS; et al. Machining risk of beryllium disease and sensitization with median exposures below 2 MU-G/M(3). Am J Ind Med 30(1):16-25. 1996.

Eisenbud et al. evaluated beryllium exposure for 11 community cases of CBD. CBD was defined based on limited radiographic and pathologic examination.[29] Measurements downwind from the plant found that the beryllium concentration at 0.75 miles was about 0.045 µg/m^3, and continuous sampling stations found that the average concentration at about 700 feet from the plant (the furthest distance within the affected area) was 0.05 µg/m^3 (range 0 to 0.46 µg/m^3). The emitted beryllium was primarily as beryllium oxide, although beryllium fluoride and beryl (beryllium ore) were also present. The authors estimated that the average exposure levels at 0.75 miles from the plant during the period of exposure monitoring were 0.004 to 0.02 µg/m^3. Averaging this value to 0.01 µg/m^3, and noting that both plant production and emissions were about 10-fold higher in earlier years, the authors estimated that the concentration at 0.75 mile was 0.01 to 0.1 µg/m^3. The similar prevalence of CBD in the community compared to workers exposed to much higher levels (100 to 1,000 µg/m^3) was attributed to the smaller particle size of beryllium emitted to the outside air compared to beryllium particles inside the plant. This study, although limited by classification of CBD, suggest a NOAEL (HEC) of 0.01 to 0.1 µg/m^3 for the development of CBD in a population exposed to beryllium in ambient air.

Summary of Beryllium Criteria

Criterion	Value	Source
EPA carcinogen classification	B1	EPA 2000
Inhalation carcinogenic potency factor	8.4E+0 (mg/kg-day)$^{-1}$	EPA 2000
Inhalation RfD	5.7E-6 (mg/kg-day)	EPA 2000
Oral RfD	2E-3 (mg/kg-day)	EPA 2000
Maximum Contaminant Level (MCL)	0.001 mg/L	EPA 1991c
Maximum Contaminant Level Goal (MCLG)	0.004 mg/L	EPA 1991c
Ambient Water Quality Criteria (AWQC)		
EPA Drinking Water Health Advisories		
Lifetime Health Advisory	Not available	EPA 1991c
Longer-term HA	Not available	
Child4 mg/L		EPA 1991c
Adult20 mg/L		EPA 1991c
Shorter-term HA		
10-day (Child)	30 mg/L	EPA 1991c
One-day HA (Child)	30 mg/L	EPA 1991c
Cal Permissible Exposure Limits, PEL	0.002 mg/m^3	CCR, Title 8, 2000*
Cal Permissible Exposure Limits, STEL	0.005 mg/m^3 **	CCR, Title 8, 2000*

* California Code of Regulations, Title 8, Section 5155, February 16, 2000.
** 30-minute time-weighted average

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CADMIUM[1]

Introduction

Cadmium is an element of the transitional metal series that occurs widely in nature, usually in sulfide or zinc ores. Elemental cadmium is insoluble in water, although many cadmium salts are quite soluble.[2,3] Cadmium is used primarily for the production of nickel-cadmium batteries and for metal plating. Cadmium is also used for pigments, plastics, synthetics, and for alloys.

Potential for Human Exposure

Releases to the Environment

Natural weathering of minerals releases small amounts of cadmium to the environment, but human activities are responsible for the majority of cadmium releases. Anthropogenic sources of cadmium include releases from mining and smelting, fuel combustion, manufacture and use of phosphate fertilizer, application of sewage sludges, waste incineration, and primary and secondary metal production.

Total air emissions of cadmium were estimated to be about 1.4 million pounds annually in the early 1980s; the majority of these emissions (1 million pounds) were from fossil fuel combustion. The remaining emissions were from smelting operations, manufacturing plants, and incinerators.

Anthropogenic sources of cadmium released to water include discharge from industrial facilities, sewage treatment plants, and leaching from landfills and soils. Cadmium may also leach into drinking water supplies from pipes in the distribution system. Estimated industrial discharges to surface water, transfer to public sewage treatment works, and underground injection for 1988 totaled 4,000, 20,000, and 2,000 pounds, respectively.

Principal sources of cadmium released to soil are land disposal of wastes containing cadmium, land application of sewage sludge, and the use of phosphate fertilizers. In 1988, reported industrial releases to land totaled 542,000 pounds.

Environmental Fate

Cadmium and cadmium compounds may exist in air as suspended particulate matter from soil erosion, combustion of fossil fuels, and industrial emissions. Cadmium emitted to air from combustion processes is typically associated with small particles (less than 10 microns in diameter) that are in the respirable range. Smaller particles are subject to long-range transport, have an atmospheric residence time of 1 to 10 days, and may be carried from 100 to a few thousand kilometers before deposited by wet or dry deposition to soil or water. Smelters and other sources typically release larger particles that are removed by wet or dry deposition close to areas downwind of the source.

Cadmium may be released to water by sources such as deposition of cadmium in air, industrial discharges, and leaching and erosion from soils. Cadmium in water is typically removed by precipitation and sorption to mineral surfaces and organic material. Cadmium may redissolve from sediments under varying ambient conditions of pH, salinity, and redox potential. Cadmium in soil may be entrained into air, eroded into water, and may leach into water depending on conditions.

Environmental Levels

Air: Cadmium concentrations in air are generally lower in rural areas than in urban areas. Cadmium air concentrations of up to 7×10^{-3} milligrams per cubic meter (mg/m^3) have been reported in urban areas, while mean levels of cadmium in ambient rural air are less than 1×10^{-6} mg/m^3. Data describing cadmium concentrations in Los Angeles or California air were not located.

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[1] Information pertaining to Cadmium is derived from Agency for Toxic Substances and Disease Registry. Toxicological Profile for Cadmium, April 1993, as well as other sources, as noted.

[2] U.S. Environmental Protection Agency. National Primary Drinking Water Regulations; Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms; Proposed Rule. 40 CFR Part 141. Fed. Reg. 50(219):46,936-47,025. November 13, 1985.

[3] U.S. Environmental Protection Agency. Water Quality Criteria; Notice of Final Ambient Water Quality Criteria Documents. Fed. Reg. 50:30,784-30,796. July 29, 1985.

Water: Cadmium concentrations in natural surface water and groundwater are generally less than 1 microgram per liter (µg/L). A survey of 969 public drinking water supplies revealed an average cadmium concentration of 3 µg/L.

Soil and Sediment: Cadmium concentrations in unpolluted soil vary depending on mineral sources and organic material. Mean cadmium levels in unpolluted topsoil are about 0.25 parts per million (ppm). Topsoil contamination is likely the mechanism for the greatest human exposure to cadmium, mediated through uptake of soil cadmium into edible plants and tobacco.

Other Environmental Media: Cadmium has been detected in nearly all food samples analyzed; cadmium concentrations are lowest in beverages and fruits and highest in leafy vegetables and potatoes. Cadmium concentrations in food samples range from trace contamination to 0.142 ppm. Cadmium is also found in cigarettes at concentrations ranging from 1 to 2 micrograms (µg) per cigarette.

Toxicokinetics

Cadmium is poorly absorbed from the gastrointestinal tract following ingestion. Absorption following inhalation exposure varies depending on particle size. Large particles (>10 microns in diameter) tend to be deposited in the upper airway, while smaller particles (about 0.1 microns) tend to penetrate into the alveoli. While some soluble cadmium compounds may undergo limited absorption in the upper airway, the major site of absorption is the alveoli. Cadmium absorption from cigarettes, as measured in humans, appears to be greater than absorptions of cadmium aerosols measured in animals. The chemical form of cadmium in cigarette smoke is likely to be similar to that produced by other combustion processes. A model was developed, based on the physiology of the human respiratory tree, to predict the kinetics of inhaled cadmium in humans. Model results suggest that only about 5% of particles greater than 10 microns in diameter will be deposited, while up to 50% of particles less than 0.1 micron will be deposited, and that between 50% and 100% of cadmium deposited in the alveoli will ultimately be absorbed. Dermal absorption may occur, however, it is slow. Dermal absorption of cadmium may be of concern in situations where concentrated solutions may contact the skin for several hours or longer.

Once absorbed, cadmium is preferentially distributed to the kidney and liver. Although concentrations are greatest in the kidney and liver, cadmium is found in virtually all tissues following exposure. Cadmium is not known to undergo any direct metabolic conversion. The cadmium (+2) ion does bind to anionic groups in proteins, especially metallothionein. Plasma cadmium circulates primarily bound to metallothionein. Metallothionein binding appears to decrease the toxicity of cadmium; toxicity may occur if excessive cadmium exposure prevents it from becoming bound to metallothionein.

Inhaled or ingested cadmium is excreted in the feces; however, almost all fecal cadmium represents material not absorbed into the body. Excretion of absorbed cadmium occurs very slowly, with urinary and fecal excretion being about equal. Cadmium tends to accumulate in exposed organisms.

Qualitative Description of Health Effects

Cadmium is not an essential element. The toxicology of cadmium has been reviewed by Freiberg, et al., IARC, and EPA.[4,5,6,7,8] Injection of cadmium into laboratory animals results in injection-site sarcomas and testicular tumors of the Leydig cells.[9] A relationship between human exposure to cadmium and cancer of

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4 Freiberg, L.T., M. Piscator, and G. Nordberg. Cadmium in the Environment. 2nd ed. CRC Press, Cleveland, Ohio. 1974.

5 International Agency for Research on Cancer. IARC Monographs on the Evaluation of the Carcinogenic Risks to Humans. Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42. Supplement 7. IARC. Lyon, France. 1987.

6 U.S. Environmental Protection Agency. Health Assessment Document for Cadmium. Research Triangle Park, North Carolina. EPA 600/8-81-023. 1981.

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9 U.S. Environmental Protection Agency. Health Assessment Document for Cadmium. Research Triangle Park, North Carolina. EPA 600/8-81-023. 1981.

the prostate, lung, or kidney has been suggested by several epidemiological studies.[10,11] Cadmium may impair DNA repair but has not been shown to be mutagenic. It is a well-documented animal teratogen.

Cadmium bioaccumulates in mammals, particularly in the kidney and liver.[12,13] Epidemiological studies have revealed an association between nonmalignant pulmonary diseases and inhalation of cadmium. Renal tubular dysfunction, of which the first sign is proteinuria, occurs at lower levels of oral or inhalation exposure to cadmium and may be the primary defect responsible for the bone damage characteristic of Itai-Itai disease. It is also suspected that chronic exposure to cadmium produces hypertension, anemia, sensory loss (particularly smell), endocrine alterations, and immunosuppression in humans.

Quantitative Description of Health Effects

Ingestion Toxicity

EPA has identified an oral reference dose (RfD) for cadmium. The fraction of ingested cadmium that is absorbed appears to vary with the source (i.e., food versus drinking water), therefore, an oral RfD has been developed for cadmium in water of 5E-04 milligrams per kilogram per day (mg/kg-day) and for cadmium in food of 1E-03 mg/kg-day.[14]

A concentration of 200 micrograms of cadmium per gram of wet human renal cortex (µg/g) is the highest renal level not associated with significant proteinuria.[15] A toxicokinetic model is available to determine the level of chronic human oral exposure (no-observed-adverse-effects-level [NOAEL]) which results in 200 µg/g wet human renal cortex; the model assumes that 0.01% day of the cadmium body burden is eliminated per day.[16] Assuming 2.5% absorption of cadmium from food or 5% from water, the toxicokinetic model predicts that the NOAEL for chronic cadmium exposure is 0.005 and 0.01 mg/kg-day from water and food, respectively (i.e., levels which would result in 200 µg/g wet weight human renal cortex). Thus, based on an estimated NOAEL of 0.005 mg/kg-day for cadmium in drinking water and an uncertainty factor (UF) of 10, an RfD of 0.0005 mg/kg-day (water) was calculated; an equivalent RfD for cadmium in food is 0.001 mg/kg-day.[17]

The maximum contaminant level (MCL) established by EPA for cadmium in drinking water is 5 µg/L.[18] This value is based on a reference dose of 5×10^{-4} mg/kg-day after allowing for contribution to daily exposure by other routes.

Cadmium and some of its compounds are known to be carcinogenic in experimental animals exposed by injection or inhalation, but the carcinogenic effects are absent when cadmium is administered orally (EPA 1985b).[19,20]

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[10] Thun, M.J., T.M. Schnorr, A.B. Smith, W.E. Halperin, and B.A. Lemen. Mortality Among a Cohort of U.S. Cadmium Production Workers - An Update. *JNCI*. 74:325-333. 1985.

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Inhalation Toxicity

EPA has classified cadmium as a Group B1, Probable Human Carcinogen, based on limited evidence from occupational epidemiological studies and sufficient evidence of carcinogenicity in rats and mice.[21]

Evidence that exposure to airborne cadmium compounds increases the risk of cancer in humans is limited.[22,23] Although several studies of exposed workers have suggested that airborne cadmium increases the risk of cancer of the lung and prostate, most of the results have been inconclusive because of small sample sizes, lack of statistical significance, confounding effects of other exposures, or other factors. The most recent study,[24] however, showed a significant increase in the number of lung cancer deaths (16 observed versus 6.99 expected) among a group of cadmium smelter workers. Although this finding may be somewhat confounded by the effects of smoking and exposure to arsenic, EPA[25] concluded that neither of the latter was sufficient to explain the observed effect.[26]

Animal studies have provided sufficient evidence of cadmium carcinogenicity via inhalation.[27] Exposure of Wistar rats by inhalation to cadmium as cadmium chloride at concentrations of 12.5, 25 and 50 micrograms per cubic meter ($\mu g/m^3$) for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors.[28] Intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats but rather mammary tumors in males and tumors at multiple sites in males.[29] Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide and sulfide compounds of cadmium to rats and mice.[30]

EPA based its quantitative risk assessment for inhaled cadmium on the study by Thun, et al.[31,32] Estimates of exposure levels and exposure durations were used to compile a single average measure of cumulative exposure of the exposed workers. This was converted to a lifetime average exposure level. The extent of the deviations of the exposure estimates from the actual exposure is unknown. The data were then fitted to a model, which assumed that the effect of exposure to cadmium would be to increase the background rate of lung cancer, by a factor proportional to the lifetime average exposure level. The best estimate of the exposure and response of the exposed population gave rise to a unit risk estimate of 1.8×10^{-3} $(\mu g/m^3)^{-1}$. An inhalation unit risk for cadmium based on the Takenaka et al.[33] analysis is 9.2E-2 $(\mu g/m^3)^{-1}$. While this estimate is higher than that derived from human data [1.8×10^{-3} $(\mu g/m^3)^{-1}$] and thus more conservative, it was felt that the use of available human data was more reliable because of species variations in response and the type of exposure (cadmium salt versus cadmium fume and cadmium oxide).

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21 U.S. Environmental Protection Agency. Integrated Risk Information System. Cadmium. 2000.

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24 Thun, M.J., T.M. Schnorr, A.B. Smith, W.E. Halperin, and B.A. Lemen. Mortality Among a Cohort of U.S. Cadmium Production Workers - An Update. JNCI. 74:325-333. 1985.

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28 Takenaka, S., H. Oldiges, H. Koenig, D. Hochrainer, and G. Oberdoerster. Carcinogenicity of Cadmium Chloride Aerosols in W Rats. JNCI. 70:367-373. 1983.

29 Sanders, C.L. and J.A. Mahaffey. Carcinogenicity of single and multiple intratracheal instillations of cadmium oxide in the rat. Environ. Res. 33: 227-233. 1984.

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The California Environmental Protection Agency (Cal EPA) has developed an inhalation cancer potency factor for cadmium of 1.5E+01 (mg/kg-day)-1 based on data presented in Thun et al.,[34] IARC,[35] CDHS,[36,37] and Cal EPA.[38]

The American Conference of Governmental Industrial Hygienists[39] recommends a time-weighted average Threshold Limit Value (TLV) of 0.01mg/m^3 for total cadmium and cadmium compounds in dust and particulates; for the respirable fraction of cadmium and cadmium compounds,[40] ACGIH recommends a TLV of 0.002 mg/m^3.

Summary of Cadmium Criteria

Criterion	Value	Source
EPA carcinogen classification	Group B1	EPA 2000
EPA inhalation Cancer Slope Factor	6.3×10^{0}	EPA 2000
Cal EPA Inhalation Unit Risk Value	4.2×10^{-3} ($\mu g/m^3$)	CalEPA 1994
Cal EPA Inhalation Cancer Potency Factor	$1.5 \times 10^{+1}$ $(mg/kg\text{-}day)^{-1}$	CalEPA 1994
Oral RfD (water)	5×10^{-4} mg/kg-day	EPA 2000
Oral RfD (food)	1×10^{-3} mg/kg-day	EPA 2000
Maximum Contaminant Level (MCL)	5 µg/L	EPA 1996
Maximum Contaminant Level Goal (MCLG)	5 µg/L	EPA 1996
EPA Drinking Water Health Advisories		
Lifetime Health Advisory (HA)	5 µg/L	EPA 1996
Longer-term HA		
Child	5 µg/L	EPA 1996
Adult	20 µg/L	EPA 1996
Shorter-term HA		
10-day HA (child)	40 µg/L	EPA 1996
One-day HA (child)	40 µg/L	EPA 1996
Cal Permissible Exposure Limits, PEL	0.005 mg/m^{3} *	CCR, Title 8, 2000**
Cal Permissible Exposure Limits, STEL	270 mg/m^3	CCR, Title 8, 2000*

* Soluble salts or metal dust, as Cd
** California Code of Regulations, Title 8, Section 5155, February 16, 2000.

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[36] California Department of Health Services. Report to the Air Resources Board on Cadmium. Part B. Health Effects of Cadmium. Epidemiological Studies and Surveillance Section. Berkeley, California. 1986.

[37] California Department of Health Services. Risk-Specific Intake Levels for the Proposition 65 Carcinogen Cadmium. Reproductive and Cancer Hazard Assessment Section. Berkeley, California. 1990.

[38] California Environmental Protection Agency. (April 10, 1995, April 1, 1996) California Cancer Potency Factors Update. Standards and Criteria Workgroup, Cal EPA. November, 1994.

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CHROMIUM (VI)[1]

Introduction

Chromium is a naturally occurring metal present in low concentrations in the earth's crust. Chromium occurs in several oxidation states ranging from chromium (-II) to chromium (VI). Chemical, physical, and toxicological properties of chromium vary by form; chromium (VI) is profiled in this report.

Chromium (VI) is the second most stable chromium compound, after chromium (III). Although chromium occurs naturally, it is present primarily as chromium (III) in chromite ore. Natural occurrence of hexavalent chromium (chromium [VI]) is infrequent; it occurs in nature in the rare mineral crocoite ($PbCrO_4$). Chromium (VI) is primarily produced from anthropogenic sources.[2]

Chromium (VI) is used extensively in industry, mainly for plating metals such as stainless and alloy steels and aluminum. It is also used as an additive in cleansing agents, paints, catalysts, fungicides, and wood preservatives.

Potential for Human Exposure

Releases to the Environment

Human activities are responsible for the majority of chromium (VI) environmental releases. The Toxics Release Inventory (TRI) indicates that an estimated 9.3 million pounds of chromium were released to air in 1988 from manufacturing and processing facilities in the United States. This should not be considered total chromium releases to air, as only certain types of facilities are required to report to TRI. Of reported chromium releases to air from man-made sources, approximately 64% is chromium (III) from combustion processes and steel production and about 32% is chromium (VI) from chemical manufacture, primary metal production, chrome plating, and cooling towers that use chromate chemicals as rust inhibitors. Chromium (VI) has been detected in fly ash from coal-fired power plants, although the majority of chromium released from combustion processes is chromium (III).

Chromium compound releases to land and water totaled nearly 200 million pounds from 1987 to 1993, according to the Toxics Release Inventory. About 99% of these releases were to land.[3] Disposal of chromium-containing commercial products, releases from industrial organic chemical industries, and disposal of coal fly ash are sources of chromium release to soil. Wastewater discharges from electroplating and textile industries are the most significant anthropogenic sources of chromium (VI) in surface water and groundwater.

Environmental Fate

Chromium is present in air primarily in particulate form. Chromium (VI) in air may be reduced at a significant rate to chromium (III). Chromium particulates have a short residence time in air (<10 days), with the main mechanisms of removal being precipitation and fallout.

Chromium is not volatile; therefore, transport of chromium from water to air is unlikely, except by transport in windblown sea sprays. Chromium (VI) in surface water will eventually be reduced to chromium (III) by organic matter in the water. In water systems, most chromium (III) is expected to transfer to sediment with only small amounts remaining in solution.

Chromium speciation in groundwater depends on redox potential and pH. Chromium (VI) predominates under high oxidation conditions, generally found in shallow aquifers. Speciation also depends on pH; chromium (III) species predominate in more acidic pH, while chromium (VI) is more likely to predominate under pH ranging from 6 - 8.

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1 Information pertaining to Chromium (VI) is derived from Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Chromium. Prepared by Syracuse Research Corporation under subcontract to Clement International Corporation. Prepared for U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. October, 1991, as well as other sources, as noted.

2 Agency for Toxic Substances and Disease Registry. ATSDR Tox FAQs (Frequently Asked Questions). Chromium. April, 1993.

3 U.S. Environmental Protection Agency. National Primary Drinking Water Regulations. Contaminant Specific Fact Sheets. Inorganic Chemicals. Technical Version. Office of Water. EPA 811-95-002-T. 1995.

In most soils, chromium is present as chromium (III). The reduction of chromium (VI) to chromium (III) is facilitated by low pH. In soil and aquatic systems residence times of chromium (III) are much longer than in the atmosphere. Chromium (III) in soil is not very mobile and removal via leaching and runoff is slow.

Environmental Levels

Air: Total chromium concentration in air in the United States is typically less than 0.01 micrograms per cubic meter (µg/m^3) in rural areas and 0.01 to 0.03 µg/m^3 in urban areas.

Water: Chromium concentrations in U.S. river water range from <1 to 30 micrograms per liter (µg/L); concentrations of chromium in lake water generally do not exceed 5 µg/L. The higher levels are related to anthropogenic pollution. The median chromium concentration in Canadian drinking water was <2.0 µg/L, with chromium concentrations ranging from <2.0 to 8.0 µg/L.

Soil and Sediment: Chromium concentrations in soil vary depending on the parent rock composition and presence of contamination. Chromium ranged in concentration from 1 to 2,000 milligrams per kilogram (mg/kg) in U.S. soils.

Other Environmental Media: Total chromium concentrations in most foods are low, generally ranging from <20 to 520 micrograms per kilogram (µg/kg). Chromium concentrations in oysters, mussels, clams, and mollusks are higher, ranging from <0.1 to 6.8 mg/kg (dry weight). Cigarette tobacco contains 0.24 to 14.6 mg/kg chromium; estimates of chromium levels in inhaled cigarette smoke are not available.

Workers in industries that use chromium can be exposed to chromium concentrations two orders of magnitude greater than the general population. For most occupations, exposure is due to both chromium (III) and chromium (VI) present as soluble and insoluble fractions; exposures primarily to chromium (VI) alone occur in the chrome plating and pigment industries. In the past, workers in these industries may have been exposed to airborne concentrations of chromium ranging from 5 to 600 µg/m^3; better emission controls have caused chromium exposures to decline significantly since the 1970's.

Toxicokinetics

Absorption of chromium (VI) takes place following inhalation, ingestion, and dermal exposure. Following inhalation exposure, chromium (VI) is transported more rapidly and extensively to the bloodstream than chromium (III). Studies indicate that 53 - 85% of chromium (VI) compounds of particle size less than 5 micrometers (:m) are cleared from the lungs by absorption or mucociliary clearance in the pharynx, the rest remain in the lungs.

Absorption of chromium (VI) following oral exposure ranges from about 2 percent[4] to about 10 percent.[5] Absorption is probably limited by reduction of chromium (VI) to chromium (III) in the low pH of the gastric juice, although in vivo measurements have not been made. Chromium (III) absorption may be as little as one-tenth that for chromium (VI).[6]

Dermal absorption is implied by experiments using human volunteers in which some chromium (VI) was found in urine following dermal exposure.[7] In a single animal study, a dermal absorption rate of 0.69 to 0.725 micromoles per hour per centimeter squared (µmol/h/cm^2) was estimated for Na_2CrO_4 for guinea pig skin.[8] This flux may be compared to that of water vapor for humans (28 µmol/h/cm^2),[9] anisole (9 µmol/h/cm^2), aniline (20 µmol/h/cm^2), benzaldehyde (18.5 µmol/h/cm^2) and 2-phenyl ethanol (5 µmol/h/cm^2) (all from Barry, et al. 1984).[10] All the latter are relatively water soluble organic compounds

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4 Ogawa, E. Experimental Study on Absorption, Distribution and Excretion of Trivalent and Hexavalent Chromes. Japanese J. Pharmocol. 26:92. 1976.

5 Donaldson, J.R., R.M. and R.F. Barreras. J. Lab. Clin. Med. 68, 489-493. 1966.

6 Donaldson, J.R., R.M. and R.F. Barreras. J. Lab. Clin. Med. 68, 489-493. 1966.

7 Samitz, M.H. and J. Shrager. Patch Test Reactions of Hexavalent and Trivalent Chromium Compounds. Arch. Dermatol. 94:304-306. 1966.

8 Wahlberg, J.E. and E. Skog. Percutaneous Absorption of Trivalent and Hexavalent Chromium. Arch. Dermatol. 92:315-318. 1965.

9 Scheuplein, R.J. and I.H. Blank. Mechanism of Percutaneous Absorption. IV. Penetration of Nonelectrolytes (Alcohols) from Aqueous Solutions and from Pure Liquids. J. Invest. Dermatol. 60:286-326. 1973.

10 Barry, B.W., S.W. Harrison, and P.H. Dugard. Vapor and Liquid Diffusion of Model Penetrants through Human Skin: Correlation with Thermodynamic Activity. J. Pharm. Pharmacol. 37:226-236. 1984.

with molecular weight similar to that of chromate anion (93-122 versus 114), and have flux rates 10 times or more greater.

Metabolism and sequestration of chromium (VI) in the body may play an important role in limiting systemic toxicity. Chromium (VI) can be rapidly reduced to chromium (III) by ascorbate; reduction of chromium (VI) to chromium (III) by glutathione can also occur; much of absorbed chromium (VI) may be "detoxified" by this route.[11, 12] Reduction of chromium (VI) can also result in the formation of chromium (V), which may interact with deoxyribonucleic acid (DNA), eventually leading to cancer. It is unlikely that significant oxidation of chromium (III) takes place in vivo.[13,14] Moreover, chromium (III) is also effectively reduced in the plasma and can subsequently undergo transformation to high molecular weight complexes which have little biologic activity.[15] If the capacity to reduce chromium (VI) is not exceeded, and chromium (III) polymerization proceeds at a sufficient rate, little active chromium (VI) or chromium (III) will be delivered to potential target organs following absorption into the bloodstream.

Of chromium compounds distributed to target organs, the highest concentrations are typically found in the lungs, hilar lymph nodes, spleen, liver, kidney, and heart. Chromium can be transferred to fetuses through the placenta and to infants through breast milk.

Following absorption and metabolism, chromium is primarily excreted through urine as chromium (III). Chromium (III) has the ability to form chromium protein complexes which can be eliminated by the kidneys. Due to low absorption, the majority of ingested chromium is excreted in the feces. Following administration of an acute, oral dose of chromium (III) or chromium (VI) to humans, the amount of chromium in 6 days of fecal collection was 99.6% for chromium (III) and 89.4% for chromium (VI). The amount of chromium in a 24-hour urine collection was 0.5% and 2.1% for chromium (III) and chromium (VI) compounds, respectively, corresponding to the amount of chromium absorbed into the bloodstream.

Qualitative Description of Health Effects

Hexavalent chromium compounds are strong oxidizing agents and are severely irritating and corrosive. Acute inhalation exposure to chromium (VI) may cause asthma attacks in sensitive individuals; concentrations at which these effects occur were not described. Acute inhalation exposure to chromium fumes may also cause "metal fume fever," a flu-like illness with metallic taste, fever, chills, and muscle aches lasting about 24 hours.[16] Acute ingestion of chromium (VI) may cause stomach upset and ulcers, convulsions, kidney and liver damage, and possibly death. These effects were observed in individuals following ingestion of chromium (VI) concentrations ranging from 4.1 milligrams to several grams; individuals consuming unknown quantities of compounds containing chromium (VI) also experienced these effects. Ingestion of large single doses ($\leq$ 2 grams) of chromium (VI) can cause renal tubular necrosis.[17] Skin contact with chromium (VI) may lead to skin ulcers and allergic reactions, such as redness and swelling.[18]

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[11] Wiegand, H.J., H. Ottenwaelder, and H.M. Bolt. The Reduction of Chromium (VI) to Chromium (III) by Glutathione: An Intracellular Redox Pathway in the Metabolism of the Carcinogen Chromate. Toxicology. 33(3-4):341-248. 1984.

[12] Korallus, U. Biological Activity of Chromium (VI) – Against Chromium (III) Compounds: New Aspects of Biological Monitoring. In: D.M. Serrone, ed. Chromium Symposium 1986: An Update. Pittsburgh, Pennsylvania: Industrial Health Foundation Inc., pp. 210-230. 1986.

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[15] Anderson, R.A. Nutritional Role of Chromium. Sci. Total. Environ. 17:13-29. 1981.

[16] U.S. Environmental Protection Agency. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-R-96-001. February, 1996.

[17] Langard, S. and T. Norseth. Chromium. In: L. Friberg, G.F. Nordberg, V.B. Vouk, eds. Handbook on the Toxicology of Metals, Vol. II. Amsterdam: Elsevier Science Publishers, pp. 185-210. 1986.

[18] Agency for Toxic Substances and Disease Registry. ATSDR Tox FAQs (Frequently Asked Questions). Chromium. April, 1993.

Chronic inhalation of dust containing chromium (VI) concentrations greater than 2 $\mu g/m^3$ may cause respiratory irritation, perforation or ulceration of the nasal septum and decreased spirometric values.[19] In addition, several investigators have associated chronic exposure to chromium (VI) dust with emphysema, chronic bronchitis, polyps, chronic inflammation, and other respiratory conditions in occupational settings.

No systematic adverse effects have been reported in humans following chronic oral exposure to chromium (VI) compounds. Similarly, chronic systemic effects have not been reported in animals even after lifetime oral exposure to chromium (VI).[20] Interperitoneal injection of chromium (VI) can, however, cause a variety of effects, including renal tubular necrosis, in animals.[21] Thus, the lack of systemic toxicity in humans and animals following chronic oral exposure is not due to the lack of intrinsic toxicity. Rather, as discussed below, it is likely due to the kinetics of absorption and distribution of chromium (VI) following ingestion.

Dermal exposure to chromium (VI) can cause irritation and ulceration when exposures are large. Further, smaller exposures may lead to hypersensitivity reactions. Recent reports in abstract form suggest that 10 percent of sensitized individuals will respond to 10 parts per million (ppm) K_2CrO_4 in a patch test[22] and that to protect the most sensitive individuals, a clean-up level to 5 micrograms per centimeter squared ($\mu g/cm^2$) on surfaces would have to be achieved.[23]

Finally, chronic exposure to chromium-bearing dusts via inhalation has been associated with lung cancer in occupationally exposed workers in a number of studies. Unfortunately, exposure data have not been sufficient to clearly establish the form(s) of chromium responsible for the increases in lung cancer. However, it has been generally accepted that chromium (VI) compounds are likely to be the key etiologic agents. This is consistent with the findings that in vitro chromium (VI) compounds can enter cells readily, while chromium (III) compounds are largely excluded. Likewise, chromium (VI) but not chromium (III) is effective at low concentrations in induction of chromosome aberrations and sister chromatid exchanges (SCEs), gene mutation and cell transformation. A few studies have measured increases in chromosome aberrations and SCEs in the peripheral lympnocytes of workers exposed to soluble chromium (VI) compounds.

Chronic oral exposure to chromium (VI) compounds did not cause increased tumor incidence in rats.[24]

Quantitative Description of Health Effects

EPA has classified inhaled chromium (VI) as Group A - Human Carcinogen.[25] EPA[26] based its quantitative risk assessment for inhaled hexavalent chromium on a study by Mancuso.[27] Mancuso's study showed excess risks of lung cancer in workers exposed to chromates between 1931 and 1937 and followed until 1974. Lung cancer risks increased with duration of exposure and with age. Estimates of cumulative exposure to soluble, insoluble, and total chromium were derived from a single set of industrial hygiene measurements taken in 1949. Smoking habits of the workers were not determined or discussed. For lifetime exposure the "unit risk" was calculated to be 1.2×10^{-2} $(\mu g/m^3)^{-1}$.[28] Expressed in terms of total

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[19] Lindberg, R., G. Hedenstierna. Chromeplating: Symptoms, Finding in the Upper Airways, and Effects on Lung Function. Arch Environ Health. 38:367-374. 1983.

[20] Mackenzie, R.D., R.V. Byerrum, C.F. Decker, C.A. Hoppert, and F.L. Langham. Chronic Toxicity Studies II. Hexavalent and Trivalent Chromium Administered in Drinking Water to Rats. AMA Arch Ind Health. 18:232-234. 1958.

[21] U.S. Environmental Protection Agency. Health Effects Assessment for Hexavalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-019. 1984.

[22] Mylanvarapu, V.B. and Trur-Jenn Sun. Chromium Contact Dematitis - A Health Based Risk Assessment Approach. The Toxicologist. 11(1):194 (Abstract). 1991.

[23] Symms, K.G. A Health Assessment of Chromium Residues Following Cleanup of a Large Dichromate Spill at a Public Facility. The Toxicologist. 11(1):194 (Abstract). 1991.

[24] Mackenzie, R.D., R.V. Byerrum, C.F. Decker, C.A. Hoppert, and F.L. Langham. Chronic Toxicity Studies II. Hexavalent and Trivalent Chromium Administered in Drinking Water to Rats. AMA Arch Ind Health. 18:232-234. 1958.

[25] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). Chromium VI. 2000.

[26] U.S. Environmental Protection Agency. Health Effects Assessment for Hexavalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-019. 1984.

[27] Mancuso, T. F. International Conference on Heavy Metals in the Environment. Toronto, Canada. October 27-31. 1975.

[28] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). Chromium VI. 2000.

intake via inhalation, the cancer potency factor was calculated as 41 (milligrams per kilogram per day [mg/kg-day])$^{-1}$.[29]

Confidence in EPA's unit risk is attenuated by several factors. Although results of studies of chromium exposure are consistent across locations and investigators and a dose-response relationship has been established, the Mancuso study based its exposure calculations on the assumption that the ratio between chromium (III) and chromium (VI) was 6:1. This was the assumed minimum chromium (VI) content and could lead to a 7-fold underestimation of risks. On the other hand, the 1949 hygiene data may have underestimated actual exposures that could lead to overestimation of risk. Finally, the implicit assumption in the study that smoking rates were similar in the worker and general populations may cause an overestimation of risk, since smoking rates are often higher among industrial workers.[30]

For oral exposure to chromium (VI), a subchronic allowable intake (AIS) of 0.025 mg/kg-day was derived in the Health Effects Assessment for Hexavalent Chromium.[31] The AIS was based on a one-year study in which rats were exposed to 0 to 25 mg/L chromium (VI) as potassium chromate in drinking water. Increased tissue concentrations of chromium, but no adverse health effects were reported at the highest dose.[32,33]

An oral chronic allowable intake (AIC) of 0.005 mg/kg-day was derived from the same study, with application of an uncertainty factor of 100 to account for both the expected interhuman and interspecies variability in chemical toxicity and an additional safety factor of five to adjust for less than lifetime exposure.[34] This AIC has been adopted by EPA as the reference dose (RfD) for chromium (VI) compounds. EPA has given the RfD, database, and study used to develop the RfD a low confidence rating. Low confidence is placed in the selected study due to the small number of animals tested, the number of parameters measured, and the lack of toxic effects at the highest dose tested. Confidence in the database is low because the supporting studies are of low quality and teratogenic and reproductive endpoints are not well studied.[35] Low confidence in the RfD follows.

The California EPA (Cal EPA) has developed cancer potency factors for chromium (VI); the Cal EPA oral cancer potency factor for chromium (VI) is 0.42 $(mg/kg\text{-}day)^{-1}$ and the inhalation cancer potency factor is 525 $(mg/kg\text{-}day)^{-1}$. These cancer potency factors are based on EPA estimates of cancer potency as well as independent evaluation of available data by California Department of Health Services.[36]

The drinking water maximum contaminant level (MCL) and maximum contaminant level goal (MCLG) are for total chromium; both the MCL and MCLG are 0.1 mg/L. Health Advisories (HAs) are available for total chromium only. The one-day, 10-day, and longer-term HAs for children are 1, 1, and 0.2 mg/L, respectively. The longer-term HA for adults is 8×10^{-1} mg/L, and the lifetime HA is 1×10^{-1} mg/L.[37]

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[29] U.S. Environmental Protection Agency. Health Effects Assessment for Hexavalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-019. 1984a.

[30] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). Chromium VI. 2000.

[31] U.S. Environmental Protection Agency. Health Effects Assessment for Hexavalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-019. 1984.

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[36] California Environmental Protection Agency. California Environmental Protection Agency Criteria for Carcinogens. Hazardous Waste Toxicology Section. 1995.

[37] U.S. Environmental Protection Agency. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-R-96-001. February, 1996.

Summary of Chromium Criteria

Criterion	Value	Source
EPA carcinogen classification (inhalation of Chromium VI only)	Group A	EPA 2000
Inhalation carcinogenic potency factor	42 $(mg/kg\text{-}day)^{-1}$	EPA 2000
Cal EPA inhalation cancer potency factor	525 $(mg/kg\text{-}day)^{-1}$	Cal EPA 1995
Cal EPA oral cancer potency factor	0.42 $(mg/kg\text{-}day)^{-1}$	Cal EPA 1995
Oral RfD (Chromium VI)	0.005 (mg/kg-day)	EPA 2000
Maximum Contaminant Level (MCL) (Total Chromium)	0.1 mg/L	EPA 1996
Maximum Contaminant Level Goal (MCLG) (Total Chromium)	0.1 mg/L	EPA 1996
EPA Drinking Water Health Advisories		
Lifetime Health Advisory (HA)	0.1 mg/L	EPA 1996
Longer-term HA		
Child	0.2 mg/L	EPA 1996
Adult	0.8 mg/L	EPA 1996
Shorter-term HA		
10-day HA (child)	1 mg/L	EPA 1996
One-day HA (child)	1 mg/L	EPA 1996
Cal Permissible Exposure Limits, PEL	0.5 mg/m^3 *	CCR, Title 8, 2000**
Cal Permissible Exposure Limits, PEL	0.05 mg/m^3 ***	CCR, Title 8, 2000**

* Chromium metal, chromium(II) compounds, chromium (III) compounds
** California Code of Regulations, Title 8, Section 5155, February 16, 2000.
*** Chromium (IV) compounds (water soluble and certain water insoluble compounds)

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DIESEL PARTICULATE EMISSIONS

Introduction

Diesel fuel is a complex mixture of thousands of individual compounds, most with carbon numbers between 10 and 22. Most of these compounds are members of the paraffinic, naphthenic, or aromatic classes of hydrocarbons. Generally, more than half of the molecules in diesel fuels contains at least 15 carbon atoms.

Exhaust from diesel fuel combustion is comprised of gases, vapors, and fine particles. Regulated components of diesel exhaust include, but are not limited to, carcinogens such as benzene, arsenic, nickel, 1,3-butadiene, and formaldehyde, and systemic toxicants such as carbon monoxide, fine particulate matter (PM), nitrogen oxides, sulfur dioxide, and various polycyclic aromatic hydrocarbons (PAHs), including benzo(a)pyrene. Most researchers, including World Health Organization (WHO),[1] believe that the PM fraction is responsible for the majority of the risk from exposures to diesel exhaust because many of the harmful organics and metals present in the exhaust are carried on or within diesel particles (California Air Resources Board [CARB], 1997).[2] Diesel PM is formed primarily through the incomplete combustion of diesel fuel. PM in diesel exhaust can be emitted from on- and off-road vehicles, stationary area sources, and stationary point sources.

Typical diesel exhaust particles have diameters ranging from 0.1 to 0.25 micrometers (μm). The particles are mainly aggregates of spherical elemental carbon particles coated with organic and inorganic substances.[3]

Diesel exhaust PM is removed from the atmosphere through physical processes including accretion (aggregation) of particles, atmospheric fall-out (dry deposition), and atmospheric removal by precipitation (wet deposition). According to Pierson et al.,[4] diesel PM is expected to remain in the atmosphere from five to 15 days.

Toxicokinetics

The primary route by which humans are exposed to diesel exhaust PM is via inhalation, although it may be absorbed dermally and gastrointestinally to lesser degrees. No information is available regarding the extent of absorption or the distribution of diesel exhaust PM in the human body.

Data on the excretion and lung clearance of diesel exhaust PM are limited. The available information suggests that diesel exhaust PM and/or its metabolic products are excreted primarily in urine.

Qualitative Description of Health Effects

Human exposures to diesel exhaust PM are primarily associated with vehicle engine emissions, although point and area stationary sources may make significant contributions in some instances. Numerous epidemiological and clinical studies have conclusively shown that exposure to PM in diesel emissions is associated with increases in respiratory illnesses such as bronchitis, emphysema and asthma, as well as premature deaths from cardio-pulmonary disorders[5]. A study by Pope et al.[6] demonstrated that human exposures to airborne respirable PM present in diesel emissions are associated with increased morbidity and mortality, with observed effects including respiratory symptoms, changes in lung function, and increased hospitalizations for respiratory and cardiovascular disease. Pulmonary function was observed

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1 World Health Organization. Diesel fuel and exhaust emissions. Environmental Health Criteria 171. Geneva. pp. 91-343. 1996.

2 California Air Resources Board. Toxic Air Contaminant Identification – Diesel Exhaust. AB 1807. September, 1997.

3 California Air Resources Board. Toxic Air Contaminant Identification – Diesel Exhaust. AB 1807. September, 1997.

4 Pierson, W.R., R.A. Gorse, A.C. Szkariat, W.W. Brachaczek et al. Mutagenicity and chemical characteristics of carbonaceous particulate matter from vehicles on the road. Environ. Sci. Technol. 17: 31-44. 1983.

5 California Air Resources Board. Toxic Air Contaminant Identification – Diesel Exhaust. AB 1807. September, 1997.

6 Pope, C.A., M.J. Thun, M.M. Namboodir, D.W., Dockery et al. Particulate air pollution as a predictor of mortality in a prospective study of US adults. Am. J. Respir. Crit. Care Med. 151 (3 pt 1): 669-74. 1995.

to improve in workers when diesel exhaust was removed, according to a pair of studies by Ulfvarson et al.[7,8]

The noncancer toxicity of diesel emissions is considered to be due to the insoluble carbon particle core based on the fact that, in numerous chronic animal studies, long-term effects seen with whole diesel exhaust (including PM) are generally not observed or are significantly reduced in laboratory animals exposed to similar concentrations of diesel exhaust filtered to remove most of the particles.[9]

Diesel exhaust, particularly the PM fraction, may be carcinogenic as well, based on epidemiology and experimental studies. High levels of both diesel exhaust and carbon black (which lacks adsorbed organic compounds) have produced lung tumors in laboratory rats.[10]

As presented in CARB,[11] epidemiological studies in truck drivers, transport and equipment workers, dock workers, and railway workers reported statistically significant increases in the incidence of lung cancer associated with exposure to diesel exhaust. Two studies reported no category with a risk ratio elevated for exposure to diesel exhaust. Statistically significant increases in tumor incidence were observed in several studies involving rats exposed to diesel exhaust for at least 24 months.[12] In addition, a 1995 report by the Health Effects Institute (HEI)[13] showed a weak association lung cancer and diesel exposure in occupationally exposed individuals

Sufficient data are not available regarding the ability of diesel exhaust PM to induce reproductive, developmental, or teratogenic effects in humans.[14]

Quantitative Description of Health Effects

According to the US EPA's Integrated Risk Information System (IRIS)[15] database, diesel particulate emissions have not undergone a complete evaluation and determination under the IRIS program for evidence of human carcinogenic potential; EPA has heretofore not derived a cancer slope factor for diesel exhaust. However, under Proposition 65 the State of California has determined that diesel engine exhaust is a carcinogen. [16] As a result, a cancer unit risk factor was derived for whole diesel exhaust by the State of California. In addition, the International Agency for Research on Cancer (IARC) concluded in 1989 that sufficient evidence exists that whole diesel exhaust probably causes cancer and classified diesel exhaust in Group 2A (probable human carcinogen). In addition, the National Institute of Occupational Safety and Health (NIOSH) recommended that whole diesel exhaust be considered a potential occupational carcinogen.[17]

Several inhalation assays performed in rodents have demonstrated that diesel exhaust causes cancer. For example, increases in the incidence of lung tumors were observed in seven studies in which rats were

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7 Ulfvarson, U., R. Alexandersson, M. Dahlgvist, U. Elkholm, and B. Bergstrom. Pulmonary function in workers exposed to diesel exhausts: the effect of control measures. Am. J. Ind. Med. 19(3): 283-9. 1991.

8 Ulfvarson, U., R. Alexandersson. Reduction in adverse effect on pulmonary function after exposure to filtered diesel exhaust. Am. J. Ind. Med. 17(3): 341-7. 1990.

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10 Mauderly J.L., M.B. Snipes, E.B. Barr, S.A. Belinsky, et al. Part I, Neoplastic and nonneoplastic lung lesions. Inn: Pulmonary Toxicity of Inhaled Diesel Exhaust and Carbon Black in Chronically Exposed Rats. Research Report Number 68. Health Effects Institute, Cambridge, MA. 1994.

11 California Air Resources Board (CARB). Toxic Air Contaminant Identification – Diesel Exhaust. AB 1807. September, 1997.

12 California Environmental Protection Agency (CalEPA). Health Risk Assessment for Diesel Exhaust. Office of Environmental Health Hazard Assessment. Air Toxicology and Epidemiology Section. 1998.

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14 California Air Resources Board (CARB). Toxic Air Contaminant Identification – Diesel Exhaust. AB 1807. September, 1997.

15 California Air Resources Board. Draft Report of Advisory Committee, Risk Management Subcommittee – Scenario Ten, Commercial Airport Activities. January, 2000.

16 California Code of Regulations. Title 22. Division 2. Part 2. Subdivision 1. Chapter 3.§12000. Chemicals Known to the State to Cause Cancer or Reproductive Toxicity. March 10, 2000.

17 National Institute of Occupational Safety and Health (NIOSH). Division of Standards Development and Technology Transfer. Current Intelligence Bulletin 50 – Carcinogenic Effects of Exposure to Diesel Exhaust. Publication No. 88-116. Cincinnati, OH. August, 1988.

exposed to greater than 2 mg/m^3 of whole diesel exhaust for at least 24 months.[18,19,20] Diesel exhaust concentrations of 2.0 mg/m3 and greater were observed to exhaust the lung clearance capacity in rats in these studies. Similar studies using mice and hamsters produced mixed and negative results, respectively. Based on the results of the studies using rats, CARB derived a cancer unit risk factor of 3.0 x 10^{-1} (mg/m^3) for diesel exhaust, particularly the PM fraction.

For quantification of non-cancer effects, EPA has derived a Reference Concentration (RfC) for inhalation of whole diesel engine emissions, based on the results of two separate chronic inhalation studies conducted on rats by Ishinishi et al.[21] and Mauderly et al.[22] For the Ishinishi et al.[23] study, groups of Fischer 344 rats were exposed to different concentrations of either whole or filtered diesel exhaust for 30 months. The critical effect observed in the Ishinishi et al.[24] study was histological changes in the lung (lowest observed adverse effect level [LOAEL] of 0.9 milligrams per cubic meter {mg/m^3}). The Mauderly et al.[25] study involved exposure of 364-367 rats and mice per exposure level to target diesel exhaust concentrations for up to 30 months. Critical effects observed in the Mauderly et al.[26] study were inflammatory, histological and biochemical changes in the lung and impaired particle clearance (LOAEL of 3.47 mg/m^3). The chronic RfC for diesel exhaust was developed using the results of the studies and an uncertainty factor of 30 which reflects a factor of 10 to protect sensitive individuals and a factor of 3 to adjust for interspecies extrapolation. The resulting RfC is 5 x 10^{-3} mg/m^3. This RfC equates to a daily dose of 0.00143 mg/kilograms per day (kg-day).

Summary of Diesel Exhaust PM Criteria

Criterion	Value	Source
RfC	5 x 10^{-3} mg/m^3	EPA 2000
California Cancer Unit Risk Factor	3.0 x 10^{-1} (mg/m^3)$^{-1}$	CARB 2000

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DIOXINS AND FURANS

Introduction

Polychlorinated dibenzo-p-dioxins (PCDDs) are a family of 75 congeners, each of which is an isomer of one of eight homologous PCDDs with varying degrees of chlorination. Polychlorinated dibenzofurans (PCDFs) are a closely related family of 135 compounds containing up to eight chlorine atoms. These families are referred to generically as "dioxins" and "furans," respectively. PCDDs and PCDFs are considered together because of their similar toxic effects. PCDD/PCDF isomers are not naturally occurring substances, but are formed as contaminants or impurities during chemical production or pyrolysis. Although there is general agreement that PCDD/PCDF isomers are produced by burning wood and by incinerators burning chlorinated wastes,[1] PCDD/PCDF isomer production from combusting coal and hydrocarbons (such as occurs in gas burners and auto and truck engines) has not been confirmed.[2] Experiments indicate that dioxin is produced during the burning of specific chemicals such as chlorinated phenols, polychlorinated benzenes, and polychlorinated diphenyl esters.[3]

Toxicokinetics

Absorption of 2,3,7,8-tetrachlorodibenzo(p)dioxin (2,3,7,8-TCDD) appears to be efficient (>87 percent) based on a study involving a single human volunteer.[4] Studies in animals support this finding[5] and also indicate that the presence of food in the gastrointestinal tract may limit absorption. Fries and Marrow[6] found that absorption was about 50 percent when TCDD was administered in the diet of rats. When TCDD is administered bound to soil, absorption is also attenuated, suggesting that binding to soil constituents can lower bioavailability.[7] When administered bound to activated charcoal, no TCDD absorption could be measured, suggesting that the organic content of soil may be a limiting factor in determining absorption.[8]

Absorption of TCDD following inhalation has not been well studied and the literature contains no data on which to base estimates of absorption efficiency.[9] However, EPA assumed that absorption through the lungs would be efficient in extrapolating the oral slope factor for TCDD to the inhalation route.

Qualitative Description of Health Effects

Mechanisms of Action

Much current knowledge on the toxic effects of dioxins and furans comes from studies using the single potent dioxin congener, 2,3,7,8-TCDD. Generalization to other agents is assumed based on similar biochemical and molecular properties and on a few studies using other congeners.

The current consensus on mechanism of action for dioxins and furans is that selective binding to a high affinity receptor protein in the cytosol of mammalian cells is responsible for the exceptionally high toxicity and unusual spectrum of effects seen following exposure.

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2,3,7,8-TCDD binds selectively to a high affinity "receptor" protein in the cytosol of mammalian cells.[10, 11, 12] The TCDD-receptor complex is translocated to the nucleus of the cell where it binds to DNA and alters gene expression as indicated by increased mRNA synthesis. Receptor binding is associated with the induction of aryl hydrocarbon hydroxylase (AHH) and a variety of other enzymes. This induction has been demonstrated in a number of different tissues, but is particularly marked in the liver, kidney, thymus, and skin, which are important target organs for 2,3,7,8-TCDD toxicity. The affinity of the cytosolic receptor for 2,3,7,8-TCDD varies widely among and within species. At least in mice, this variability is genetically controlled and is associated with the Ah gene locus,[13] a locus that is also associated with induction of AHH. A number of researchers have recently determined that the sensitivity of experimental animals to many of the biological effects of 2,3,7,8-TCDD is associated with AHH inducibility and segregates with the Ah locus during cross-breeding experiments in mice.[14] These findings are important for risk assessment because they show genetic variability in susceptibility to biological effects of 2,3,7,8-TCDD. Especially wide variations in susceptibility could be expected in a genetically heterogenous species such as humans.

Acute Toxicity

The most frequently observed adverse health effect resulting from acute exposure to dioxins in humans is chloracne. TCDD is known to be one of the most potent compounds in producing chloracne; however, sufficient data on exposure are not available to define the dose necessary to produce this effect. Chloracne develops several days to months after exposure to dioxins and may persist for as long as 29 years after exposure.[15] Although chloracne has been reported in most or all cases of occupational exposure, in many cases only a portion of the workers subject to exposure developed chloracne, suggesting variability in susceptibility. It is believed that humans can develop chloracne following exposure to 2,3,7,8-TCDD by any route.

Effect on the immune system also appear to be associated with exposure to 2,3,7,8-TCDD. In a clinical study of 154 former residents of a mobile home park (Quail Run, Missouri) where the soil was contaminated with 2,3,7,8-TCDD, Knudsen, et al.[16] reported a significant reduction in delayed hypersensitivity responses to standard antigens among a subgroup of 51 residents, compared with 93 controls. Measures of T-cell functioning were also depressed, although not significantly, among the residents. These results suggest an association between impairment of the immune system and exposure to 2,3,7,8-TCDD. However, actual exposures were not documented or measured in this study.

A number of studies investigated reproductive outcomes in human populations exposed to 2,3,7,8-TCDD, but these studies are severely compromised by difficulties in documenting exposure, and in establishing rates of adverse reproductive outcomes in comparison populations. For example, Hanify, et al.[17] found a statistical association between incidence of birth defects (heart defects and talipes) and wide-area spraying of 2,4,5-T. Overall, however, the evidence for an association between exposure to 2,3,7,8-TCDD and adverse reproductive outcomes is inconclusive.

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Carcinogenicity

Several Swedish epidemiological studies have reported an association between occupational exposure to phenoxy acid herbicides or chlorophenol and increased incidence of certain cancers, including soft tissue sarcomas, non-Hodgkin's lymphomas, and nasopharyngeal cancers.[18, 19, 20] The presumptive link between these exposures and cancer is the presence of 2,3,7,8-TCDD or other dioxin isomers as impurities in phenoxy acids and chlorophenol.

A case-control study of similar design in New Zealand failed to demonstrate a significantly increased relative risk for soft tissue sarcoma among individuals exposed to phenoxy herbicides or chlorophenol.[21, 22] Lynge[23] reported excess incidences of soft-tissue sarcomas among Danish workers employed in the manufacture of phenoxy herbicides, but most of the herbicides involved were not contaminated with 2,3,7,8-TCDD. There have been several case reports of soft-tissue sarcomas among United States workers exposed to phenoxy acids and/or dioxins.[24, 25, 26, 27] However, Fingerhut, et al.[28] showed that some of these reports were based on erroneous pathological diagnoses. In a small but well-controlled study, Thiess, et al.[29] reported a significant excess of stomach cancers among worker presumptively exposed to 2,3,7,8-TCDD in a chemical reactor accident 23 years earlier. Other cancer studies have been inadequate to show either positive or negative results. Although some of these results suggest a possible association between exposure to 2,3,7,8-TCDD and increased risk of cancer, the evidence taken as a whole is inconclusive.

Several factors complicate the interpretation of the toxic effects of 2,3,7,8-TCDD, especially the extrapolation of animal data to predict likely effects in humans.

The studies regarding the toxicity of 2,3,7,8-TCDD in humans did not include adequate characterization of exposure. Many studies were of human populations exposed to phenoxy acids or chlorophenol, in which contamination with 2,3,7,8-TCDD is likely but was not verified or measured. In addition, quantitative characterization of exposure was not provided in any of the studies. Therefore, the human data are useful only for qualitative comparison with the animal data.

Another factor that complicates the extrapolation of the toxicity data of 2,3,7,8-TCDD in animals to humans is that the persistence of 2,3,7,8-TCDD in humans is not known. 2,3,7,8-TCDD is relatively persistent in the environment and in many living systems. It is concentrated in the fat and liver of most species following absorption. In rodents, the biological half-life of 2,3,7,8-TCDD ranges from 10 to

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43 days. However, McNulty, et al.[30] reported much longer persistence in the tissue of rhesus monkeys, probably greater than one year. Poiger and Schlatter[31] estimated that about 90 percent of the body burden of 2,3,7,8-TCDD in a single volunteer was sequestered in fat, and calculated a half-life of 2,120 days assuming first order kinetics. This data is consistent with the high bioconcentration potential of 2,3,7,8-TCDD as calculated by Geyer, et al.[32] Though there is little hard data on which to estimate persistence of dioxins and furans in humans, it seems reasonable from the above, to assume a relatively long half-life.

A final factor that complicates extrapolation of animal toxicity data to humans is that toxic responses to 2,3,7,8-TCDD vary widely among and within species. For example guinea pigs, rhesus monkeys, and chickens are extremely sensitive to the acute toxic effects of 2,3,7,8-TCDD, rats and mice are intermediate in sensitivity, and hamsters are relatively insensitive. The significance of intraspecies variability is that some individuals may be much more susceptible than others, requiring the use of large safety factors to protect the most sensitive individuals.

Quantitative Description of Health Effects

The oral slope factor for 2,3,7,8-TCDD is based on a feeding study in rats in which dose-dependent increase in tumors were seen at various sites depending on the sex of the animal.[33], [34] Extrapolation of these data using a linear multistage model results in a slope factor 1.5 x 105. Toxicologically, there seems little reason to believe that route of entry effects toxicologic outcome. Thus, the slope factor for inhalation exposure was assumed to be the same as for oral. However, the slope factors are under review by EPA.[35] EPA has approved a method to assess the carcinogenicity of other dioxin isomers by applying toxicity equivalency factors (TEFs) to these isomers.[36] These TEFs may be multiplied by the cancer slope factor for 2,3,7,8-TCDD to estimate the cancer slope factor for other dioxin isomers. These TEFs are listed in Table 1.

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Table 1

Toxicity Equivalency Factors for Chlorinated Dibenzo-p-dioxins and -Dibenzofurans[1]

Compound	TEF
Mono, Di, and TriCDDs	0
2,3,7,8-TCDD	1
Other TCDDs	0
2,3,7,8 – PeCDD	0.5
Other PeCDDs	0
2,3,7,8 – HxCDD	0.1
Other HxCDDs	0
2,3,7,8 – HpCDD	0.01
Other HpCDDs	0
OCDD	0.001
Mono, Di-, and TriCDFS	0
2,3,7,8 – TCDF	0.1
Other TCDFs	0
1,2,3,7,8 – PeCDF	0.05
2,3,4,7,8 – PeCDF	0.5
Other PeCDFs	0
2,3,7,8 – HxCDF	0.1
Other HxCDFs	0
2,3,7,8 – HpCDF	0.01
Other HpCDFs	0
OCDF	0.001

[1] EPA (U.S. Environmental Protection Agency). 1989. Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans (CDDs and CDFs) and 1989 Update. EPA/625/3-89/016.

No RfD or RfC have been established for TCDD. However, the proposed MCL (5×10^{-8} mg/L in EPA 1991)[37] suggests that an acceptable daily intake (ADI) might be 1×10^{-9} mg/kg-day assuming that a 70 kg human consumes 2L of contaminated water per day ([5×10^{-8} mg/L x 2L/day]/70 kg). This is consistent with the estimate for a chronic daily dose (1×10^{-9} mg/kg-day) associated with "minimal risk for effects other than cancer".[38] The ATSDR estimate is based on a three-generation study in rats exposed to 2,3,7,8-TCDD diets at doses of 0.001, 0.01, and 0.1 µg/kg-day.[39] The lowest dose resulted in renal effects, decreased fetal weight, and changes in the gestational index. The low dose, when adjusted by an uncertainty factor of 1,000, resulted in an "RfD" of 1×10^{-9} mg/kg-day^{-1}. For subchronic effects, ATSDR[40] suggests that a daily dose of 1 x 10-6 mg/kg-day is also associated with minimal risk for effects other than cancer provided the exposure period is 14 days or less.

[37] U.S. Environmental Protection Agency. Uptake/Biokinetic Model for Lead, Version 0.6. August, 1991.

[38] Agency for Toxic Substances and Disease Registry. Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. (DRAFT). 1987.

[39] Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A. Schwetz. Three-Generation Reproduction Study of Rats Given 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the Diet. Toxicol. Appl. Pharmacol. 50:241-251. 1979.

[40] Agency for Toxic Substances and Disease Registry. Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. (DRAFT). 1987.

Summary of Dioxins and Furans Criteria

Criterion	Value	Source
EPA carcinogen classification	B2	EPA 1992a
Oral slope factor	$1.5 \times 10^{+5}$ (mg/kg-day)$^{-1}$	EPA 1992b
Inhalation slope factor	$1.5 \times 10^{+5}$ (mg/kg-day)$^{-1}$	EPA 1992b
Maximum Contaminant Level (MCL)	3×10^{-7} (mg/L)	EPA 1992c
Maximum Contaminant Level Goal (MCLG)	Zero	EPA 1992c
Ambient Water Quality Criteria (AWQC) (10^{-4} to 10^{-7})	1.3×10^{-9} to 1.3×10^{-12} (mg/L)	EPA 1984

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FORMALDEHYDE

Introduction

Formaldehyde is a volatile organic compound that exists as a colorless and flammable gas at room temperature.[1] It is characterized by a pungent odor at concentrations above 0.83 parts per million (ppm).[2] Formaldehyde is produced both by natural and anthropogenic processes. It is a product of incomplete combustion and is also formed in the atmosphere by photochemical reactions.[3] Formaldehyde is released into the air by burning wood, coal, kerosene, and natural gas, by automobiles, and by cigarettes.[4,5] Additionally, vehicles powered by methanol emit formaldehyde.[6] Formaldehyde is also a naturally occurring body constituent used in the biosynthesis of purines, thymidine, and some amino acids.[7]

Formaldehyde is used in many applications. It is used as a bactericide, fungicide, and as an embalming fluid.[8] It is used in the wood products industry, primarily in adhesives for bonding pressed wood products such as plywood and particle board.[9] Formaldehyde is used in permanent press fabrics, in home insulation and as a stabilizer in gasoline.[10, 11] It is used as a preservative in some paints, coatings, and cosmetics and as a finish used to coat paper products.[12]

Potential for Human Exposure

Releases to the Environment

Formaldehyde is a product of incomplete combustion and is released into the air by burning wood, coal, kerosene, and natural gas, by automobiles, and by cigarettes; it is also a naturally occurring substance.[13], [14] Formaldehyde can be released to soil, water, and air by industrial sources; it has been detected in industrial emissions and in municipal and industrial aqueous effluents, including those resulting from chemical, oil, and coal processing.[15] Formaldehyde can off-gas from materials made with it.[16] Materials containing formaldehyde is more likely to off-gas in warm, humid weather.[17] Automobile

1 British Columbia Ministry of Environment, Lands, and Parks. Final Air Quality Objectives for Formaldehyde and the Interim Air Quality Objective for Fine Particulate: PM10. Air Resources Branch. Environmental Protection Department. Ministry of Environment, Lands, and Parks. January, 1995.

2 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

3 British Columbia Ministry of Environment, Lands, and Parks. Final Air Quality Objectives for Formaldehyde and the Interim Air Quality Objective for Fine Particulate: PM10. Air Resources Branch. Environmental Protection Department. Ministry of Environment, Lands, and Parks. January, 1995.

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10 British Columbia Ministry of Environment, Lands, and Parks. Final Air Quality Objectives for Formaldehyde and the Interim Air Quality Objective for Fine Particulate: PM10. Air Resources Branch. Environmental Protection Department. Ministry of Environment, Lands, and Parks. January, 1995.

11 National Toxicology Program. NTP Annual/Biennial Report on Carcinogens. 7th Annual Report on Carcinogens. 1994.

12 U.S. Consumer Product Safety Commission. An Update on Formaldehyde. October, 1990.

13 Budavari, S. (Editor). The Merck Index. An Encyclopedia of Chemicals, Drugs, and Biologicals. Eleventh Edition. Published by Merck & Co., Inc. Rahway, N.J., USA. 1989.

14 U.S. Consumer Product Safety Commission. An Update on Formaldehyde. October, 1990.

15 National Toxicology Program. NTP Annual/Biennial Report on Carcinogens. 7th Annual Report on Carcinogens. 1994.

16 U.S. Consumer Product Safety Commission. An Update on Formaldehyde. October, 1990.

exhaust is a major source of formaldehyde in ambient air. Interior air also contains formaldehyde; two subpopulations with particularly high potential for exposure to formaldehyde in indoor air are residents of mobile homes containing particle board and plywood and residents living in conventional homes insulated with urea-formaldehyde foam[18, 19]

The Toxics Release Inventory (TRI) listed 793 industrial facilities that produced, processed, or otherwise used formaldehyde in 1988.[20] These facilities reported formaldehyde releases to the environment, which were estimated to total 23.6 million pounds.[21] Not all industrials are required to report to TRI; therefore, estimates of formaldehyde releases should not be considered total releases.

Environmental Fate

Formaldehyde in air is degraded by photochemical processes and has a short half-life.[22] Formaldehyde vapors can react with hydrogen chloride, if present in the air, to form bis chloromethyl ether, a human carcinogen.[23] Formaldehyde is soluble but unstable in water; the half-life of formaldehyde in water is between 2 and 20 days.[24, 25] Due to formaldehyde's volatile nature, it is unlikely to be present in soil in significant concentrations. About 99% of formaldehyde in the environment will eventually end up in the air; the rest will be present in water.[26]

Environmental Levels

AIR: The highest levels of formaldehyde have been detected in indoor air, where it is released from various consumer products.[27] Formaldehyde concentrations in indoor air have been reported to range from 0.10 to 3.68 parts per million (ppm).[28] Formaldehyde has also been detected in ambient air; rural areas generally have lower formaldehyde air concentrations than urban areas.[29,30]

Formaldehyde concentrations in air were characterized for Pico Rivera, a Los Angeles suburb, for the summer of 1994 in the 1995 National Air Quality and Emissions Trends Report.[31] The mean concentration of formaldehyde in air in Pico Rivera in the summer of 1994 was 4.6 parts per billion (ppb) (494 observations). The maximum concentration of formaldehyde detected in air of Pico Rivera in the summer of 1994 was 64.5 ppb.

Water: Formaldehyde has been detected in rainwater, lake water, and some waterways.[32] It most likely occurs in natural waters from industrial discharges.[33] Concentrations were not reported, however, as described above, approximately 99% of formaldehyde in the environment is in air.

Soil and Sediment: Information describing formaldehyde concentrations in soils and sediments were not located; due to the volatile nature of this compound it is unlikely that significant concentrations will be present in soils or sediments.

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17 U.S. Consumer Product Safety Commission. An Update on Formaldehyde. October, 1990.

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19 U.S. Environmental Protection Agency. Formaldehyde. Office of Air Quality Planning and Standards. 1998.

20 National Toxicology Program. NTP Annual/Biennial Report on Carcinogens. 7th Annual Report on Carcinogens. 1994.

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23 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

24 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

25 National Toxicology Program. NTP Annual/Biennial Report on Carcinogens. 7th Annual Report on Carcinogens. 1994.

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30 U.S. Environmental Protection Agency. Formaldehyde. Office of Air Quality Planning and Standards. 1998.

31 California Environmental Protection Agency. California Environmental Protection Agency Criteria for Carcinogens. 1995.

32 National Toxicology Program. NTP Annual/Biennial Report on Carcinogens. 7th Annual Report on Carcinogens. 1994.

33 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

Other Environmental Media: Cigarette smoke is reported to contain 20 to 90 micrograms (:g) of formaldehyde per cigarette. Formaldehyde may be present in food, either naturally or as a result of contamination. Plants, such as kidney beans and barley, can absorb gaseous formaldehyde through their leaves. Maize leaves can form formaldehyde naturally during photosynthesis.[34]

Toxicokinetics

Information describing the kinetics of formaldehyde is limited. Toxicity studies indicate it is absorbed through inhalation and ingestion.[35] Studies performed by the U.S. Consumer Product Safety Commission (CPSC) did not indicate formaldehyde penetration of intact skin.[36] Information describing distribution following formaldehyde exposure was not located. Acetaldehyde, the closest aldehyde to formaldehyde in structure[37] was detected in the blood, liver, kidney, spleen, heart, and other muscle tissues following inhalation exposure.[38] Low levels of acetaldehyde were detected in embryos following maternal intraperitoneal injection (pregnant mice) and following maternal exposure to ethanol (pregnant mice and rats).[39] No information was located describing metabolism or excretion of formaldehyde. Physiological and biochemical processes linking formaldehyde exposure with adverse health effects are not completely understood.

Qualitative Description of Health Effects

Acute dermal exposure to either airborne or liquid formaldehyde may cause skin irritation, including a rash and burning feeling. It can also cause severe burns, leading to permanent damage. Burns may be delayed for hours after contact, even if no burn is felt initially. Contact with airborne formaldehyde may cause severe eye burns that also may be delayed for hours.[40] Acute inhalation exposure to formaldehyde concentrations above 0.1 ppm causes nose, mouth, and throat irritation.[41,42] The severity of the irritation increases with increasing concentration. Exposures to formaldehyde concentrations of 100 ppm can cause buildup of fluid in the lungs and spasm of the windpipe and can cause death.[43]

EPA has determined that formaldehyde is a B1 probable human carcinogen. Chronic dermal exposure to formaldehyde can cause skin sensitization; if skin becomes sensitized, very low future exposures can cause itching and rash.[44] Chronic inhalation exposure to formaldehyde can cause an asthma-like allergy; future exposures can cause asthma attacks with shortness of breath, wheezing, cough, and chest tightness.[45] Reproductive effects, such as menstrual disorders and problem pregnancies, have been reported in female workers exposed to formaldehyde.[46] Possible confounding factors were not evaluated in this study. A study of workers responsible for sterilizing hospital equipment did not report an association between formaldehyde exposure and increased spontaneous abortions.[47] Developmental effects have not been observed in animal studies with formaldehyde.[48]

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34 National Toxicology Program. NTP Annual/Biennial Report on Carcinogens. 7th Annual Report on Carcinogens. 1994.

35 U.S. Environmental Protection Agency. Integrated Risk Information System. 2000.

36 National Toxicology Program. NTP Annual/Biennial Report on Carcinogens. 7th Annual Report on Carcinogens. 1994.

37 U.S. Environmental Protection Agency. Integrated Risk Information System. 2000.

38 World Health Organization. Acetaldehyde. Report No. 167. 1995.

39 World Health Organization. Acetaldehyde. Report No. 167. 1995.

40 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

41 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

42 Occupational Safety and Health Administration. Fact Sheet Number OSHA 90-27. OSHA's Final Rule on Occupation Exposure to Formaldehyde. 1990.

43 Occupational Safety and Health Administration. Fact Sheet Number OSHA 90-27. OSHA's Final Rule on Occupation Exposure to Formaldehyde. 1990.

44 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

45 U.S. Environmental Protection Agency. Toxics Release Inventory (TRI) Facts. Formaldehyde. May, 1989.

46 U.S. Environmental Protection Agency. Formaldehyde. Office of Air Quality Planning and Standards. 1998.

47 U.S. Environmental Protection Agency. Formaldehyde. Office of Air Quality Planning and Standards. 1998.

48 U.S. Environmental Protection Agency. Formaldehyde. Office of Air Quality Planning and Standards. 1998.

In a study of health effects from off-gassing of formaldehyde in mobile homes in California, Liu et al.[49] observed burning, tearing eyes, stinging skin, fatigue, and sleeping problems in the summer months and burning, tearing eyes, chest pain, dizziness, sleeping problems, and sore throat in the winter months. Symptoms were more severe in smokers and people with chronic respiratory diseases or allergies. Formaldehyde levels ranged from non-detect (detection limit of 0.01 ppm) to 0.46 ppm.

Quantitative Description of Health Effects

EPA has classified formaldehyde as Group B1 - probable human carcinogen, based on limited evidence in humans and sufficient evidence in animals. EPA has developed an inhalation unit risk value of 1.3×10^{-5} (:g/m^3).[50] Human data include nine studies that show statistically significant associations between site-specific respiratory neoplasms and exposure to formaldehyde or products containing formaldehyde.[51] An increased incidence of nasal squamous cell carcinomas was observed in long-term inhalation studies in rats and mice. The classification is supported by in vitro genotoxicity data and formaldehyde's structural relationship to other carcinogenic aldehydes such as acetaldehyde.[52]

As described above, human carcinogenicity data are limited. At least 28 relevant epidemiologic studies have been conducted. Among these, three were well conducted and specifically designed to detect small to moderate increases in human health risks associated with formaldehyde exposure. Blair et al.[53,54] studies workers at 10 plants who were in some way exposed to formaldehyde and observed significant excesses in lung and nasopharyngeal cancer deaths. Despite a lack of significant trends with increasing concentration or cumulative exposure, lung cancer mortality was significantly elevated in analyses with or without a 20-year latency allowance. No explicit control was made for smoking status.

Stayner et al.[55] reported statistically significant excesses in deaths from buccal cavity tumors among garment workers exposed to formaldehyde. The highest standard mortality ratio was for workers with long employment duration and follow-up period (latency). Vaughan et al.[56,57] examined occupational and residential exposures, controlling for smoking and alcohol consumption. This study showed a significant association between nasopharyngeal cancer and having lived 10 or more years in a mobile home, especially for mobile homes built in the 1950's to 1970's, a period of increased formaldehyde-resin usage.

The remaining studies had limited ability to detect small to moderate increases in formaldehyde risks due to small sample size, small numbers of observed site-specific deaths, and insufficient follow-up. Even with these potential limitations, 6 of the remaining 25 studies reported significant associations between excess site-specific respiratory cancers and formaldehyde exposures.

Animal carcinogenicity data are sufficient to classify formaldehyde as a probable human carcinogen. The principal evidence comes from studies in both sexes of two strains of rats [58, 59, 60] and males of one strain

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[49] Liu, K., F. Huang, S. Hayword, J. Wesolowski, and K. Sexton. Irritant Effects of Formaldehyde Exposure in Mobile Homes. Environmental Health Perspectives. 94: 91-94. 1991.

[50] U.S. Environmental Protection Agency. Integrated Risk Information System. 2000.

[51] U.S. Environmental Protection Agency. Integrated Risk Information System. 2000.

[52] U.S. Environmental Protection Agency. Integrated Risk Information System. 2000.

[53] Blair, A., P.A. Stewart, R.N. Hoover, et al. Mortality Among Industrial Workers Exposed to Formaldehyde. J. Natl. Cancer Inst. 76(6):1071-1084. 1986.

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[59] Albert, R.E., A. Sellakumar, S. Laskin, M. Kuschner, N. Nelson, and C. Snyder. Gaseous Formaldehyde and Hydrogen Chloride Induction of Nasal Cancer in the Rat. J. Natl. Cancer Inst. 68(4): 597-603.

[60] Tobe, M., T. Kaneko, Y. Uchida, et al. Studies of the Inhalation Toxicity of Formaldehyde. National Sanitary and Medical Laboratory Service (Japan). p. 1-94. 1985.

of mice [61] all showing squamous cell carcinomas. The Kerns et al.[62] study was used to determine the inhalation unit risk value of 1.3×10^{5} ($\mu g/m^3$).

Kerns et al.[63] exposed about 120 animals per sex per species (Fischer 344 rats and B6C3F1 mice) to 0, 2, 5.6, or 14.3 ppm formaldehyde, 6 hours per day, 5 days per week for 24 months. Five animals per group were sacrificed at 6 and 12 months and 20 per group were sacrificed at 18 months. At 24 and 27 months the number sacrificed is unclear. The studies were terminated at 30 months. From the 12th month on, male and female rats in the highest dose group showed significantly increased mortality compared with controls. In the 5.6 ppm group, male rats showed a significant increase in mortality from 17 months on. Squamous cell carcinomas were seen in the nasal cavities of 51/117 male rats and 52/115 female rats at 14.3 ppm by experiments end. Squamous cell carcinomas of the nasal cavity were observed in 1/119 male rats and 1/116 female rats at 5.6 ppm. No such tumors were observed in the groups exposed to 0 or 2 ppm formaldehyde. Polypoid adenomas of the nasal mucosa were seen in rats at all doses in a significant dose-related trend, albeit one that falls off after a peak. Among the mice, squamous cell carcinomas were observed in two males at 14.3 ppm; no other lesions were noteworthy.

EPA has also developed an oral reference dose (RfD) for formaldehyde of 2×10^{-1} mg/kg-day, based on results of a rat 2-year bioassay.[64] Til et al.[65] administered formaldehyde to male and female Wistar rats (70 per sex per dose) in drinking water for up to 24 months at mean doses of 0, 1.2, 15, or 82 mg/kg-day for males and 0, 1.8, 21, or 109 mg/kg-day for females. Significant adverse effects, in the form of reduced weight gain and histopathology, were observed in the high-dose groups. A lowest-observed-adverse-effect-level (LOAEL) of 82 mg/kg-day was indicated by the study. A no-observed-adverse-effects-level (NOAEL) of 15 mg/kg-day was indicated by the study. EPA applied an uncertainty factor of 100 to the NOAEL to account for inter- and intraspecies differences. EPA places high confidence in the study used to develop the RfD, since it consisted of adequate numbers of animals of both sexes, as well as a thorough examination of toxicological and histological parameters. Confidence in the database is medium as several additional chronic bioassays and reproductive and developmental studies support the critical effect and study. Medium confidence in the RfD follows.[66]

The California Environmental Protection Agency (Cal EPA) has developed its own cancer potency factor for inhalation exposure to formaldehyde gas.[67] The Cal EPA cancer potency factor of 0.021 $(mg/kg\text{-}day)^{-1}$ was developed through evaluation of data provided in Swenberg et al.,[68] Kerns et al.,[69] EPA [70,71,72,] and Cal EPA.[73]

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61 Kerns, W.D., K. Pavkov, D. Donofrio, E. Gralla, and J. Swenberg. Carcinogenicity of Formaldehyde in Rats and Mice after Long-Term Inhalation Exposure. Cancer Res. 43: 4382-4392. 1983.

62 Kerns, W.D., K. Pavkov, D. Donofrio, E. Gralla, and J. Swenberg. Carcinogenicity of Formaldehyde in Rats and Mice after Long-Term Inhalation Exposure. Cancer Res. 43: 4382-4392. 1983.

63 Kerns, W.D., K. Pavkov, D. Donofrio, E. Gralla, and J. Swenberg. Carcinogenicity of Formaldehyde in Rats and Mice after Long-Term Inhalation Exposure. Cancer Res. 43: 4382-4392. 1983.

64 U.S. Environmental Protection Agency. Integrated Risk Information System. 2000.

65 Til, H., R. Woutersen, V. Feron, V. Hollanders, H. Falke, and J. Clary. Two-year Drinking Water Study of Formaldehyde in Rats. Food Chem. Toxicol. 27(2): 77-87. 1989.

66 U.S. Environmental Protection Agency. Integrated Risk Information System. 2000.

67 California Environmental Protection Agency. California Environmental Protection Agency Criteria for Carcinogens. 1995.

68 Swenberg, J.A., W. Kerns, R. Mitchell, E. Gralla, and K. Pavkov. Induction of Squamous Cell Carcinomas of the Rat Nasal Cavity by Inhalation Exposure to Formaldehyde Vapor. Cancer Res. 40: 3398-3402. 1980.

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70 U.S. Environmental Protection Agency. Qualitative and Quantitative Carcinogenic Risk Assessment for Formaldehyde. Office of Pesticides and Toxic Substances. Washington, D.C. EPA-450/5-87-003. 1987.

71 U.S. Environmental Protection Agency. Assessment of Health Risks to Garment Workers and Certain Home Workers from Exposure to Formaldehyde. Office of Pesticides and Toxic Substances. Washington, D.C. 1987.

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73 California Environmental Protection Agency. Final Report on the Identification of Formaldehyde as a Toxic Air Contaminant. Part B. Health Assessment. 1992.

The United States Occupational Safety and Health Administration (OSHA) has passed a final rule on occupational exposure to formaldehyde.[74] Under OSHA, the permissible exposure level (PEL) for formaldehyde in all workplaces is 1 ppm averaged over 8 hours, and the short-term exposure level (STEL) (i.e., exposure during any 15-minute period) is 2 ppm. An action level of 0.5 ppm, measured over 8 hours, was also set; if exposure is maintained below the STEL and the action level, exposure monitoring and certain employee training may be discontinued.

Currently there is no maximum contaminant level (MCL) for formaldehyde in drinking water. In addition, one-day, 10-day, and longer-term health advisories (HA) have been established as 10, 5, and 5 mg/L, respectively, for a 10 kg child. Longer-term and lifetime HAs have been set for adults at 20 and 1 mg/L, respectively.[75]

Summary of Formaldehyde Criteria

Criterion	Value	Source
EPA carcinogen classification	Group B1	EPA 2000
Oral Slope Factor	Not available	EPA 2000
Inhalation Slope Factor	1.3×10^{5} (:g/m^3)$^{-1}$	EPA 2000
Oral Reference Dose (RfD)	2×10^{-1} mg/kg-day	EPA 2000
Inhalation Reference Concentration (RfC)	Not available	EPA 2000
Cal EPA Inhalation Cancer Potency Factor	0.021 (mg/kg-day)$^{-1}$	Cal EPA 1995
Maximum Contaminant Level (MCL)	Not available	EPA 1996
EPA Drinking Water Health Advisories (HA)		
10 kg Child		
One-day HA	10 mg/L	EPA 1996
Ten-day HA	5 mg/L	EPA 1996
Longer-term HA	5 mg/L	EPA 1996
Adult		
Longer-term HA	20 mg/L	EPA 1996
Lifetime HA	1 mg/L	EPA 1996
Cal Permissible Exposure Limits, PEL	0.75 ppm	CCR, Title 8, 2000*
Cal Permissible Exposure Limits, STEL	2 ppm	CCR, Title 8, 2000*

* California Code of Regulations, Title 8, Section 5155, February 16, 2000.

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NAPHTHALENE

Introduction

Naphthalene is a solid substance of white color with the odor of mothballs or tar. It is predominantly used in the manufacture of dyes, resins, and mothballs.

Naphthalene is released into the environment predominantly through volatilization from mothballs and through residential and industrial burning of oil and coal. Minor releases into the environment are due to the wood preserving and coal tar producing industries.

Naphthalene is not persistent in the environment. It is rapidly broken down in the atmosphere by reaction with hydroxyl radicals and possibly by photolysis. In soils and aquatic systems, bacterial decomposition is the most important mode of degradation.

Toxicokinetics

Naphthalene is absorbed dermally, gastrointestinally, and through the lungs. No information is available regarding the extent of absorption or the distribution of naphthalene in the human body.

The metabolism of naphthalene is complex and many metabolites have been recognized. Key metabolites are 2-naphthoquinones, 1,2 naphthoquinones, and 3-glutathione adducts. These metabolites have been associated with hemolysis,[1] cataract formation,[2] and pulmonary toxicity,[3] respectively.

Data about the excretion of naphthalene are limited. The available information suggests that naphthalene and/or its metabolic products are excreted primarily in urine.

Qualitative Description of Health Effects

Most human exposures to naphthalene are associated with naphthalene-containing mothballs. Nausea, headache, abdominal pain, confusion, anemia, jaundice, renal disease, and cataract development are the most frequently observed ailments after exposure to naphthalene.

Several reports describe the symptoms of naphthalene exposure via inhalation in humans. Infants who had been exposed to naphthalene through contact with mothball-treated cloths and blankets developed jaundice and hemolytic anemia. Exposure was predominantly by inhalation, since direct contact with treated blankets and cloth did not occur.[4] Other symptoms in humans who had inhaled naphthalene vapor included vomiting and abdominal pain[5] and development of cataracts.[6]

Oral exposure to naphthalene has reportedly been fatal in some cases, and has been observed in individuals who ingested naphthalene-containing mothballs.[7,8] In one of these cases, 40 mothballs were ingested[9], but it is not known how much naphthalene was absorbed. An LD50 of 354 mg/kg of body weight was estimated for naphthalene ingestion in mice.[10]

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1 Mackell, J.V., F. Rieders, H. Bridgers, H., et al. Acute Hemolytic Anemia Due to Ingestion of Naphthalene Mothballs. *J. Clinical Aspects Pediatrics.* 7: 722-727. 1951.

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9 Kurz, J.M. Naphthalene Poisoning: Critical Care Nursing Techniques. *Dimens. Crit. Care Nurs.* 6:264-270. 1987.

10 Plasterer, M.R., W.S. Bradshaw, G.M. Booth, et al. Developmental Toxicity of Nine Selected Compounds Following Prenatal Exposure in the Mouse: Naphthalene p-Nitrophenol, Sodium Selemite, Dimethyl Phthalate, Ethylenethiouzea, and four Glycol

Oral exposure to naphthalene has frequently been associated with the development of hematolytic anemia [11, 12, 13] and with renal toxicity.[14] Observed neurological effects following ingestion of naphthalene included lethargy and convulsion,[15] and confusion.[16] Histologically separation of neural fibers and swelling of myelin sheaths were noted.[17] The development of cataracts has also been associated with oral exposure to naphthalene in humans[18] and in animals.[19, 20]

Data regarding symptoms due to dermal contact with naphthalene are limited. Two reports link dermal contact with naphthalene-containing diapers to the development of hemolytic anemia in infants.[21, 22]

Quantitative Description of Health Effects

Naphthalene has been classified as a Group D carcinogen. Chemicals in this category are not classifiable as to human carcinogenicity.

A reference dose of 8.6×10^{-4} mg/kg/day for inhalation exposure to naphthalene was presented in EPA's Integrated Risk Information System (IRIS) database. The principle study was performed by the National Toxicology Program (NTP)[23] in which B6C3F1 mice (75/sex/group) were exposed to naphthalene (scintillation grade, > 99% pure) at target concentrations of 0, 10, and 30 ppm (0, 52, 157 mg/m3) for 6 hr/day, 5 days/week, for 103 weeks.[24]

Survival of the male controls was significantly lower than in the exposed males. Reduced survival was related to wound trauma and lesions from increased fighting in this group. Similar effects were not seen in the exposed males, because they tended to huddle in cage corners during exposure periods and so fought less. There was no significant difference in survival between the treatment and control females. There were no treatment-related ocular lesions in the selected mice that underwent ophthalmologic examinations at 6-mo intervals. There were no biologically significant changes in hematology parameters at day 14 of the study. Final mean body weights of the treated animals were within 10% of the corresponding controls.

Inflammation, metaplasia of the olfactory epithelium, and hyperplasia of the respiratory epithelium were noted in the noses of virtually all exposed mice of both sexes, but in only one control female mouse. These effects were slightly more severe in the high-concentration group. See Table 1 for incidence data.

Ether Derivatives. *Toxicol Environ Health.* 15:25-38. 1985.

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[24] National Toxicology Program. Toxicology and Carcinogenesis Studies of Napthalene in B6C3F1 Mice (Inhalation Studies). Technical Report Series No. 410. NIH Publication No. 92-3141. 1992.

The lesions were focal or multifocal, occurred mainly in the posterior nasal cavity, and were minimal to mild in severity. Inflammatory lesions included substantia propria edema, congestion, mixed inflammatory cell infiltrates, necrotic debris, and intraluminal serous to fibrinopurulent exudate. Respiratory epithelial hyperplasia resulted in a thickened, folded, irregular mucosal surface. Olfactory epithelial metaplasia often involved ciliated columnar or pseudocolumnar respiratory-like epithelial cells replacing the usual olfactory cell layer. The lesions were collectively considered features of a generalized inflammatory and regenerative process.

A Reference Dose (RfD) for oral exposure to naphthalene was developed, based on the results of an animal study conducted by the National Toxicity Program (NTP). For the purposes of this study, Fischer 344 rats were orally exposed (by gavage) to 50 mg of naphthalene per kg of body weight per day for 13 weeks.[25] The critical effect upon which the RfD is based was decreased body weight gain. The chronic RfD was developed using the results of the study and an uncertainty factor of 10,000. The resulting oral RfD is 4 x 10-3 mg/kg-day.

Health Advisories (HAs) for naphthalene exposures are presented in EPA.[26] The one-day and 10-day HAs for children are both 0.5 mg/liter, and the longer term HA for children is 0.4 mg/liter. The longer term HA for adults is 1 mg/liter, and the lifetime HA is 0.02 mg/liter.

Summary of Naphthalene Criteria

Criterion	Value	Source
RfD (inhalation)	8.6×10^{-4} mg/kg/day	EPA 2000
Health Advisories		
Child one-day	0.5 mg/L	EPA 1996
Child 10-day	0.5 mg/L	EPA 1996
Child longer term	0.4 mg/L	EPA 1996
Adult longer term	1 mg/L	EPA 1996
Lifetime	0.2 mg/L	EPA 1996 – Office of Water
Cal Permissible Exposure Limits, PEL	50 mg/m^3	CCR, Title 8, 2000*
Cal Permissible Exposure Limits, STEL	75 mg/m^3	CCR, Title 8, 2000*

* California Code of Regulations, Title 8, Section 5155, February 16, 2000.

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POLYCYCLIC AROMATIC HYDROCARBONS

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a class of compounds consisting of two or more aromatic (benzene) rings. They form as a result of incomplete burning of organic compounds or by the partial breakdown of hydrocarbon compounds due to ultraviolet radiation. PAHs are commonly found as components of coal tar, soot, vehicle exhaust, creosote, refuse and wood burning emissions, and petroleum oils.[1] PAHs can occur naturally or as a result of human activity.

Over 100 different PAH compounds have been identified, but only a few have been adequately characterized toxicologically. Information in this profile has been summarized from the Agency for Toxic Substances and Disease Registry (ATSDR) profile on PAHs[2] and other sources, as indicated.

Potential for Human Exposure

Releases to the Environment

Most direct PAH releases to the environment are to air. PAHs are released from both man-made and natural sources. Natural sources include forest fires and volcanoes. Man-made sources contribute a much greater volume of PAHs to the environment than natural sources. Residential wood burning (i.e., stoves and fireplaces), industrial processes, and vehicle emissions are major man-made sources of PAHs. Composition of the PAH mixture released to the environment varies with the source; for example, emissions from vehicles contain a greater proportion of benzo(g,h,i)perylene and pyrene than other PAHs while emissions from residential wood burning contain a greater proportion of acenaphthylene. Additionally, vehicle emissions are low in benzo(a)pyrene (B(a)P) while emissions from burning of refuse are high in B(a)P.[3]

Vehicle emissions are a major contributor to PAHs in urban and suburban air. One study of PAH sources in city air pollution indicated that traffic contribution of PAHs to street air was 90% on workdays and 60% on weekends[4]. Traffic contribution to PAHs in city background air was estimated to be 40%. Nielsen et al[5] determined that PAH concentrations in air decreased in the order of street > city background air and suburbs > village > open land.

Sources of PAHs in surface water include deposition of airborne PAHs, direct industrial and municipal discharges, accidental oil spills, and urban storm water runoff. A study of organic pollutants in the coastal environment off San Diego, California, indicated that PAHs in the surface water and sediments of San Diego Bay were predominantly derived from combustion sources, such as boat and automobile exhaust.[6]

Deposition of airborne PAHs is believed to be the primary source of PAHs in soils, as evidenced by the presence of PAHs in soils distant from any industrial activity. Sludge disposals from sewage treatment plants, industrial discharges, and use of fertilizers are also potential sources of PAHs in soils.

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Environmental Fate

PAHs in air are present either in the gaseous phase or sorbed to particulates. Three-ring PAHs are found primarily in the gaseous phase, while five- and six-ring PAH compounds are present mainly sorbed to particulates, and four-ring PAHs may be found in either phase. PAHs in air may be carried over short or long distances and are removed by wet or dry deposition. Atmospheric residence time and transport distance depends on the size of the particles to which PAHs are sorbed. PAHs in the atmosphere can undergo photooxidation and can react with other atmospheric pollutants.

PAHs in surface water tend to volatilize, bind to particulates or sediments, or accumulate in aquatic biota. Microbial degradation, photooxidation, and chemical oxidation are also removal processes for PAHs in surface water. PAHs in sediments and soil can biodegrade or accumulate in receptors. PAHs with low molecular weight may volatilize from soil and sediments. PAHs can enter groundwater and be transported within an aquifer.

Environmental Levels

PAHs are found throughout the environment in air, water, and soil. PAHs seldom occur as single compounds in the environment; rather, they occur as complex mixtures of numerous compounds.[7] Standard EPA analytical methods test for the presence of only seventeen of the PAHs potentially occurring in environmental samples.[8] The following is a discussion of PAH concentrations in air, water, sediment, soil, and other environmental media.

Air: Data suggest that PAH concentrations in air are greater in urban areas than in rural areas. An ATSDR[9] summary of 1970 data from the U.S. National Air Surveillance Network indicated that B(a)P concentrations in 120 U.S. cities ranged from 0.2 to 19.3 nanograms per cubic meter (ng/m^3), while B(a)P concentrations in nonurban areas ranged from 0.1 to 1.2 ng/m^3. Studies also indicate that PAH concentrations in air are greater in the winter than in the summer. Seasonal variations were observed in a 1974-1975 study in Los Angeles; PAH concentrations in air ranged from 0.5 to 10.9 ng/m^3 in the winter (average of 2.09 ng/m^3) and from 0.1 to 3.7 ng/m^3 in the summer (average of 0.62 ng/m^3). A later study showed a similar pattern of seasonal variation; PAH concentrations in Los Angeles air (1981-1982) ranged from 0.4 to 4.5 ng/m^3 during the winter and from 0.1 to 1.5 ng/m^3 in the summer. Average concentrations in 1981-1982 were 1.26 and 0.43 ng/m^3 for winter and summer, respectively.

Individual PAH concentrations in Los Angeles air in 1974-1975 ranged from 0.18 ng/m^3 for benz(a)anthracene to 3.27 ng/m^3 for benzo(g,h,i)perylene. Median concentrations for most individual PAHs were less than 0.6 ng/m^3. High levels of automobile emissions probably contributed to the relatively high benzo(g,h,i)perylene concentrations.

Water: PAHs have been detected in surface waters throughout the United States. PAH concentrations in surface waters used as drinking water in four U.S. cities (Huntington, West Virginia; Buffalo, New York; and Pittsburgh and Philadelphia, Pennsylvania) ranged from 4.7 nanograms per liter (ng/L) in Buffalo to 600 ng/L in Pittsburgh. PAHs were detected in the Mississippi River at concentrations ranging from 1 ng/L for six compounds to 34 ng/L for phenanthrene. The highest concentration of phenanthrene was detected near an industrial area, implicating industrial effluent or surface water runoff from this area as a possible source. Surface water samples from San Diego Bay had total PAH concentrations of 42.2 ng/L (filtrates) and 1,440 ng/L (particulates)[10].

Soil and Sediment: PAHs have been detected in soils throughout the world. Benzo(g,h,i)perylene and fluoranthene have been detected at concentrations greater than 0.15 milligrams per kilogram (mg/kg) in arctic soils. Soil samples from remote wooded areas of Wyoming contained total PAH concentrations of up to 0.21 mg/kg. PAH concentrations are greater in urban soils. Total PAH concentrations of 4 to

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7 LaGoy, P.K. and T.C. Quirk. Establishing Generic Remediation Goals for the Polycyclic Aromatic Hydrocarbons: Critical Issues. Environmental Health Perspectives. 102(4): 348-352. 1994.

8 LaGoy, P.K. and T.C. Quirk. Establishing Generic Remediation Goals for the Polycyclic Aromatic Hydrocarbons: Critical Issues. Environmental Health Perspectives. 102(4): 348-352. 1994.

9 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

10 Zeng, E.Y. and C.L. Vista. Organic Pollutants in the Coastal Environment off San Diego, California. 1. Source Identification and Assessment by Compositional Indices of Polycyclic Aromatic Hydrocarbons. Environmental Toxicology and Chemistry, 16(2): 179-188. 1997.

8 mg/kg were found in soil near a complex road interchange in Switzerland. Bradley et al[11] collected surface soil samples from urban locations in three New England cities: Boston and Springfield, Massachusetts and Providence, Rhode Island. Total PAHs in urban soils ranged from 2.3 to 167 mg/kg for these cities.[12]

PAH concentrations in sediments are generally greater than those detected in surface water. PAH concentrations in sediments from Cape Cod and Buzzards Bay in Massachusetts and the Gulf of Maine have been reported to range from 0.54 to 1.3 mg/kg. Total PAH concentrations in bottom sediments from the main stem of Chesapeake Bay ranged from 0.045 to 8.92 mg/kg. Total PAH concentrations in sediments from San Diego Bay were 983 nanograms per gram (ng/g) (dry weight) in January 1994 and 898 ng/g (dry weight) in June 1994.[13]

Other Sources of PAHS: PAHs are found in crude oils and refined petroleum products, including gasoline, kerosene, diesel fuel, heating oils, and motor oil.

PAHs have been detected in unprocessed and processed foods. PAH concentrations in unprocessed foods depend on the source of the food. For example, vegetables and fruits obtained from an environment polluted with PAH may contain higher concentrations of PAHs than those obtained from nonpolluted environments.[14] PAH concentrations in food are influenced by the method of cooking (i.e., time of cooking, distance from heat source, and drainage of fat during cooking). In a composite sample of foods characterized as typical of the U.S. diet, PAH concentrations in all food groups were less than 2 parts per billion (ppb).[15]

Chewing tobacco, snuff, and mainstream and sidestream cigarette smoke contain PAHs. Smoking has been estimated to result in exposure to 0.4 micrograms (ug) of B(a)P per day.[16] Snuff has been reported to contain B(a)P concentrations ranging from 0.42 to 63 ppb.[17]

Toxicokinetics

PAHs are absorbed through the lungs, gastrointestinal tract, and skin. Absorption rates vary among the different compounds and are also affected by the type of material in which the PAH is carried (e.g., water, food, oil compounds). Absorption following inhalation exposure is also influenced by carrier particle size. Limited information indicates that PAHs absorbed from the lungs or gastrointestinal tract distributes primarily to soft tissues including the lungs, liver, kidney, and fatty tissue. There is little distribution of dermally absorbed PAHs.

Metabolism of PAHs occurs in all tissues. Enzymatic activity, however, varies among tissues and affects the degree of metabolism and bioavailability of PAHs. The primary method of metabolism is via oxidation by microsomal enzymes. PAHs are known enzyme inducers, that is, they cause enhanced enzymatic activity by increasing the rate of enzyme synthesis. Microsomal enzymes (mixed function oxidases) are responsible for the formation of epoxide metabolic intermediates that bind covalently to DNA. Some DNA adducts formed by binding to DNA cause mutations during DNA synthesis.

Excretion of PAHs following inhalation exposure is reportedly rapid. The larger portion is excreted in the feces following inhalation and oral exposure.

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[11] Bradley, L.J., B.H. Magee, and S.L. Allen. Background Levels of Polycyclic Aromatic Hydrocarbons (PAH) and Selected Metals in New England Urban Soils. Journal of Soil Contamination. 3(4):1-13. 1994.

[12] Bradley, L.J., B.H. Magee, and S.L. Allen. Background Levels of Polycyclic Aromatic Hydrocarbons (PAH) and Selected Metals in New England Urban Soils. Journal of Soil Contamination. 3(4):1-13. 1994.

[13] Zeng, E.Y. and C.L. Vista. Organic Pollutants in the Coastal Environment off San Diego, California. 1. Source Identification and Assessment by Compositional Indices of Polycyclic Aromatic Hydrocarbons. Environmental Toxicology and Chemistry, 16(2): 179-188. 1997.

[14] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

[15] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

[16] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

[17] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

Carcinogenic Health Effects

Qualitative Description of Carcinogenic Health Effects

Several PAHs, especially those with four or more benzene rings, have been established as complete carcinogens in animals, capable of tumor initiation, promotion, and progression.[18,19] Among the most potent and best studied of carcinogenic PAHs is B(a)P. A significant amount of knowledge of toxicologic actions of PAHs is based on extrapolation of animal studies with B(a)P to other carcinogenic members of the class. PAHs are carcinogenic in various species and by all routes of exposure. In most cases (e.g., after dermal exposures), tumors develop both at the site of contact and systemically.

Metabolism plays a critical role in carcinogenesis induced by PAHs. These compounds are activated to "ultimate" carcinogens, which can react directly with DNA, via mixed function oxidase enzymes in many tissues. Differences in metabolic capabilities probably are the basis for differences in sensitivity to carcinogenic effects of PAHs both among species and among organ systems.

Although PAHs are among the more potent animal carcinogens found in tobacco smoke, the presence of other carcinogenic and potentially carcinogenic chemicals, tumor promoters, initiators, and cocarcinogens in smoke makes it impossible to determine the quantitative association between PAH exposure and lung cancer in humans due to exposure to tobacco smoke. A similar argument can be made for other complex mixtures containing PAHs that have been associated with increased cancer incidence (e.g., soot, coal tar). Thus, data on human cancer are indirect and weak. On the basis of available toxicological information, EPA has classified seven PAHs as Group B2 carcinogens:[20]

- B(a)P
- Indeno(1,2,3-c, d)pyrene
- Dibenzo(a,h)anthracene
- Chrysene
- Benzo(k)fluoranthene
- Benzo(b)fluoranthene
- Benz(a)anthracene

The B2 classification indicates sufficient evidence for carcinogenesis in animals, but inadequate evidence in humans. These categorizations were found appropriate by EPAs Carcinogen Risk Assessment Verification Endeavor (CRAVE) workgroup. Slope factors verified by CRAVE have undergone extensive peer review and represent an Agency consensus. Files for these PAHs are available on EPAs electronic Integrated Risk Information System (IRIS) database.[21]

An oral cancer slope factor is available for B(a)P.[22] EPA has determined that available data for other carcinogenic PAHs are insufficient for the calculation of cancer slope factors. EPA has developed an estimated order of potential potencies for carcinogenic PAHs based on the cancer slope factor for B(a)P.[23]

Carcinogenesis assays using lower molecular weight PAHs have been generally negative, and many of the compounds have been classified into Group D - Not Classified (acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, phenanthrene, pyrene). However, several PAHs, notably, pyrene, act as cancer promoters or co-carcinogens in animal studies.

Quantitative Description of Carcinogenic Health

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[18] Nielsen, T., H.E. Jorgensen, J.C. Larsen, and M. Poulsen. City Air Pollution of Polycyclic Aromatic Hydrocarbons and Other Mutagens: Occurrence, Sources, and Health Effects. The Science of the Total Environment. 189/190:41-49. 1996.

[19] Tannheimer, S., S. Barton, S. Ethier, and S. Burchiel. Carcinogenic Polycyclic Aromatic Hydrocarbons Increase Intracellular Ca2+ and Cell Proliferation in Primary Human Mammary Epithelial Cells. Carcinogenesis. 18(6): 1177-1182. 1997.

[20] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[21] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[22] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[23] California Environmental Protection Agency. Benzo(a)pyrene as a Toxic Air Contaminant. Part B. Heath Effects of Benzo(a)pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA. 1993.

Effects

Although PAHs in general are well studied as carcinogens, EPA has determined that data suitable for development of a cancer slope factor are available only for B(a)P. Data for other carcinogenic PAHs are insufficient for calculating cancer slope factors for one or more of the following reasons:[24]

- Data were from exposures not typically used in deriving quantitative estimates for oral or inhalation exposure (e.g., skinpainting or subcutaneous exposure).
- Study populations were too small.
- Studies were done at only one exposure level.
- Dose-response data were not reported.

EPA has used cancer potency estimates for B(a)P as a "benchmark" to determine relative carcinogenic potential for other PAHs. Studies on the carcinogenicity of B(a)P are summarized in the following paragraph; these studies were presented in IRIS[25] as the basis for the oral cancer slope factor identified for B(a)P.

Neal and Rigdon[26] administered B(a)P in the diet at concentrations of 0, 1, 10, 20, 30, 40, 45, 50, 100, and 250 mg/kg to Swiss mice. Treatment time was variable up to a maximum of 197 days. Forestomach tumors were observed in mice receiving 20 mg/kg or more B(a)P. The authors indicated that tumor incidence increased related to concentration and number of doses administered. Brune, et al.[27] administered B(a)P to Sprague-Dawley rats by caffeine gavage resulting in annual doses of 6, 18, or 39 mg/kg. Untreated and gavage controls were included. There was a statistically significant association between dose and the proportions of rats with tumors of the forestomach, esophagus, or larynx. These data were used to derive an oral slope factor of 7.3 $(mg/kg\text{-}day)^{-1}$ based on the geometric mean from all four data sets (male and female rats and mice).[28] This slope factor has been verified by CRAVE and is presented in IRIS.

Hamsters were exposed to B(a)P at concentrations of 0, 2.2, 9.5, or 46.5 mg/m3 for over 60 weeks. Trend analysis showed a statistically significant tendency for the proportion of animals with respiratory tract and upper digestive tract tumors to increase steadily with increased dose. The inhalation slope factor was withdrawn from HEAST; the latest version of HEAST[29] does not present an inhalation slope factor for B(a)P or any other PAH.

The California Environmental Protection Agency (Cal EPA) has developed its own set of cancer potency factors for use in risk assessments required by regulatory programs in California. Cal EPA has developed cancer potency factors for inhalation and oral exposure to B(a)P based on data provided in Thyssen et al.,[30] EPA,[31] and Cal EPA.[32] Cal EPA has developed an oral cancer potency factor of 9 $(mg/kg\text{-}day)^{-1}$ for B(a)P.[33] For inhalation, Cal EPA has developed a cancer potency factor of 3.9 $(mg/kg\text{-}day)^{-1}$. CRAVE has not currently approved of an inhalation unit risk for B(a)P.

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[24] California Environmental Protection Agency. Benzo(a)pyrene as a Toxic Air Contaminant. Part B. Heath Effects of Benzo(a)pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA. 1993.

[25] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[26] Neal, J. and R.H. Rigdon. Gastric Tumors in Mice Fed Benzo(a)Pyrene ? A Qualitative Study. Tox. Rep. Biol. Med. 25:553-557. 1967.

[27] Brune, H., R.P. Deutch-Wenzel, M. Habs, S. Ivankovic, and D. Schmahl. Investigation of the Tumorigenic Response to Benzo(a)pyrene in Aqueous Caffeine Solution Applied Orally to Sprague-Dawley Rats. J. Cancer Res. Clin. Oncol. pp. 102, 153-157. 1982.

[28] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[29] U.S. Environmental Protection Agency. Health Effects Assessment Summary Tables (HEAST). Office of Solid Waste and Emergency Response. EPA/540/R-97/036. July, 1997.

[30] Thyssen, J., J. Althoff, G. Kimmerle, and U. Mohr. Inhalation Studies with Benzo(a)Pyrene in Syrian Golden Hamsters. J. Natl. Cancer Inst. 66:575-577. 1981.

[31] U.S. Environmental Protection Agency. Health Effects Assessment for Benzo[a]pyrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September. EPA 540/1-86-022. 1984.

[32] U.S. Environmental Protection Agency. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Office of Research and Development. EPA/600/R-93/089. July, 1993.

[33] Siegel, D. Personal communication with Dr. Dave Siegel, California Environmental Protection Agency, regarding change in Cal

No slope factors are available for dermal exposure to PAHs. Further, it may not be appropriate to extrapolate slope factors from oral exposure to the dermal route for two reasons. First, the skin is a major target organ for carcinogenic effects of PAHs following dermal exposure. B(a)P has been shown to cause skin tumors in mice, rats, rabbits, and guinea pigs following dermal application.[34] Increased incidences of distant site tumors have also been reported in animals as a consequence of dermal exposure to B(a)P. Route of entry effects compromise route-to-route extrapolation, and EPA[35] uses benzo(a)pyrene as an example of a chemical for which route of entry effects preclude the extrapolation of the oral slope factor to the dermal route.

Second, the skin is also a site of metabolism of PAHs. Even for chemicals absorbed into the blood stream, the form of the chemical, and hence its biological activity, may be altered. Dermal absorption is generally measured using radioactive compounds that do not provide an indication of the form of the chemical that reaches the blood stream. Thus, it is not appropriate to consider quantitatively risks for internal cancers based on absorption estimates from dermal exposure.

For the above reasons, quantitative evaluation of toxicity of PAHs following dermal exposure is very uncertain. However, Cal EPA suggests quantitative evaluation of dermal exposure to PAHs despite the above uncertainties. For such evaluations, the oral slope factor is used, with a correction applied to account for the differences in absorption of PAHs following oral and dermal exposure.

The Office of Health and Environmental Assessment (OHEA) of EPA has provided estimated orders of potential potency for Group B2 (probable human carcinogen) PAHs relative to B(a)P.[36] Mouse skin painting data sets were used to develop the comparative potencies on the basis that the data sets provide a complete set of comparisons. Comparative potencies developed by OHEA are shown in Table 1. These values are recommended by EPA for interim use; further research is underway.[37] Because of the differences in toxicokinetics following oral and dermal exposure, this method is uncertain. Relative potencies for different PAHs could vary by route of exposure.

Cal EPA has also developed relative potencies for PAHs, referred to as potency equivalency factors (PEFs), with B(a)P as the reference compound. PEFs have been developed by Cal EPA for all PAHs ranked by EPA as Group B2 carcinogens. Cal EPA has also developed PEFs for PAHs identified as Group 2A (probably carcinogenic to humans) and Group 2B (possibly carcinogenic to humans) by the International Agency for Research on Cancer (IARC). These consist of benzo(j)fluoranthene, dibenz(a,h)acridine, dibenz(a,j)acridine, 7H-dibenzo(c,g)carbazole, dibenzo(a,e)pyrene, dibenzo(a,h)pyrene, dibenzo(a,i)pyrene, dibenzo(a,l)pyrene, 7,12-dimethylbenz(a)anthracene, 1,6-dinitropyrene, 1,8-dinitropyrene, 5-methylchrysene, 6-nitrochrysene, 2-nitrofluorene, 1-nitropyrene, and 4-nitropyrene.

Cal EPA has developed an oral and inhalation cancer slope factor for dibenz(a,h)anthracene of 4.1 $(mg/kg\text{-}day)^{-1}$. This slope factor was developed by an expedited method and was based on a study by Snell and Steward[38] in which alveolar cell carcinomas were observed in male mice exposed orally to dibenz(a,h)anthracene in water for 60 weeks. Cal EPA also developed an oral and inhalation cancer slope factor for 7,12-dimethylbenz(a)anthracene of 250 $(mg/kg\text{-}day)^{-1}$. This slope factor was also developed by an expedited method, based on a study by Chouroulinkov et al.[39] in which tumors were observed in the intestines of female mice following exposure to 7,12-dimethylbenz(a)anthracene in the diet for 60 weeks. Cal EPA PEFs are shown in Table 1.

EPA B(a)P oral cancer potency factor. January 8, 1998.

[34] International Agency for Research on Cancer. Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic compounds. Monographs on the Evaluation of Carcinogenic Risk of the Chemical to Man, Vol. 3. Lyon, France. 1983.

[35] U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part A. Interim Final. OSWER Directive 9285.701A, Office of Solid Waste and Emergency Response. Washington, DC. EPA 540/1-89/002. 1989.

[36] U.S. Environmental Protection Agency. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Office of Research and Development. EPA/600/R-93/089. July, 1993.

[37] U.S. Environmental Protection Agency. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Office of Research and Development. EPA/600/R-93/089. July, 1993.

[38] Snell, K.C. and H.L. Stewart. Pulmonary adenomatosis induced in DBA/2 mice by oral administration of dibenz(a,h)anthracene. J. Nat. Cancer Inst. 28: 1043:1051. 1962.

[39] Chouroulinkov, I., A. Gentil, and M. Guerin. Etude de l?activite carcinogene du 9,12-dimethyl-benzanthracene et du 3,4-benzopyrene administres par voie digestive. Bull. Cancer. 54: 67-78. 1967.

Systemic Health Effects

Qualitative Description of Systemic Health Effects

Adverse systemic effects associated with PAH exposure have been observed in animals but generally not in humans. Exceptions include dermal effects, immunological effects, and gastrointestinal effects, although information is minimal. Skin disorders have been observed in humans following exposure to mixtures of carcinogenic PAHs; additionally, warts were observed following application of benzo(a)pyrene to human skin. An increased incidence of melanosis of the colon and rectum (unusual deposits of black pigments) was observed in humans consuming anthracene-containing laxatives for prolonged periods of time. However, no definitive conclusions can be drawn due to study limitations. Immunosuppression was observed in coke oven workers exposed chronically to complex mixtures of air pollutants composed primarily of PAHs.[40]

Chronic high doses of PAHs can produce toxicity in renal, hepatic, and hematologic systems of animals. Adverse reproductive and development effects have been observed in animals exposed to benzo(a)pyrene.

Quantitative Description of Systemic Health Effects

EPA has developed oral reference doses (RfDs) for several of the noncarcinogenic PAHs based on their potential to cause adverse systemic effects.[41] These RfDs and associated references are listed on Table 2.

[40] Szczeklik, A., J. Szczeklik, Z. Galuszka, J. Musial, et al. Humoral Immunosuppression in Men Exposed to Polycyclic Aromatic Hydrocarbons and Related Carcinogens in Polluted Environments. Environmental Health Perspectives. 102(3): 302-304. 1994.

[41] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

Table 1

Potential Potency Estimates for PAHs

Chemical	Cal EPA RelativePEF[1, 2]	Resulting Cal EPA Oral Potency Factor (mg/kg-day)$^{-1}$	Resulting Cal EPA Inhalation Potency Factor (mg/kg-day)$^{-1}$	EPA Classification	Relative Potency[2, 3] (OHEA)	Resulting OHEA Oral Slope Factor (mg/kg-day)$^{-1}$
Benz(a)anthracene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2[4]	0.1	7.3×10^{-1}
Benzo(b)fluoranthene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2	0.1	7.3×10^{-1}
Benzo(j)fluoranthene	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
Benzo(k)fluoranthene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2	0.01	7.3×10^{-2}
Benzo(a)pyrene	1.0	9.0[5]	3.9	B2	1.0	7.3
Chrysene	0.01	9.0×10^{-2}	3.9×10^{-2}	B2	0.001	7.3×10^{-3}
Dibenz(a,h)acridine	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
Dibenz(a,j)acridine	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
Dibenz(a,h)anthracene	NA	4.1	4.1	B2	1.0	7.3
7H-dibenzo(c,g)carbazole	1.0	9.0	3.9	na	na	na
Dibenzo(a,e)pyrene	1.0	9.0	3.9	na	na	na
Dibenzo(a,h)pyrene	10	90	39	na	na	na
Dibenzo(a,i)pyrene	10	90	39	na	na	na
Dibenzo(a,l)pyrene	10	90	39	na	na	na
7,12-Dimethylbenz(a)anthracene	NA	250	250	na	na	na
1,6-Dinitropyrene	10	90	39	na	na	na
1,8-Dinitropyrene	1.0	9.0	3.9	na	na	na
Indeno(1,2,3-cd)pyrene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2	0.1	7.3×10^{-1}
5-Methylchrysene	1.0	9.0	3.9	na	na	na
6-Nitrochrysene	10	90	39	na	na	na
2-Nitrofluorene	0.01	9.0×10^{-2}	3.9×10^{-2}	na	na	na
1-Nitropyrene	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
4-Nitropyrene	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na

PEF = Potency Equivalency Factor

1 Source: Cal EPA, 1994
2 Relative to B(a)P
3 Source: EPA 1993
4 Probable human carcinogen
5 D. Siegel, 1998
na = not available

Table 2

Oral RfDs for PAHs

Compound Status	Exposure	Species	Critical Effect	Uncertainty Factor	Modifying Factor	Reference Dose	Reference
Acenapthene/Verified (11/15/89	175 mg/kg-day daily by gavage for 90 days (NOAEL); 350 mg/kg-day (LOAEL)	Mouse	Hepatotoxicity	3,000	1	6×10^{-2} mg/kg-day	EPA 1989b as presented in EPA 1997a
Anthracene/Verified (11/15/89)	1,000 mg/kg-day daily by gavage for 90 days (NOEL) (HDT)	Mouse	No observed effects	3,000	1	3×10^{-1} mg/kg-day	EPA 1899c as presented in EPA 1997a
Fluoranthene/Verified (11/15/89)	125 mg/kg-day daily by gavage via corn oil for 13 weeks (NOAEL); 250 mg/kg-day (LOAEL)	Mouse	Nephropathy, increased relative liver weights, hematological and clinical effects	3,000	1	4×10^{-2} mg/kg-day	EPA 1988 as presented in EPA 1997a
Fluorene/Verified (11/15/89)	Gavage via corn oil 125 mg/kg-day for 13 weeks (NOAEL); 250 mg/kg-day (LOAEL)	Mouse	Decreased red blood cell, packed cell volume and hemoglobin	3,000	1	4×10^{-2} mg/kg-day	EPA 1989d as presented in EPA 1997a
Pyrene/Verified (11/15/89)	75 mg/kg-day by gavage via corn oil for 13 weeks (NOAEL); 125 mg/kg-day (LOAEL)	Mouse	Nephropathy and decreased kidney weight	3,000	1	3×10^{-2} mg/kg-day	EPA 1989e as presented in EPA 1997a

EPA 1997a. Integrated Risk Information System. Electronic Database.
NOAEL = No-observed-adverse-effects-leve
LOAEL = Lowest-observed-adverse-effects-level
NOEL = No-observed-effects-level
HDT = Highest Dose Tested

Summary of PAH Toxicity Criteria

Criterion	Value	Source
EPA Carcinogen Classification for: B(a)P, Indeno(1,2,3-c,d)pyrene, Dibenzo(a,h)anthracene, Chrysene Benzo(k)fluoranthene, Benzo(b)fluoranthene, and Benzo(a)anthracene	B2	EPA 1997a
EPA Oral Cancer Slope Factor (B(a)P)	$7.3 \times 10^{+0}$ (mg/kg-day)$^{-1}$	EPA 1997a
EPA Oral Reference Doses		
Acenapthene	6E-02 mg/kg-day	EPA 1997a
Anthracene	3E-01 mg/kg-day	EPA 1997a
Fluoranthene	4E-02 mg/kg-day	EPA 1997a
Fluorene	4E-02 mg/kg-day	EPA 1997a
Pyrene	3E-02 mg/kg-day	EPA 1997a
Cal EPA Oral Cancer Slope Factor (B(a)P)	9.0 (mg/kg-day)$^{-1}$	Cal EPA 1994
Cal EPA Inhalation Cancer Slope Factor (B(a)P)	3.9 (mg/kg-day)$^{-1}$	Cal EPA 1994

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POLYNUCLEAR AROMATIC HYDROCARBONS

Introduction

Polynuclear aromatic hydrocarbons (PNAs) (synonymous with polycyclic aromatic hydrocarbons (PAHs)) are a class of compounds consisting of two or more aromatic (benzene) rings. They form as a result of incomplete burning of organic compounds or by the partial breakdown of hydrocarbon compounds due to ultraviolet radiation. PNAs are commonly found as components of coal tar, soot, vehicle exhaust, creosote, refuse and wood burning emissions, and petroleum oils.[1] PNAs can occur naturally or as a result of human activity.

Over 100 different PNA compounds have been identified, but only a few have been adequately characterized toxicologically. Information in this profile has been summarized from the Agency for Toxic Substances and Disease Registry (ATSDR) profile on PAHs[2] and other sources, as indicated.

Potential for Human Exposure

Releases to the Environment

Most direct PNA releases to the environment are to air. PNAs are released from both man-made and natural sources. Natural sources include forest fires and volcanoes. Man-made sources contribute a much greater volume of PNAs to the environment than natural sources. Residential wood burning (i.e., stoves and fireplaces), industrial processes, and vehicle emissions are major man-made sources of PNAs. Composition of the PNA mixture released to the environment varies with the source; for example, emissions from vehicles contain a greater proportion of benzo(g,h,i)perylene and pyrene than other PNAs while emissions from residential wood burning contain a greater proportion of acenaphthylene. Additionally, vehicle emissions are low in benzo(a)pyrene (B(a)P) while emissions from burning of refuse are high in B(a)P.[3]

Vehicle emissions are a major contributor to PNAs in urban and suburban air. One study of PNA sources in city air pollution indicated that traffic contribution of PNAs to street air was 90% on workdays and 60% on weekends[4]. Traffic contribution to PNAs in city background air was estimated to be 40%. Nielsen et al[5] determined that PNA concentrations in air decreased in the order of street > city background air and suburbs > village > open land.

Sources of PNAs in surface water include deposition of airborne PNAs, direct industrial and municipal discharges, accidental oil spills, and urban storm water runoff. A study of organic pollutants in the coastal environment off San Diego, California, indicated that PNAs in the surface water and sediments of San Diego Bay were predominantly derived from combustion sources, such as boat and automobile exhaust.[6]

Deposition of airborne PNAs is believed to be the primary source of PNAs in soils, as evidenced by the presence of PNAs in soils distant from any industrial activity. Sludge disposal from sewage treatment plants, industrial discharges, and use of fertilizers is also potential sources of PNAs in soils.

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1 U.S. Environmental Protection Agency. Health Effects Assessment for Benzo[a]pyrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September. EPA 540/1-86-022. 1984.

2 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

3 Johnson, D. PAH: Ambient Air Quality Monitoring in British Columbia. British Columbia Environment, Lands, and Parks. Air Quality Unit, Environmental Impact Assessment Section. Environmental Protection Programme, Skeena Region. March, 1995.

4 Nielsen, T., H.E. Jorgensen, J.C. Larsen, and M. Poulsen. City Air Pollution of Polycyclic Aromatic Hydrocarbons and Other Mutagens: Occurrence, Sources, and Health Effects. The Science of the Total Environment. 189/190:41-49. 1996.

5 Nielsen, T., H.E. Jorgensen, J.C. Larsen, and M. Poulsen. City Air Pollution of Polycyclic Aromatic Hydrocarbons and Other Mutagens: Occurrence, Sources, and Health Effects. The Science of the Total Environment. 189/190:41-49. 1996.

6 Zeng, E.Y. and C.L. Vista. Organic Pollutants in the Coastal Environment off San Diego, California. 1. Source Identification and Assessment by Compositional Indices of Polycyclic Aromatic Hydrocarbons. Environmental Toxicology and Chemistry, 16(2): 179-188. 1997.

Environmental Fate

PNAs in air are present either in the gaseous phase or sorbed to particulates. Three-ring PNAs are found primarily in the gaseous phase, while five- and six-ring PNA compounds are present mainly sorbed to particulates, and four-ring PNAs may be found in either phase. PNAs in air may be carried over short or long distances and are removed by wet or dry deposition. Atmospheric residence time and transport distance depends on the size of the particles to which PNAs are sorbed. PNAs in the atmosphere can undergo photooxidation and can react with other atmospheric pollutants.

PNAs in surface water tend to volatilize, bind to particulates or sediments, or accumulate in aquatic biota. Microbial degradation, photooxidation, and chemical oxidation are also removal processes for PNAs in surface water. PNAs in sediments and soil can biodegrade or accumulate in receptors. PNAs with low molecular weight may volatilize from soil and sediments. PNAs can enter groundwater and be transported within an aquifer.

Environmental Levels

PNAs are found throughout the environment in air, water, and soil. PNAs seldom occur as single compounds in the environment; rather, they occur as complex mixtures of numerous compounds.[7] Standard EPA analytical methods test for the presence of only seventeen of the PNAs potentially occurring in environmental samples.[8] The following is a discussion of PNA concentrations in air, water, sediment, soil, and other environmental media.

Air: Data suggest that PNA concentrations in air are greater in urban areas than in rural areas. An ATSDR[9] summary of 1970 data from the U.S. National Air Surveillance Network indicated that B(a)P concentrations in 120 U.S. cities ranged from 0.2 to 19.3 nanograms per cubic meter (ng/m^3), while B(a)P concentrations in nonurban areas ranged from 0.1 to 1.2 ng/m^3. Studies also indicate that PNA concentrations in air are greater in the winter than in the summer. Seasonal variations were observed in a 1974-1975 study in Los Angeles; PNA concentrations in air ranged from 0.5 to 10.9 ng/m^3 in the winter (average of 2.09 ng/m^3) and from 0.1 to 3.7 ng/m^3 in the summer (average of 0.62 ng/m^3). A later study showed a similar pattern of seasonal variation; PNA concentrations in Los Angeles air (1981-1982) ranged from 0.4 to 4.5 ng/m^3 during the winter and from 0.1 to 1.5 ng/m^3 in the summer. Average concentrations in 1981-1982 were 1.26 and 0.43 ng/m^3 for winter and summer, respectively.

Individual PNA concentrations in Los Angeles air in 1974-1975 ranged from 0.18 ng/m^3 for benz(a)anthracene to 3.27 ng/m^3 for benzo(g,h,i)perylene. Median concentrations for most individual PNAs were less than 0.6 ng/m^3. High levels of automobile emissions probably contributed to the relatively high benzo(g,h,i)perylene concentrations.

Water: PNAs have been detected in surface waters throughout the United States. PNA concentrations in surface waters used as drinking water in four U.S. cities (Huntington, West Virginia; Buffalo, New York; and Pittsburgh and Philadelphia, Pennsylvania) ranged from 4.7 nanograms per liter (ng/L) in Buffalo to 600 ng/L in Pittsburgh. PNAs were detected in the Mississippi River at concentrations ranging from 1 ng/L for six compounds to 34 ng/L for phenanthrene. The highest concentration of phenanthrene was detected near an industrial area, implicating industrial effluent or surface water runoff from this area as a possible source. Surface water samples from San Diego Bay had total PNA concentrations of 42.2 ng/L (filtrates) and 1,440 ng/L (particulates)[10].

Soil and Sediment: PNAs have been detected in soils throughout the world. Benzo(g,h,i)perylene and fluoranthene have been detected at concentrations greater than 0.15 milligrams per kilogram (mg/kg) in arctic soils. Soil samples from remote wooded areas of Wyoming contained total PNA concentrations of up to 0.21 mg/kg. PNA concentrations are greater in urban soils. Total PNA concentrations of 4 to

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7 LaGoy, P.K. and T.C. Quirk. Establishing Generic Remediation Goals for the Polycyclic Aromatic Hydrocarbons: Critical Issues. Environmental Health Perspectives. 102(4): 348-352. 1994.

8 LaGoy, P.K. and T.C. Quirk. Establishing Generic Remediation Goals for the Polycyclic Aromatic Hydrocarbons: Critical Issues. Environmental Health Perspectives. 102(4): 348-352. 1994.

9 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

10 Zeng, E.Y. and C.L. Vista. Organic Pollutants in the Coastal Environment off San Diego, California. 1. Source Identification and Assessment by Compositional Indices of Polycyclic Aromatic Hydrocarbons. Environmental Toxicology and Chemistry, 16(2): 179-188. 1997.

8 mg/kg were found in soil near a complex road interchange in Switzerland. Bradley et al[11] collected surface soil samples from urban locations in three New England cities: Boston and Springfield, Massachusetts and Providence, Rhode Island. Total PNAs in urban soils ranged from 2.3 to 167 mg/kg for these cities.[12]

PNA concentrations in sediments are generally greater than those detected in surface water. PNA concentrations in sediments from Cape Cod and Buzzards Bay in Massachusetts and the Gulf of Maine have been reported to range from 0.54 to 1.3 mg/kg. Total PNA concentrations in bottom sediments from the main stem of Chesapeake Bay ranged from 0.045 to 8.92 mg/kg. Total PNA concentrations in sediments from San Diego Bay were 983 nanograms per gram (ng/g) (dry weight) in January 1994 and 898 ng/g (dry weight) in June 1994.[13]

Other Sources of PNAs: PNAs are found in crude oils and refined petroleum products, including gasoline, kerosene, diesel fuel, heating oils, and motor oil.

PNAs have been detected in unprocessed and processed foods. PNA concentrations in unprocessed foods depend on the source of the food. For example, vegetables and fruits obtained from an environment polluted with PNA may contain higher concentrations of PNAs than those obtained from nonpolluted environments.[14] PNA concentrations in food are influenced by the method of cooking (i.e., time of cooking, distance from heat source, and drainage of fat during cooking). In a composite sample of foods characterized as typical of the U.S. diet, PNA concentrations in all food groups were less than 2 parts per billion (ppb).[15]

Chewing tobacco, snuff, and mainstream and sidestream cigarette smoke contain PNAs. Smoking has been estimated to result in exposure to 0.4 micrograms (ug) of B(a)P per day.[16] Snuff has been reported to contain B(a)P concentrations ranging from 0.42 to 63 ppb.[17]

Toxicokinetics

PNAs are absorbed through the lungs, gastrointestinal tract, and skin. Absorption rates vary among the different compounds and are also affected by the type of material in which the PNA is carried (e.g., water, food, oil compounds). Absorption following inhalation exposure is also influenced by carrier particle size. Limited information indicates that PNAs absorbed from the lungs or gastrointestinal tract distributes primarily to soft tissues including the lungs, liver, kidney, and fatty tissue. There is little distribution of dermally absorbed PNAs.

Metabolism of PNAs occurs in all tissues. Enzymatic activity, however, varies among tissues and affects the degree of metabolism and bioavailability of PNAs. The primary method of metabolism is via oxidation by microsomal enzymes. PNAs are known enzyme inducers, that is, they cause enhanced enzymatic activity by increasing the rate of enzyme synthesis. Microsomal enzymes (mixed function oxidases) are responsible for the formation of epoxide metabolic intermediates that bind covalently to DNA. Some DNA adducts formed by binding to DNA cause mutations during DNA synthesis.

Excretion of PNAs following inhalation exposure is reportedly rapid. The larger portion is excreted in the feces following inhalation and oral exposure.

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[11] Bradley, L.J., B.H. Magee, and S.L. Allen. Background Levels of Polycyclic Aromatic Hydrocarbons (PAH) and Selected Metals in New England Urban Soils. Journal of Soil Contamination. 3(4):1-13. 1994.

[12] Bradley, L.J., B.H. Magee, and S.L. Allen. Background Levels of Polycyclic Aromatic Hydrocarbons (PAH) and Selected Metals in New England Urban Soils. Journal of Soil Contamination. 3(4):1-13. 1994.

[13] Zeng, E.Y. and C.L. Vista. Organic Pollutants in the Coastal Environment off San Diego, California. 1. Source Identification and Assessment by Compositional Indices of Polycyclic Aromatic Hydrocarbons. Environmental Toxicology and Chemistry, 16(2): 179-188. 1997.

[14] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

[15] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

[16] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

[17] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft. Prepared by Clement International Corporation for ATSDR. October, 1993.

Carcinogenic Health Effects

Qualitative Description of Carcinogenic Health Effects

Several PNAs, especially those with four or more benzene rings, have been established as complete carcinogens in animals, capable of tumor initiation, promotion, and progression.[18,19] Among the most potent and best studied of carcinogenic PNAs is B(a)P. A significant amount of knowledge of toxicologic actions of PNAs is based on extrapolation of animal studies with B(a)P to other carcinogenic members of the class. PNAs are carcinogenic in various species and by all routes of exposure. In most cases (e.g., after dermal exposures), tumors develop both at the site of contact and systemically.

Metabolism plays a critical role in carcinogenesis induced by PNAs. These compounds are activated to "ultimate" carcinogens, which can react directly with DNA, via mixed function oxidase enzymes in many tissues. Differences in metabolic capabilities probably are the basis for differences in sensitivity to carcinogenic effects of PNAs both among species and among organ systems.

Although PNAs are among the more potent animal carcinogens found in tobacco smoke, the presence of other carcinogenic and potentially carcinogenic chemicals, tumor promoters, initiators, and cocarcinogens in smoke makes it impossible to determine the quantitative association between PNA exposure and lung cancer in humans due to exposure to tobacco smoke. A similar argument can be made for other complex mixtures containing PNAs that have been associated with increased cancer incidence (e.g., soot, coal tar). Thus, data on human cancer are indirect and weak. On the basis of available toxicological information, EPA has classified seven PNAs as Group B2 carcinogens:[20]

- B(a)P
- Indeno(1,2,3-c, d)pyrene
- Dibenzo(a,h)anthracene
- Chrysene
- Benzo(k)fluoranthene
- Benzo(b)fluoranthene
- Benz(a)anthracene

The B2 classification indicates sufficient evidence for carcinogenesis in animals, but inadequate evidence in humans. These categorizations were found appropriate by EPAs Carcinogen Risk Assessment Verification Endeavor (CRAVE) workgroup. Slope factors verified by CRAVE have undergone extensive peer review and represent an Agency consensus. Files for these PNAs are available on EPAs electronic Integrated Risk Information System (IRIS) database.[21]

An oral cancer slope factor is available for B(a)P.[22] EPA has determined that available data for other carcinogenic PNAs are insufficient for the calculation of cancer slope factors. EPA has developed an estimated order of potential potencies for carcinogenic PNAs based on the cancer slope factor for B(a)P.[23]

Carcinogenesis assays using lower molecular weight PNAs have been generally negative, and many of the compounds have been classified into Group D - Not Classified (acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, phenanthrene, pyrene). However, several PNAs, notably, pyrene, act as cancer promoters or co-carcinogens in animal studies.

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[18] Nielsen, T., H.E. Jorgensen, J.C. Larsen, and M. Poulsen. City Air Pollution of Polycyclic Aromatic Hydrocarbons and Other Mutagens: Occurrence, Sources, and Health Effects. The Science of the Total Environment. 189/190:41-49. 1996.

[19] Tannheimer, S., S. Barton, S. Ethier, and S. Burchiel. Carcinogenic Polycyclic Aromatic Hydrocarbons Increase Intracellular Ca2+ and Cell Proliferation in Primary Human Mammary Epithelial Cells. Carcinogenesis. 18(6): 1177-1182. 1997.

[20] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[21] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[22] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[23] California Environmental Protection Agency. Benzo(a)pyrene as a Toxic Air Contaminant. Part B. Heath Effects of Benzo(a)pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA. 1993.

Quantitative Description of Carcinogenic Health Effects

Although PNAs in general are well studied as carcinogens, EPA has determined that data suitable for development of a cancer slope factor are available only for B(a)P. Data for other carcinogenic PNAs are insufficient for calculating cancer slope factors for one or more of the following reasons:[24]

- Data were from exposures not typically used in deriving quantitative estimates for oral or inhalation exposure (e.g., skinpainting or subcutaneous exposure).
- Study populations were too small.
- Studies were done at only one exposure level.
- Dose-response data were not reported.

EPA has used cancer potency estimates for B(a)P as a "benchmark" to determine relative carcinogenic potential for other PNAs. Studies on the carcinogenicity of B(a)P are summarized in the following paragraph; these studies were presented in IRIS[25] as the basis for the oral cancer slope factor identified for B(a)P.

Neal and Rigdon[26] administered B(a)P in the diet at concentrations of 0, 1, 10, 20, 30, 40, 45, 50, 100, and 250 mg/kg to Swiss mice. Treatment time was variable up to a maximum of 197 days. Forestomach tumors were observed in mice receiving 20 mg/kg or more B(a)P. The authors indicated that tumor incidence increased related to concentration and number of doses administered. Brune, et al.[27] administered B(a)P to Sprague-Dawley rats by caffeine gavage resulting in annual doses of 6, 18, or 39 mg/kg. Untreated and gavage controls were included. There was a statistically significant association between dose and the proportions of rats with tumors of the forestomach, esophagus, or larynx. These data were used to derive an oral slope factor of 7.3 $(mg/kg\text{-}day)^{-1}$ based on the geometric mean from all four data sets (male and female rats and mice).[28] This slope factor has been verified by CRAVE and is presented in IRIS.

Hamsters were exposed to B(a)P at concentrations of 0, 2.2, 9.5, or 46.5 mg/m3 for over 60 weeks. Trend analysis showed a statistically significant tendency for the proportion of animals with respiratory tract and upper digestive tract tumors to increase steadily with increased dose. The inhalation slope factor was withdrawn from HEAST; the latest version of HEAST[29] does not present an inhalation slope factor for B(a)P or any other PNA.

The California Environmental Protection Agency (Cal EPA) has developed its own set of cancer potency factors for use in risk assessments required by regulatory programs in California. Cal EPA has developed cancer potency factors for inhalation and oral exposure to B(a)P based on data provided in Thyssen et al.,[30] EPA,[31] and Cal EPA.[32] Cal EPA has developed an oral cancer potency factor of

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[24] California Environmental Protection Agency. Benzo(a)pyrene as a Toxic Air Contaminant. Part B. Heath Effects of Benzo(a)pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA. 1993.

[25] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[26] Neal, J. and R.H. Rigdon. Gastric Tumors in Mice Fed Benzo(a)Pyrene? A Qualitative Study. Tox. Rep. Biol. Med. 25:553-557. 1967.

[27] Brune, H., R.P. Deutch-Wenzel, M. Habs, S. Ivankovic, and D. Schmahl. Investigation of the Tumorigenic Response to Benzo(a)pyrene in Aqueous Caffeine Solution Applied Orally to Sprague-Dawley Rats. J. Cancer Res. Clin. Oncol. pp. 102, 153-157. 1982.

[28] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

[29] U.S. Environmental Protection Agency. Health Effects Assessment Summary Tables (HEAST). Office of Solid Waste and Emergency Response. EPA/540/R-97/036. July, 1997.

[30] Thyssen, J., J. Althoff, G. Kimmerle, and U. Mohr. Inhalation Studies with Benzo(a)Pyrene in Syrian Golden Hamsters. J. Natl. Cancer Inst. 66:575-577. 1981.

[31] U.S. Environmental Protection Agency. Health Effects Assessment for Benzo[a]pyrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September. EPA 540/1-86-022. 1984.

[32] U.S. Environmental Protection Agency. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Office of Research and Development. EPA/600/R-93/089. July, 1993.

9 $(mg/kg\text{-}day)^{-1}$ for B(a)P.[33] For inhalation, Cal EPA has developed a cancer potency factor of 3.9 $(mg/kg\text{-}day)^{-1}$. CRAVE has not currently approved of an inhalation unit risk for B(a)P.

No slope factors are available for dermal exposure to PNAs. Further, it may not be appropriate to extrapolate slope factors from oral exposure to the dermal route for two reasons. First, the skin is a major target organ for carcinogenic effects of PNAs following dermal exposure. B(a)P has been shown to cause skin tumors in mice, rats, rabbits, and guinea pigs following dermal application.[34] Increased incidences of distant site tumors have also been reported in animals as a consequence of dermal exposure to B(a)P. Route of entry effects compromise route-to-route extrapolation, and EPA[35] uses benzo(a)pyrene as an example of a chemical for which route of entry effects preclude the extrapolation of the oral slope factor to the dermal route.

Second, the skin is also a site of metabolism of PNAs. Even for chemicals absorbed into the blood stream, the form of the chemical, and hence its biological activity, may be altered. Dermal absorption is generally measured using radioactive compounds, which do not provide an indication of the form of the chemical, which reaches the blood stream. Thus, it is not appropriate to consider quantitatively risks for internal cancers based on absorption estimates from dermal exposure.

For the above reasons, quantitative evaluation of toxicity of PNAs following dermal exposure is very uncertain. However, Cal EPA suggests quantitative evaluation of dermal exposure to PNAs despite the above uncertainties. For such evaluations, the oral slope factor is used, with a correction applied to account for the differences in absorption of PNAs following oral and dermal exposure.

The Office of Health and Environmental Assessment (OHEA) of EPA has provided estimated orders of potential potency for Group B2 (probable human carcinogen) PNAs relative to B(a)P.[36] Mouse skin painting data sets were used to develop the comparative potencies on the basis that the data sets provide a complete set of comparisons. Comparative potencies developed by OHEA are shown in Table 1. EPA recommends these values for interim use; further research is underway.[37] Because of the differences in toxicokinetics following oral and dermal exposure, this method is uncertain. Relative potencies for different PNAs could vary by route of exposure.

Cal EPA has also developed relative potencies for PNAs, referred to as potency equivalency factors (PEFs), with B(a)P as the reference compound. CAL EPA has developed PEFs for all PNAs ranked by EPA as Group B2 carcinogens. Cal EPA has also developed PEFs for PNAs identified as Group 2A (probably carcinogenic to humans) and Group 2B (possibly carcinogenic to humans) by the International Agency for Research on Cancer (IARC). These consist of benzo(j)fluoranthene, dibenz(a,h)acridine, dibenz(a,j)acridine, 7H-dibenzo(c,g)carbazole, dibenzo(a,e)pyrene, dibenzo(a,h)pyrene, dibenzo(a,i)pyrene, dibenzo(a,l)pyrene, 7,12-dimethylbenz(a)anthracene, 1,6-dinitropyrene, 1,8-dinitropyrene, 5-methylchrysene, 6-nitrochrysene, 2-nitrofluorene, 1-nitropyrene, and 4-nitropyrene.

Cal EPA has developed an oral and inhalation cancer slope factor for dibenz(a,h)anthracene of 4.1 $(mg/kg\text{-}day)^{-1}$. This slope factor was developed by an expedited method and was based on a study by Snell and Steward[38] in which alveolar cell carcinomas were observed in male mice exposed orally to dibenz(a,h)anthracene in water for 60 weeks. Cal EPA also developed an oral and inhalation cancer slope factor for 7,12-dimethylbenz(a)anthracene of 250 $(mg/kg\text{-}day)^{-1}$. This slope factor was also developed by an expedited method, based on a study by Chouroulinkov et al.[39] in which tumors were

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[33] Siegel, D. Personal communication with Dr. Dave Siegel, California Environmental Protection Agency, regarding change in Cal EPA B(a)P oral cancer potency factor. January 8, 1998.

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[37] U.S. Environmental Protection Agency. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Office of Research and Development. EPA/600/R-93/089. July, 1993.

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[39] Chouroulinkov, I., A. Gentil, and M. Guerin. Etude de l?activite carcinogene du 9,12-dimethyl-benzanthracene et du 3,4-benzopyrene administres par voie digestive. Bull. Cancer. 54: 67-78. 1967.

observed in the intestines of female mice following exposure to 7,12-dimethylbenz(a)anthracene in the diet for 60 weeks. Cal EPA PEFs are shown in Table 1.

Systemic Health Effects

Qualitative Description of Systemic Health Effects

Adverse systemic effects associated with PNA exposure have been observed in animals but generally not in humans. Exceptions include dermal effects, immunological effects, and gastrointestinal effects, although information is minimal. Skin disorders have been observed in humans following exposure to mixtures of carcinogenic PNAs; additionally, warts were observed following application of benzo(a)pyrene to human skin. An increased incidence of melanosis of the colon and rectum (unusual deposits of black pigments) was observed in humans consuming anthracene-containing laxatives for prolonged periods of time. However, no definitive conclusions can be drawn due to study limitations. Immunosuppression was observed in coke oven workers exposed chronically to complex mixtures of air pollutants composed primarily of PNAs.[40]

Chronic high doses of PNAs can produce toxicity in renal, hepatic, and hematologic systems of animals. Adverse reproductive and development effects have been observed in animals exposed to benzo(a)pyrene.

Quantitative Description of Systemic Health Effects

EPA has developed oral reference doses (RfDs) for several of the noncarcinogenic PNAs based on their potential to cause adverse systemic effects.[41] These RfDs and associated references are listed on Table 2.

[40] Szczeklik, A., J. Szczeklik, Z. Galuszka, J. Musial, et al. Humoral Immunosuppression in Men Exposed to Polycyclic Aromatic Hydrocarbons and Related Carcinogens in Polluted Environments. Environmental Health Perspectives. 102(3): 302-304. 1994.

[41] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). On-line database. 2000.

Table 1

Potential Potency Estimates for PNA**s**

Chemical	Cal EPA RelativePEF[1, 2]	Resulting Cal EPA Oral Potency Factor (mg/kg-day)$^{-1}$	Resulting Cal EPA Inhalation Potency Factor (mg/kg-day)$^{-1}$	EPA Classification	Relative Potency[2, 3] (OHEA)	Resulting OHEA Oral Slope Factor (mg/kg-day)$^{-1}$
Benz(a)anthracene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2[4]	0.1	7.3×10^{-1}
Benzo(b)fluoranthene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2	0.1	7.3×10^{-1}
Benzo(j)fluoranthene	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
Benzo(k)fluoranthene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2	0.01	7.3×10^{-2}
Benzo(a)pyrene	1.0	9.0[5]	3.9	B2	1.0	7.3
Chrysene	0.01	9.0×10^{-2}	3.9×10^{-2}	B2	0.001	7.3×10^{-3}
Dibenz(a,h)acridine	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
Dibenz(a,j)acridine	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
Dibenz(a,h)anthracene	NA	4.1	4.1	B2	1.0	7.3
7H-dibenzo(c,g)carbazole	1.0	9.0	3.9	na	na	na
Dibenzo(a,e)pyrene	1.0	9.0	3.9	na	na	na
Dibenzo(a,h)pyrene	10	90	39	na	na	na
Dibenzo(a,i)pyrene	10	90	39	na	na	na
Dibenzo(a,l)pyrene	10	90	39	na	na	na
7,12-Dimethylbenz(a)anthracene	NA	250	250	na	na	na
1,6-Dinitropyrene	10	90	39	na	na	na
1,8-Dinitropyrene	1.0	9.0	3.9	na	na	na
Indeno(1,2,3-cd)pyrene	0.1	9.0×10^{-1}	3.9×10^{-1}	B2	0.1	7.3×10^{-1}
5-Methylchrysene	1.0	9.0	3.9	na	na	na
6-Nitrochrysene	10	90	39	na	na	na
2-Nitrofluorene	0.01	9.0×10^{-2}	3.9×10^{-2}	na	na	na
1-Nitropyrene	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na
4-Nitropyrene	0.1	9.0×10^{-1}	3.9×10^{-1}	na	na	na

PEF = Potency Equivalency Factor

1 Source: Cal EPA, 1994
2 Relative to B(a)P
3 Source: EPA 1993
4 Probable human carcinogen
5 D. Siegel, 1998
na = not available

Table 2

Oral RfDs for PNAs

Compound Status	Exposure	Species	Critical Effect	Uncertainty Factor	Modifying Factor	Reference Dose	Reference
Acenapthene/Verified (11/15/89	175 mg/kg-day daily by gavage for 90 days (NOAEL); 350 mg/kg-day (LOAEL)	Mouse	Hepatotoxicity	3,000	1	6×10^{-2} mg/kg-day	EPA 1989b as presented in EPA 1997a
Anthracene/Verified (11/15/89)	1,000 mg/kg-day daily by gavage for 90 days (NOEL) (HDT)	Mouse	No observed effects	3,000	1	3×10^{-1} mg/kg-day	EPA 1899c as presented in EPA 1997a
Fluoranthene/Verified (11/15/89)	125 mg/kg-day daily by gavage via corn oil for 13 weeks (NOAEL); 250 mg/kg-day (LOAEL)	Mouse	Nephropathy, increased relative liver weights, hematological and clinical effects	3,000	1	4×10^{-2} mg/kg-day	EPA 1988 as presented in EPA 1997a
Fluorene/Verified (11/15/89)	Gavage via corn oil 125 mg/kg-day for 13 weeks (NOAEL); 250 mg/kg-day (LOAEL)	Mouse	Decreased red blood cell, packed cell volume and hemoglobin	3,000	1	4×10^{-2} mg/kg-day	EPA 1989d as presented in EPA 1997a
Pyrene/Verified (11/15/89)	75 mg/kg-day by gavage via corn oil for 13 weeks (NOAEL); 125 mg/kg-day (LOAEL)	Mouse	Nephropathy and decreased kidney weight	3,000	1	3×10^{-2} mg/kg-day	EPA 1989e as presented in EPA 1997a

EPA 1997a. Integrated Risk Information System. Electronic Database.
NOAEL = No-observed-adverse-effects-leve
LOAEL = Lowest-observed-adverse-effects-level
NOEL = No-observed-effects-level
HDT = Highest Dose Tested

Summary of PNA Toxicity Criteria

Criterion	Value	Source
EPA Carcinogen Classification for: B(a)P, Indeno(1,2,3-c,d)pyrene, Dibenzo(a,h)anthracene, Chrysene Benzo(k)fluoranthene, Benzo(b)fluoranthene, and Benzo(a)anthracene	B2	EPA 1997a
EPA Oral Cancer Slope Factor (B(a)P)	$7.3 \times 10^{+0}$ (mg/kg-day)$^{-1}$	EPA 1997a
EPA Oral Reference Doses		
Acenapthene	6E-02 mg/kg-day	EPA 1997a
Anthracene	3E-01 mg/kg-day	EPA 1997a
Fluoranthene	4E-02 mg/kg-day	EPA 1997a
Fluorene	4E-02 mg/kg-day	EPA 1997a
Pyrene	3E-02 mg/kg-day	EPA 1997a
Cal EPA Oral Cancer Slope Factor (B(a)P)	9.0 (mg/kg-day)$^{-1}$	Cal EPA 1994
Cal EPA Inhalation Cancer Slope Factor (B(a)P)	3.9 (mg/kg-day)$^{-1}$	Cal EPA 1994

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XYLENES

Introduction

Xylenes (*ortho, para,* and *meta* isomers) are used as solvents for paints, inks, and adhesives and as components of detergents and other industrial and household products. The three xylene isomers have very similar but not identical toxicologic properties. These three compounds generally have similar chemical and biological characteristics and therefore will be discussed together.

Toxicokinetics

Although the available data are limited, inference from metabolism and excretion studies suggests that absorption of orally administered xylenes is nearly complete. Data from animals and humans suggest that approximately 60 percent of an inhaled dose is absorbed following ingestion. Dermal absorption is reported to be minor following exposure to xylene vapors but may be significant following contact with the liquid.[1] Elimination of xylenes is through urinary excretion of metabolites and through pulmonary exhalation of unchanged solvent.[2]

Qualitative Description of Health Effects

Carcinogenicity

EPA[3] does not consider xylenes to be carcinogenic, based on negative animal and human data. The National Toxicology Program (NTP)[4] has tested xylenes for carcinogenicity by administering the compound orally to rats and mice. Fifty male and female F344 rats were treated by gauge with mixed xylenes at doses of 0, 250, or 500 mg/kg-day, five days/week for 103 weeks. Similarly, $B6C3F_1$ mice received 0, 500, or 1,000 mg/kg-day. NTP concluded at the end of the study that there was no evidence of carcinogenicity of xylene for rats or mice at any dose tested.

The frequency of sister chromatid exchanges and chromosomal aberrations were nearly identical between a group of 17 paint industry workers exposed to xylene and their respective referents.[5] In vitro, xylene caused no increase in the number of sister chromatid exchanges in human lymphocytes.[6]

Studies indicate that xylene isomers, technical grade xylene or mixed xylene are not mutagenic in tests with Salmonella typhimurium[7] nor in mutant reversion assays with Escherichia coli.[8] Technical grade xylene, but not o- and m-xylene, was weakly mutagenic in Drosophila recessive lethal tests. Chromosomal aberrations were not increased in bone marrow cells of rats exposed to xylenes by inhalation.[9] Xylenes were not found to be mutagenic in a battery of short-term tests.[10]

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1 U.S. Environmental Protection Agency. *Drinking Water Criteria Document of Xylenes (Final Draft).* Environmental Criteria and Assessment Office, Cincinnati, Ohio. ECAO-CIN-416. EPA 600/X-84-185-1. March, 1985.

2 U.S. Environmental Protection Agency. National Primary Drinking Water Regulations; Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms, Proposed Rule. *Fed. Reg*. 50:46,936-47,025. November 13, 1985.

3 U.S. Environmental Protection Agency. *Integrated Risk Information System.* 2000.

4 National Toxicology Program. *Carcinogenic Bioassay for Xylenes*. 1986.

5 Haglund, U., I. Lundberg and L. Zech. Chromosome aberrations and sister chromatid exchanges in Swedish paint industry workers. Scand. J. Work Environ. Health. 6: 291-298. 1980.

6 Gerner-Smidt, P. and U. Friedrich. The mutagenic effect of benzene, toluene and xylene studied by the SCE technique. Mutat. Res. 58: 313-316. 1978.

7 Florin, I., L. Rutberg, M. Curvall and C.R. Enzell. Screening of tobacco smoke constituents for mutagenicity using the Ames' test. Toxicology. 15: 219-232. 1980.

8 McCarroll, N.E., C.E. Piper and B.H. Keech. An E. coli microsuspension assay for the detection of DNA damage induced by direct-acting and promutagens. Environ. Mutagen. 3: 429-444. 1981.

9 Donner, M., J. Maki-Paakkanen, H. Norppa, M. Sorsa and H. Vainio. Genetic toxicology of xylenes. Mutat. Res. 74: 171-172. 1980.

10 Litton Bionetics. *Teratology Studies in Rats: Xylene.* Final Report to American Petroleum Institute, Washington, D.C. LBI Project No. 20698-5 (As cited in EPA 1984). 1978.

Teratogenicity/Reproductive Effects

Xylenes appear to be fetotoxic and may increase malformations in the offspring of exposed experimental animals. The available teratogenic studies have reported generally retarded skeletal development and body weight gains in fetuses except for one oral study in mice in which the incidence of cleft palates was increased.[11]

Acute/Chronic Effects

Most of the available toxicity data for xylenes assess adverse effects associated with exposure by inhalation. Acute exposure to relatively high concentrations of xylenes adversely affects the central nervous system and lungs, and can irritate the mucous membranes. The liver is reportedly affected by longer-term exposure to lower levels of xylenes.[12,13]

Quantitative Description of Health Effects

Using the criteria for evaluating the overall weight of evidence of carcinogenicity to humans proposed by EPA's Carcinogen Assessment Group,[14] xylenes are appropriately assigned to Group D - Not Classified because data from animal studies is inadequate.[15]

The Reference Dose (RfD) for ingestion of xylenes is 2 mg/kg-day.[16] The RfD is based on a study by National Toxicology Program[17] in which groups of 50 male and 50 female Fischer 344 rats and 50 male and 50 female B6C3F1 mice were given gavage doses of 0, 250, or 500 mg/kg/day (rats) and 0, 500, or 1000 mg/kg/day (mice) for 5 days/week for 103 weeks. The animals were observed for clinical signs of toxicity, body weight gain, and mortality. All animals that died or were killed at sacrifice were given gross necropsy and comprehensive histologic examinations. There was a dose-related increased mortality in male rats, and the increase was significantly greater in the high-dose group compared with controls. Although increased mortality was observed at 250 mg/kg/day, the increase was not significant. Although many of the early deaths were caused by gavage error, NTP[18] did not rule out the possibility that the rats were resisting gavage dosing because of the behavioral effects of xylene. Mice given the high dose exhibited hyperactivity, a manifestation of CNS toxicity. There were no compound-related histopathologic lesions in any of the treated rats or mice. Therefore, the high dose is a FEL and the low dose a NOAEL.

EPA developed one-day, 10-day, longer-term and lifetime Health Advisories (HAs) for xylenes. The one-day, 10-day and longer-term HAs for children are all 40 mg/L, and the longer-term HA for adults and the lifetime HA are 100 mg/L and 10 mg/L, respectively.[19]

The maximum contaminated level for xylenes is 10 mg/L.[20]

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[11] U.S. Environmental Protection Agency. *Drinking Water Criteria Document of Xylenes (Final Draft).* Environmental Criteria and Assessment Office, Cincinnati, Ohio. ECAO-CIN-416. EPA 600/X-84-185-1. March, 1985.

[12] U.S. Environmental Protection Agency. *Drinking Water Criteria Document for Xylene*. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-066. September, 1984.

[13] U.S. Environmental Protection Agency. *Drinking Water Criteria Document of Xylenes (Final Draft).* Environmental Criteria and Assessment Office, Cincinnati, Ohio. ECAO-CIN-416. EPA 600/X-84-185-1. March, 1985.

[14] U.S. Environmental Protection Agency. Guidelines for Carcinogenic Risk Assessment. *Red. Reg.* 51:33,992-34,003. September 24, 1986.

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[20] U.S. Environmental Protection Agency. *Drinking Water Regulations and Health Advisories.* Office of Water. Washington, D.C. 1991.

Summary of Xylenes Criteria

Criterion	Value	Source
EPA carcinogen classification	Group D	EPA 2000
Oral RfD[1]	2 mg/kg-day	EPA 2000
EPA Drinking Water Health Advisories		
Lifetime Health Advisory (HA)	10 mg/L	EPA 1991
Longer-term HA (Child)	40 mg/L	EPA 1991
Longer-term HA (Adult)	100 mg/L	EPA 1991
10-day HA (Child)	40 mg/L	EPA 1991
One-day HA (Child)	40 mg/L	EPA 1991
MCL	10 mg/L	EPA 1991
MCLG	10 mg/L	EPA 1991
Cal Permissible Exposure Limits, PEL	435 mg/m^3	CCR, Title 8, 2000[2]
Cal Permissible Exposure Limits, STEL	655 mg/m^3	CCR, Title 8, 2000[2]

[1] The oral RfDs for p-xylene is slightly less, suggesting slightly lower toxicity.
[2] California Code of Regulations, Title 8, Section 5155, February 16, 2000.

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Attachment D
Risk Calculations for Maximally Exposed Resident and School Child

Table D-1

Risk Calculation for 2005 Pre-Mitigation Conditions for the No Action/No Project Alternative Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence (mg/m^3)	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	1.26E-04	4.39E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	9.8E-08	2.9E-09	1.4E-07	4.7E-02	1.4E-03	1.3E-02
Acrolein	3.00E-05	1.53E-05	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	5.0E+00	2.2E-01	1.4E+00
Benzene	3.08E-04	1.02E-04	2.90E-02	1.02E-01	NA	1.71E-02	2.6E-06	7.3E-08	3.7E-06	NA	NA	NA
1,3-Butadiene	9.05E-05	3.40E-05	9.80E-01	5.95E-01	NA	2.29E-03	4.4E-06	1.4E-07	6.3E-06	NA	NA	NA
Formaldehyde	3.78E-04	1.44E-04	4.55E-02	2.10E-02	2.00E-01	5.71E-04	6.5E-07	2.1E-08	9.3E-07	1.8E-03	5.9E-05	5.2E-04
Xylene (total)	6.58E-04	1.74E-04	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	3.2E-04	7.2E-06	9.0E-05
PAHs												
Benzo(a)pyrene (TEFs)	2.38E-08	1.05E-08	3.10E+00[2]	3.85E+00	NA	NA	7.5E-09	2.9E-10	1.1E-08	NA	NA	NA
Naphthalene	4.28E-05	1.63E-05	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	4.8E-02	1.6E-03	1.4E-02
Dioxins												
TCDD equivalents	2.78E-11	7.29E-12	1.50E+05[3]	1.33E+05	NA	1.10E-08	3.0E-07	6.8E-09	4.3E-07	NA	NA	NA
Diesel												
Diesel PM	2.14E-04	1.33E-04	NA	1.10E+00	1.43E-03	NA	1.9E-05	1.0E-06	2.8E-05	1.4E-01	7.7E-03	4.1E-02
Metals												
Arsenic	1.15E-08	6.94E-09	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	1.1E-08	5.6E-10	1.6E-08	3.7E-05	1.9E-06	1.0E-05
Beryllium	1.66E-09	9.76E-10	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	9.6E-10	4.8E-11	1.4E-09	2.8E-04	1.4E-05	8.0E-05
Cadmium	2.75E-08	1.70E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	3.3E-08	1.8E-09	4.7E-08	4.6E-04	2.4E-05	1.3E-04
Chromium (VI)	3.33E-10	1.96E-10	4.20E+01	5.25E+02	2.86E-05	2.29E-07	1.4E-08	7.2E-10	2.1E-08	1.1E-05	5.6E-07	3.2E-06
Manganese	6.35E-07	3.72E-07	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	4.3E-02	2.1E-03	1.2E-02
						TOTAL	**2.7E-05**	**1.3E-06**	**3.9E-05**	**5.33**	**0.23**	**1.52**
						Total HI Based on Respiratory Effects				**5.28**	**0.23**	**1.51**

NA = Not Available

1 Oral Value
2 Oral Slope Factor - USEPA
3 HEAST Value
4 Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-2

Risk Calculation for 2005 Pre-Mitigation Conditions for Alternatives A, B, and C Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence (mg/m^3)	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	6.12E-05	3.60E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	4.8E-08	2.4E-09	6.8E-08	2.3E-02	1.2E-03	6.5E-03
Acrolein	2.88E-05	1.42E-05	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	4.8E+00	2.1E-01	1.4E+00
Benzene	5.18E-05	5.15E-05	2.90E-02	1.02E-01	NA	1.71E-02	4.3E-07	3.7E-08	6.2E-07	NA	NA	NA
1,3-Butadiene	3.58E-05	2.26E-05	9.80E-01	5.95E-01	NA	2.29E-03	1.8E-06	9.5E-08	2.5E-06	NA	NA	NA
Formaldehyde	2.10E-04	1.20E-04	4.55E-02	2.10E-02	2.00E-01	5.71E-04	3.6E-07	1.8E-08	5.2E-07	1.0E-03	4.9E-05	2.9E-04
Xylene (total)	4.77E-05	8.53E-05	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	2.3E-05	3.5E-06	6.5E-06
PAHs												
Benzo(a)pyrene (TEFs)	1.26E-08	7.64E-09	3.10E+00[2]	3.85E+00	NA	NA	4.0E-09	2.1E-10	5.7E-09	NA	NA	NA
Naphthalene	2.27E-05	1.46E-05	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	2.5E-02	1.4E-03	7.2E-03
Dioxins												
TCDD equivalents	1.20E-04	9.80E-06	NA	1.10E+00	1.43E-03	NA	1.1E-05	7.6E-08	1.6E-05	8.1E-02	5.6E-04	2.3E-02
Diesel												
Diesel PM	1.98E-12	3.54E-12	1.50E+05[3]	1.33E+05	NA	1.10E-08	2.2E-08	3.3E-09	3.1E-08	NA	NA	NA
Metals												
Arsenic	1.56E-08	7.77E-09	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	1.5E-08	6.3E-10	2.1E-08	5.0E-05	2.1E-06	1.4E-05
Beryllium	2.73E-09	1.32E-09	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	1.6E-09	6.5E-11	2.2E-09	4.6E-04	1.9E-05	1.3E-04
Cadmium	3.03E-08	2.14E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	3.7E-08	2.2E-09	5.2E-08	5.1E-04	3.1E-05	1.5E-04
Chromium (VI)	5.45E-10	2.63E-10	4.20E+01	5.25E+02	2.86E-05	2.29E-07	2.4E-08	9.7E-10	3.4E-08	1.8E-05	7.6E-07	5.2E-06
Manganese	8.19E-07	4.21E-07	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	5.5E-02	2.4E-03	1.6E-02
						TOTAL	**1.4E-05**	**2.4E-07**	**1.9E-05**	**5.02**	**0.21**	**1.44**
						Total HI Based on Respiratory Effects				**5.00**	**0.21**	**1.43**

NA = Not Available

[1] Oral Value
[2] Oral Slope Factor – USEPA
[3] HEAST Value
[4] Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-3

Risk Calculation for 2015 Pre-Mitigation Conditions for the No Action/No Project Alternative Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence (mg/m^3)	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	9.99E-05	4.41E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	7.76E-08	2.94E-09	1.11E-07	3.7E-02	1.4E-03	1.1E-02
Acrolein	3.33E-05	1.87E-05	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	5.6E+00	2.7E-01	1.6E+00
Benzene	1.75E-04	7.27E-05	2.90E-02	1.02E-01	NA	1.71E-02	1.46E-06	5.20E-08	2.09E-06	NA	NA	NA
1,3-Butadiene	6.38E-05	3.05E-05	9.80E-01	5.95E-01	NA	2.29E-03	3.12E-06	1.28E-07	4.45E-06	NA	NA	NA
Formaldehyde	3.27E-04	1.55E-04	4.55E-02	2.10E-02	2.00E-01	5.71E-04	5.64E-07	2.29E-08	8.05E-07	1.6E-03	6.4E-05	4.5E-04
Xylene (total)	2.92E-04	8.75E-05	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	1.4E-04	3.6E-06	4.0E-05
PAHs												
Benzo(a)pyrene (TEFs)	2.93E-08	1.59E-08	3.10E+00[2]	3.85E+00	NA	NA	9.27E-09	4.31E-10	1.32E-08	NA	NA	NA
Naphthalene	4.25E-05	2.33E-05	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	4.8E-02	2.2E-03	1.4E-02
Dioxins												
TCDD equivalents	1.38E-11	4.54E-12	1.50E+05[3]	1.33E+05	NA	1.10E-08	1.51E-07	4.26E-09	2.15E-07	NA	NA	NA
Diesel												
Diesel PM	1.94E-04	1.14E-04	NA[3]	1.10E+00	1.43E-03	NA	1.76E-05	8.82E-07	2.51E-05	1.3E-01	6.5E-03	3.7E-02
Metals												
Arsenic	1.94E-08	1.26E-08	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	1.84E-08	1.02E-09	2.63E-08	6.2E-05	3.4E-06	1.8E-05
Beryllium	3.84E-09	2.51E-09	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	2.21E-09	1.24E-10	3.15E-09	6.5E-04	3.6E-05	1.8E-04
Cadmium	3.99E-08	2.56E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	4.82E-08	2.65E-09	6.88E-08	6.7E-04	3.7E-05	1.9E-04
Chromium (VI)	7.69E-10	5.03E-10	4.20E+01	5.25E+02	2.86E-05	2.29E-07	3.32E-08	1.86E-09	4.74E-08	2.6E-05	1.4E-06	7.4E-06
Manganese	1.05E-06	6.66E-07	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	7.1E-02	3.8E-03	2.0E-02
						TOTAL	**2.3E-05**	**1.1E-06**	**3.3E-05**	**5.9**	**0.28**	**1.68**
						Total HI for Respiratory Effects				**5.8**	**0.28**	**1.67**

NA = Not Available

1 Oral Value
2 Oral Slope Factor - USEPA
3 HEAST Value
4 Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-4

Risk Calculation for 2015 Pre-Mitigation Conditions for Alternative A Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence (mg/m^3)	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	6.35E-05	4.47E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	4.94E-08	2.97E-09	7.05E-08	2.4E-02	1.4E-03	6.8E-03
Acrolein	3.57E-05	2.05E-05	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	6.0E+00	3.0E-01	1.7E+00
Benzene	1.84E-05	3.05E-05	2.90E-02	1.02E-01	NA	1.71E-02	1.54E-07	2.18E-08	2.20E-07	NA	NA	NA
1,3-Butadiene	3.66E-05	2.54E-05	9.80E-01	5.95E-01	NA	2.29E-03	1.79E-06	1.06E-07	2.56E-06	NA	NA	NA
Formaldehyde	2.07E-04	1.40E-04	4.55E-02	2.10E-02	2.00E-01	5.71E-04	3.58E-07	2.07E-08	5.12E-07	9.9E-04	5.8E-05	2.8E-04
Xylene (total)	-3.79E-05	3.82E-05	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	-1.8E-05	1.6E-06	-5.2E-06
PAHs												
Benzo(a)pyrene (TEFs)	2.74E-08	1.82E-08	3.10E+00[2]	3.85E+00	NA	NA	8.67E-09	4.93E-10	1.24E-08	NA	NA	NA
Naphthalene	5.51E-05	3.68E-05	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	6.2E-02	3.5E-03	1.8E-02
Dioxins												
TCDD equivalents	1.36E-12	3.56E-12	1.50E+05[3]	1.33E+05	NA	1.10E-08	1.49E-08	3.34E-09	2.12E-08	NA	NA	NA
Diesel												
Diesel PM	-1.10E-04	-6.38E-05	NA[3]	1.10E+00	1.43E-03	NA	-9.97E-06	-4.95E-07	-1.42E-05	-7.4E-02	-3.7E-03	-2.1E-02
Metals												
Arsenic	3.29E-08	2.07E-08	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	3.12E-08	1.68E-09	4.46E-08	1.1E-04	5.7E-06	3.0E-05
Beryllium	8.05E-09	4.94E-09	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	4.63E-09	2.43E-10	6.61E-09	1.4E-03	7.1E-05	3.9E-04
Cadmium	4.39E-08	5.11E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	5.30E-08	5.29E-09	7.57E-08	7.4E-04	7.4E-05	2.1E-04
Chromium (VI)	1.60E-09	9.84E-10	4.20E+01	5.25E+02	2.86E-05	2.29E-07	6.92E-08	3.64E-09	9.88E-08	5.4E-05	2.8E-06	1.5E-05
Manganese	1.73E-06	1.08E-06	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	1.2E-01	6.2E-03	3.3E-02
						TOTAL	**-7.4E-06**	**-3.3E-07**	**-1.1E-05**	**6.1**	**0.30**	**1.75**
						Total HI for Respiratory Effects				**6.1**	**0.30**	**1.73**

NA = Not Available

1 Oral Value
2 Oral Slope Factor – USEPA
3 HEAST Value
4 Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-5

Risk Calculation for 2015 Pre-Mitigation Conditions for Alternative B Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence [3])	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	1.62E-04	7.98E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	1.26E-07	5.31E-09	1.80E-07	6.1E-02	2.6E-03	1.7E-02
Acrolein	8.02E-05	3.55E-05	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	1.3E+01	5.1E-01	3.9E+00
Benzene	8.49E-05	5.50E-05	2.90E-02	1.02E-01	NA	1.71E-02	7.08E-07	3.94E-08	1.01E-06	NA	NA	NA
1,3-Butadiene	9.47E-05	4.56E-05	9.80E-01	5.95E-01	NA	2.29E-03	4.63E-06	1.91E-07	6.62E-06	NA	NA	NA
Formaldehyde	5.27E-04	2.52E-04	4.55E-02	2.10E-02	2.00E-01	5.71E-04	9.10E-07	3.72E-08	1.30E-06	2.5E-03	1.0E-04	7.2E-04
Xylene (total)	4.78E-05	7.82E-05	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	2.3E-05	3.2E-06	6.6E-06
PAHs												
Benzo(a)pyrene (TEFs)	5.50E-08	2.65E-08	3.10E+00[2]	3.85E+00	NA	NA	1.74E-08	7.19E-10	2.49E-08	NA	NA	NA
Naphthalene	1.10E-04	5.42E-05	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	1.2E-01	5.2E-03	3.5E-02
Dioxins												
TCDD equivalents	6.34E-12	5.53E-12	1.50E+05[3]	1.33E+05	NA	1.10E-08	6.93E-08	5.18E-09	9.90E-08	NA	NA	NA
Diesel												
Diesel PM	-1.01E-07	-1.94E-05	NA[3]	1.10E+00	1.43E-03	NA	-9.11E-09	-1.51E-07	-1.30E-08	-6.8E-05	-1.1E-03	-1.9E-05
Metals												
Arsenic	5.82E-08	2.82E-08	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	5.53E-08	2.29E-09	7.90E-08	1.9E-04	7.7E-06	5.3E-05
Beryllium	1.41E-08	6.66E-09	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	8.12E-09	3.28E-10	1.16E-08	2.4E-03	9.6E-05	6.8E-04
Cadmium	1.67E-07	8.89E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	2.02E-07	9.21E-09	2.88E-07	2.8E-03	1.3E-04	8.0E-04
Chromium (VI)	2.81E-09	1.33E-09	4.20E+01	5.25E+02	2.86E-05	2.29E-07	1.21E-07	4.91E-09	1.73E-07	9.4E-05	3.8E-06	2.7E-05
Manganese	3.00E-06	1.46E-06	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	2.0E-01	8.4E-03	5.7E-02
						TOTAL	**6.8E-06**	**1.5E-07**	**9.8E-06**	**13.9**	**0.53**	**3.97**
						Total HI for Respiratory Effects				**13.8**	**0.52**	**3.93**

NA = Not Available

1 Oral Value
2 Oral Slope Factor – USEPA
3 HEAST Value
4 Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-6

Risk Calculation for 2015 Pre-Mitigation Conditions for Alternative C Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence (mg/m^3)	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	1.28E-04	6.49E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	9.92E-08	4.32E-09	1.42E-07	4.8E-02	2.1E-03	1.4E-02
Acrolein	6.47E-05	2.94E-05	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	1.1E+01	4.2E-01	3.1E+00
Benzene	4.14E-05	3.80E-05	2.90E-02	1.02E-01	NA	1.71E-02	3.45E-07	2.72E-08	4.93E-07	NA	NA	NA
1,3-Butadiene	7.23E-05	3.68E-05	9.80E-01	5.95E-01	NA	2.29E-03	3.54E-06	1.54E-07	5.05E-06	NA	NA	NA
Formaldehyde	4.13E-04	2.04E-04	4.55E-02	2.10E-02	2.00E-01	5.71E-04	7.13E-07	3.02E-08	1.02E-06	2.0E-03	8.4E-05	5.7E-04
Xylene (total)	-3.67E-06	5.42E-05	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	-1.8E-06	2.2E-06	-5.0E-07
PAHs												
Benzo(a)pyrene (TEFs)	4.37E-08	2.20E-08	3.10E+00[2]	3.85E+00	NA	NA	1.38E-08	5.96E-10	1.97E-08	NA	NA	NA
Naphthalene	9.09E-05	4.67E-05	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	1.0E-01	4.5E-03	2.9E-02
Dioxins												
TCDD equivalents	3.64E-12	4.18E-12	1.50E+05[3]	1.33E+05	NA	1.10E-08	3.98E-08	3.92E-09	5.69E-08	NA	NA	NA
Diesel												
Diesel PM	-4.79E-05	-4.94E-05	NA[3]	1.10E+00	1.43E-03	NA	-4.33E-06	-3.82E-07	-6.19E-06	-3.2E-02	-2.8E-03	-9.2E-03
Metals												
Arsenic	4.89E-08	2.43E-08	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	4.65E-08	1.97E-09	6.64E-08	1.6E-04	6.6E-06	4.5E-05
Beryllium	1.17E-08	5.58E-09	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	6.74E-09	2.75E-10	9.62E-09	2.0E-03	8.0E-05	5.6E-04
Cadmium	6.31E-08	4.70E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	7.62E-08	4.87E-09	1.09E-07	1.1E-03	6.8E-05	3.0E-04
Chromium (VI)	2.33E-09	1.11E-09	4.20E+01	5.25E+02	2.86E-05	2.29E-07	1.01E-07	4.11E-09	1.44E-07	7.8E-05	3.2E-06	2.2E-05
Manganese	2.56E-06	1.28E-06	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	1.7E-01	7.3E-03	4.9E-02
						TOTAL	**6.4E-07**	**-1.5E-07**	**9.2E-07**	**11.2**	**0.44**	**3.19**
						Total HI for Respiratory Effects				**11.1**	**0.43**	**3.16**

NA = Not Available

[1] Oral Value
[2] Oral Slope Factor – USEPA
[3] HEAST Value
[4] Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-7

Risk Calculation for 2005 Post-Mitigation Conditions for Alternative C Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence [3])	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	-2.05E-05	-1.66E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	-1.6E-08	-1.1E-09	-2.3E-08	-7.6E-03	-5.3E-04	-2.2E-03
Acrolein	-1.10E-05	-1.14E-05	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	-1.8E+00	-1.6E-01	-5.3E-01
Benzene	-7.99E-05	-3.46E-05	2.90E-02	1.02E-01	NA	1.71E-02	-6.7E-07	-2.5E-08	-9.5E-07	NA	NA	NA
1,3-Butadiene	-2.58E-05	-1.72E-05	9.80E-01	5.95E-01	NA	2.29E-03	-1.3E-06	-7.2E-08	-1.8E-06	NA	NA	NA
Formaldehyde	-6.48E-05	-5.77E-05	4.55E-02	2.10E-02	2.00E-01	5.71E-04	-1.1E-07	-8.5E-09	-1.6E-07	-3.1E-04	-2.4E-05	-8.9E-05
Xylene (total)	-9.84E-05	-1.05E-05	NA	NA	2.00E+00 [1]	5.71E-02	NA	NA	NA	-4.7E-05	-4.3E-07	-1.3E-05
PAHs												
Benzo(a)pyrene (TEFs)	-1.32E-08	-9.11E-09	3.10E+00 [2]	3.85E+00	NA	NA	-4.2E-09	-2.5E-10	-6.0E-09	NA	NA	NA
Naphthalene	-2.52E-05	-1.63E-05	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	-2.8E-02	-1.6E-03	-8.1E-03
Dioxins												
TCDD equivalents	6.69E-05	-2.53E-05	NA	1.10E+00	1.43E-03	NA	6.0E-06	-2.0E-07	8.6E-06	4.5E-02	-1.5E-03	1.3E-02
Diesel												
Diesel PM	-5.07E-12	-1.03E-12	1.50E+05 [3]	1.33E+05	NA	1.10E-08	-5.5E-08	-9.7E-10	-7.9E-08	NA	NA	NA
Metals												
Arsenic	-5.00E-09	-5.52E-09	1.51E+01	1.16E+01	3.00E-04 [1]	8.57E-06	-4.7E-09	-4.5E-10	-6.8E-09	-1.6E-05	-1.5E-06	-4.6E-06
Beryllium	-2.46E-09	-2.03E-09	8.40E+00	7.00E+00 [4]	5.70E-06	2.86E-07	-1.4E-09	-1.0E-10	-2.0E-09	-4.1E-04	-2.9E-05	-1.2E-04
Cadmium	-4.86E-09	-1.26E-09	6.30E+00	1.47E+01	5.71E-05	2.86E-06	-5.9E-09	-1.3E-10	-8.4E-09	-8.2E-05	-1.8E-06	-2.3E-05
Chromium (VI)	-4.87E-10	-4.08E-10	4.20E+01	5.25E+02	2.86E-05	2.29E-07	-2.1E-08	-1.5E-09	-3.0E-08	-1.6E-05	-1.2E-06	-4.7E-06
Manganese	-2.36E-07	-2.60E-07	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	-1.6E-02	-1.5E-03	-4.5E-03
						TOTAL	**3.90E-06**	**-3.06E-07**	**5.57E-06**	**-1.85**	**-0.17**	**-0.53**
						Total HI Based on Respiratory Effects				**-1.83**	**-0.17**	**-0.52**

NA = Not Available

[1] Oral Value
[2] Oral Slope Factor – USEPA
[3] HEAST Value
[4] Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-8

Risk Calculation for 2015 Post-Mitigation Conditions for Alternative A Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence (mg/m^3)	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	-2.36E-05	-1.32E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	-1.84E-08	-8.79E-10	-2.62E-08	-8.8E-03	-4.2E-04	-2.5E-03
Acrolein	-7.20E-06	-7.85E-06	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	-1.2E+00	-1.1E-01	-3.5E-01
Benzene	-8.23E-05	-3.48E-05	2.90E-02	1.02E-01	NA	1.71E-02	-6.87E-07	-2.49E-08	-9.81E-07	NA	NA	NA
1,3-Butadiene	-2.32E-05	-1.40E-05	9.80E-01	5.95E-01	NA	2.29E-03	-1.14E-06	-5.88E-08	-1.62E-06	NA	NA	NA
Formaldehyde	-8.21E-05	-5.21E-05	4.55E-02	2.10E-02	2.00E-01	5.71E-04	-1.42E-07	-7.70E-09	-2.02E-07	-3.9E-04	-2.1E-05	-1.1E-04
Xylene (total)	-1.50E-04	-3.40E-05	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	-7.2E-05	-1.4E-06	-2.1E-05
PAHs												
Benzo(a)pyrene (TEFs)	-2.30E-09	-1.36E-09	3.10E+00[2]	3.85E+00	NA	NA	-7.26E-10	-3.69E-11	-1.04E-09	NA	NA	NA
Naphthalene	-4.24E-06	-2.33E-06	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	-4.7E-03	-2.2E-04	-1.4E-03
Dioxins												
TCDD equivalents	-5.15E-12	-6.68E-13	1.50E+05[3]	1.33E+05	NA	1.10E-08	-5.63E-08	-6.26E-10	-8.04E-08	NA	NA	NA
Diesel												
Diesel PM	-1.20E-04	-7.18E-05	NA[3]	1.10E+00	1.43E-03	NA	-1.08E-05	-5.56E-07	-1.54E-05	-8.0E-02	-4.1E-03	-2.3E-02
Metals												
Arsenic	7.30E-09	3.79E-09	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	6.93E-09	3.08E-10	9.89E-09	2.3E-05	1.0E-06	6.7E-06
Beryllium	1.34E-09	5.08E-10	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	7.68E-10	2.50E-11	1.10E-09	2.2E-04	7.3E-06	6.4E-05
Cadmium	2.20E-09	2.36E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	2.66E-09	2.44E-09	3.80E-09	3.7E-05	3.4E-05	1.1E-05
Chromium (VI)	2.68E-10	1.03E-10	4.20E+01	5.25E+02	2.86E-05	2.29E-07	1.16E-08	3.80E-10	1.65E-08	9.0E-06	3.0E-07	2.6E-06
Manganese	4.15E-07	2.14E-07	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	2.8E-02	1.2E-03	7.9E-03
						TOTAL	**-1.3E-05**	**-6.5E-07**	**-1.8E-05**	**-1.3**	**-0.12**	**-0.36**
						Total HI for Respiratory Effects				**-1.3**	**-0.12**	**-0.36**

NA = Not Available

[1] Oral Value
[2] Oral Slope Factor – USEPA
[3] HEAST Value
[4] Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-9

Risk Calculation for 2015 Post-Mitigation Conditions for Alternative B Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence [3])	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	3.87E-05	1.09E-05	7.70E-03	9.45E-03	2.57E-03	2.57E-03	3.01E-08	7.23E-10	4.30E-08	1.4E-02	3.5E-04	4.1E-03
Acrolein	2.07E-05	2.23E-06	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	3.5E+00	3.2E-02	1.0E+00
Benzene	-5.38E-05	-2.01E-05	2.90E-02	1.02E-01	NA	1.71E-02	-4.49E-07	-1.44E-08	-6.41E-07	NA	NA	NA
1,3-Butadiene	1.13E-05	-6.05E-07	9.80E-01	5.95E-01	NA	2.29E-03	5.51E-07	-2.54E-09	7.87E-07	NA	NA	NA
Formaldehyde	1.18E-04	2.38E-05	4.55E-02	2.10E-02	2.00E-01	5.71E-04	2.04E-07	3.53E-09	2.92E-07	5.7E-04	9.8E-06	1.6E-04
Xylene (total)	-1.06E-04	-5.44E-06	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	-5.1E-05	-2.2E-07	-1.5E-05
PAHs												
Benzo(a)pyrene (TEFs)	1.30E-08	3.35E-09	3.10E+00[2]	3.85E+00	NA	NA	4.11E-09	9.09E-11	5.87E-09	NA	NA	NA
Naphthalene	2.66E-05	7.91E-06	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	3.0E-02	7.6E-04	8.5E-03
Dioxins												
TCDD equivalents	-2.77E-12	5.71E-13	1.50E+05[3]	1.33E+05	NA	1.10E-08	-3.03E-08	5.35E-10	-4.32E-08	NA	NA	NA
Diesel												
Diesel PM	-3.81E-05	-3.86E-05	NA[3]	1.10E+00	1.43E-03	NA	-3.44E-06	-2.99E-07	-4.92E-06	-2.6E-02	-2.2E-03	-7.3E-03
Metals												
Arsenic	2.22E-08	8.21E-09	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	2.10E-08	6.68E-10	3.01E-08	7.1E-05	2.2E-06	2.0E-05
Beryllium	4.64E-09	1.42E-09	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	2.67E-09	6.98E-11	3.81E-09	7.8E-04	2.0E-05	2.2E-04
Cadmium	1.07E-07	5.60E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	1.30E-07	5.80E-09	1.85E-07	1.8E-03	8.1E-05	5.2E-04
Chromium (VI)	9.29E-10	2.83E-10	4.20E+01	5.25E+02	2.86E-05	2.29E-07	4.01E-08	1.05E-09	5.72E-08	3.1E-05	8.1E-07	8.9E-06
Manganese	1.16E-06	4.39E-07	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	7.8E-02	2.5E-03	2.2E-02
						TOTAL	**-2.9E-06**	**-3.0E-07**	**-4.2E-06**	**3.6**	**0.03**	**1.02**
						Total HI for Respiratory Effects				**3.6**	**0.03**	**1.02**

NA = Not Available

[1] Oral Value
[2] Oral Slope Factor – USEPA
[3] HEAST Value
[4] Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

Table D-10

Risk Calculation for 2015 Post-Mitigation Conditions for Alternative C Minus Baseline Conditions

Exposure Parameters	Residential Child		School Child		Residential Adult	
Inhalation Rate	15	m^3/day	6	m^3/day	20	m^3/day
Exposure Frequency	350	days/year	200	days/year	350	days/year
Exposure Duration	6	years	6	years	30	years
Body Weight	15	kg	40	kg	70	kg
Averaging Time (Carcinogen)	25550	days	25550	days	25550	days
Averaging Time (Noncarcinogen)	2190	days	2190	days	10950	days

	Location-Specific Concentrations		Toxicity Criteria				Cancer Risks			Hazard Quotients		
TAP	Concentration at Residence (mg/m^3)	Concentration at School Location (mg/m^3)	USEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	CalEPA Inhalation Slope Factor $(mg/kg\text{-}d)^{-1}$	USEPA RfDi (mg/kg-d)	CalEPA Proposed REL (mg/kg-d)	Cancer Risk to Child Resident	Cancer Risk to School Child	Cancer Risk to Adult Resident	Hazard Quotient Child Resident	Hazard Quotient School Child	Hazard Quotient Adult Resident
VOCs												
Acetaldehyde	1.73E-06	-5.67E-06	7.70E-03	9.45E-03	2.57E-03	2.57E-03	1.34E-09	-3.77E-10	1.92E-09	6.5E-04	-1.8E-04	1.8E-04
Acrolein	4.03E-06	-4.84E-06	NA	NA	5.70E-06	5.71E-06	NA	NA	NA	6.8E-01	-7.0E-02	1.9E-01
Benzene	-8.24E-05	-3.44E-05	2.90E-02	1.02E-01	NA	1.71E-02	-6.87E-07	-2.46E-08	-9.82E-07	NA	NA	NA
1,3-Butadiene	-1.06E-05	-1.02E-05	9.80E-01	5.95E-01	NA	2.29E-03	-5.19E-07	-4.29E-08	-7.42E-07	NA	NA	NA
Formaldehyde	-1.88E-06	-2.89E-05	4.55E-02	2.10E-02	2.00E-01	5.71E-04	-3.24E-09	-4.28E-09	-4.63E-09	-9.0E-06	-1.2E-05	-2.6E-06
Xylene (total)	-1.43E-04	-2.69E-05	NA	NA	2.00E+00[1]	5.71E-02	NA	NA	NA	-6.9E-05	-1.1E-06	-2.0E-05
PAHs												
Benzo(a)pyrene (TEFs)	3.24E-09	-8.02E-10	3.10E+00[2]	3.85E+00	NA	NA	1.02E-09	-2.18E-11	1.46E-09	NA	NA	NA
Naphthalene	8.72E-06	5.94E-07	NA	NA	8.57E-04	2.57E-03	NA	NA	NA	9.8E-03	5.7E-05	2.8E-03
Dioxins												
TCDD equivalents	-4.74E-12	-6.13E-13	1.50E+05[3]	1.33E+05	NA	1.10E-08	-5.19E-08	-5.74E-10	-7.41E-08	NA	NA	NA
Diesel												
Diesel PM	-7.30E-05	-6.09E-05	NA[3]	1.10E+00	1.43E-03	NA	-6.60E-06	-4.72E-07	-9.42E-06	-4.9E-02	-3.5E-03	-1.4E-02
Metals												
Arsenic	1.33E-08	4.27E-09	1.51E+01	1.16E+01	3.00E-04[1]	8.57E-06	1.26E-08	3.47E-10	1.80E-08	4.2E-05	1.2E-06	1.2E-05
Beryllium	4.62E-09	3.39E-10	8.40E+00	7.00E+00[4]	5.70E-06	2.86E-07	2.66E-09	1.67E-11	3.80E-09	7.8E-04	4.9E-06	2.2E-04
Cadmium	4.62E-09	1.43E-08	6.30E+00	1.47E+01	5.71E-05	2.86E-06	5.58E-09	1.49E-09	7.98E-09	7.8E-05	2.1E-05	2.2E-05
Chromium (VI)	4.73E-10	6.88E-11	4.20E+01	5.25E+02	2.86E-05	2.29E-07	2.04E-08	2.54E-10	2.92E-08	1.6E-05	2.0E-07	4.5E-06
Manganese	7.32E-07	2.52E-07	NA	NA	1.43E-05	1.43E-05	NA	NA	NA	4.9E-02	1.4E-03	1.4E-02
						TOTAL	**-7.8E-06**	**-5.4E-07**	**-1.1E-05**	**0.7**	**-0.07**	**0.20**
						Total HI for Respiratory Effects				**0.7**	**-0.07**	**0.19**

NA = Not Available

[1] Oral Value
[2] Oral Slope Factor – USEPA
[3] HEAST Value
[4] Beryllium Oxide Value

Source: Camp Dresser & McKee Inc., 2000.

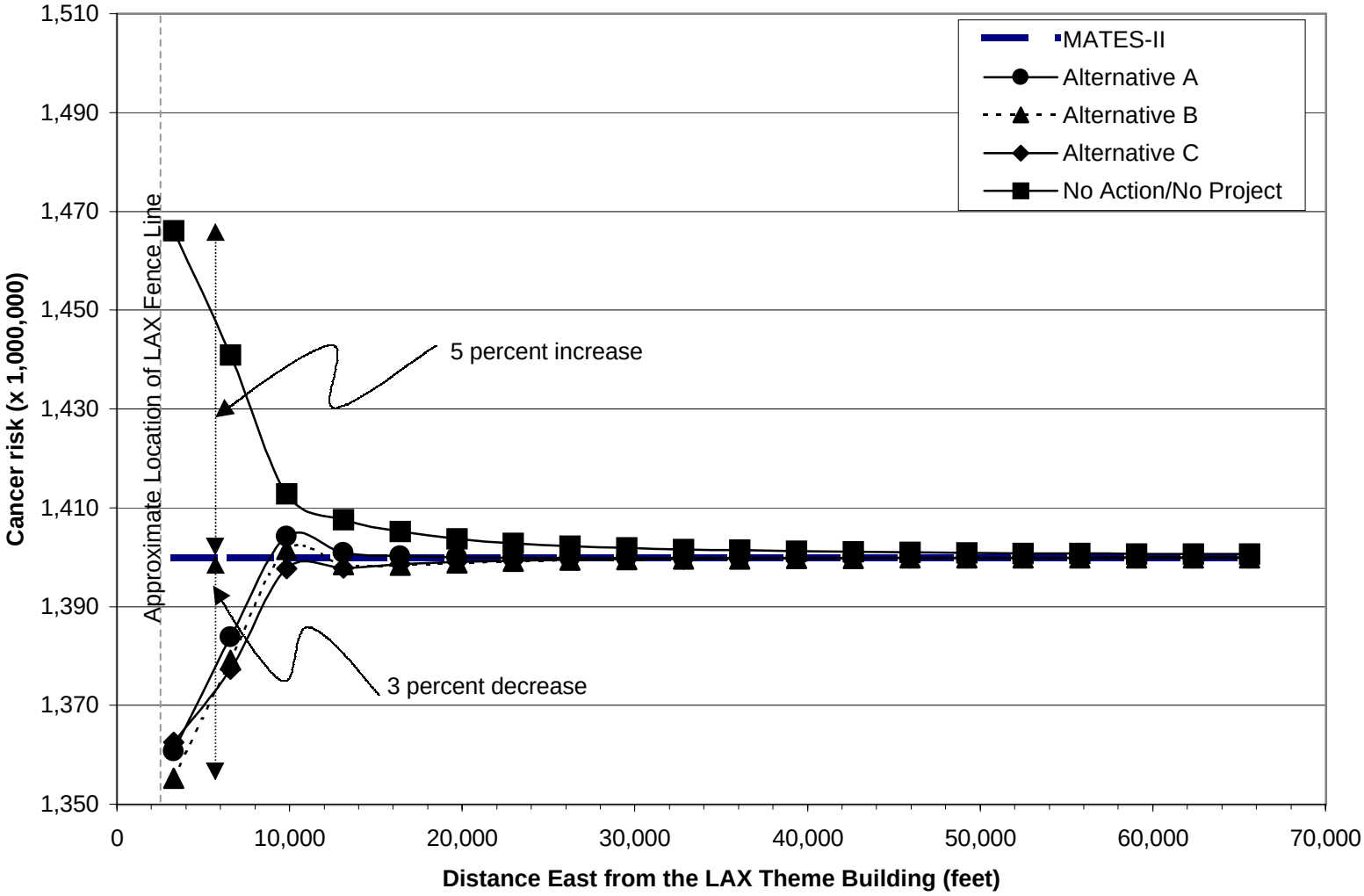
Table D-11

List of Sensitive Receptors Identified in Figure 3

Number	Name	Type	Number	Name	Type
14	Hawthorne Convalescent Center	Convalescent Home	188	Warren Lane Elementary School	Public School
18	C & H Health Care	Convalescent Home	189	Morningside High School	Public School
21	Terrace Inglewood Brierwood	Convalescent Home	190	Centinela Elementary School	Public School
24	Carewest Nursing Center	Convalescent Home	191	Kelso Elementary School	Public School
31	KLOKKE Corporation	Home	193	Hillcrest Continuation School	Public School
36	Saint Erne Healthcare Center	Home	194	Oak Street Elementary School	Public School
37	Centinela Valley Care Center	Home	197	Worthington Elementary School	Public School
50	Urban Healthcare Project Inc.	Home	198	Hudnall Elementary School	Public School
56	Mount Zion Baptist Church of Los Angeles	Home	199	Boulah Payne Elementary School	Public School
60	State of California	Hospital	200	Clyde Woodworth Elementary / Albert Monroe Middle	Public School
62	Crippled Children's Society of	Hospital	206	Lennox Middle School	Public School
66	Freeman MED Towers LP	Hospital	207	Felton Elementary School	Public School
68	Golden West Convalesent Hospital Investm	Hospital	208	Century park Elementary School	Public School
70	Burton Russell CO	Hospital	209	Inglewood High School	Public School
74	Washington Mut BK	Hospital	211	Arena High School	Public School
75	DESCO Health Care INC	Hospital	213	Whelan Elementary School	Public School
77	Catholic Healthcare West Southern California	Hospital	214	Buford Elementary School	Public School
79	Morningside United Church of Christ	Private School	219	Eucalyptus School	Public School
82	Hilltop Christian School	Private School	220	Jefferson Elementary School	Public School
85	Musical HART Evangelistic ASSN INC	Private School	222	Moffet Elementary School	Public School
88	Saint Anthony's Catholic School	Private School	223	Crozier Middle School	Public School
92	LA Southside Christian Church	Private School	237	Juan de Anza Elementary School	Public School
105	St Joseph's Catholic Church School	Private School	253	Washington School	Public School
106	South Bay Lutheran High School	Private School	258	Bennet-Kew Elementary School	Public School
107	Trinity Lutheran CH of Hawthorne	Private School	260	Hawthorne High School	Public School
117	Escuela de Montessori	Private School	262	Westpoint Heights Elementary School	Public School
119	Visitation Catholic School	Private School	265	York School	Public School
120	St. Anastasia School	Private School	271	Center Street Elementary School	Public School
121	Acacia Baptist School	Private School	272	El Segundo High School	Public School
122	St. Bernard High School	Private School	273	El Segundo Middle School	Public School
123	Faith Lutheran Church School	Private School	276	Kentwood Elementary School	Public School
124	CHABAD of the Marina	Private School	279	Loyola Village Elementary School	Public School
127	Westchester Lutheran Church	Private School	280	Westchester High School and Magnet Center	Public School
139	ST Marys' Academy of L A	Private School	281	Paseo del Rey Magnet School	Public School
140	Lindgren Ptnrshp 1	Private School	282	Cowan Avenue Elementary School	Public School
157	St Eugene's Catholic School	Private School	283	Orville Wright Junior High School	Public School
183	Ingelwood Christian School	Private School	284	Imperial Avenue School Special Education Facility	Public School
186	K-Anthony's Middle School	Private School			

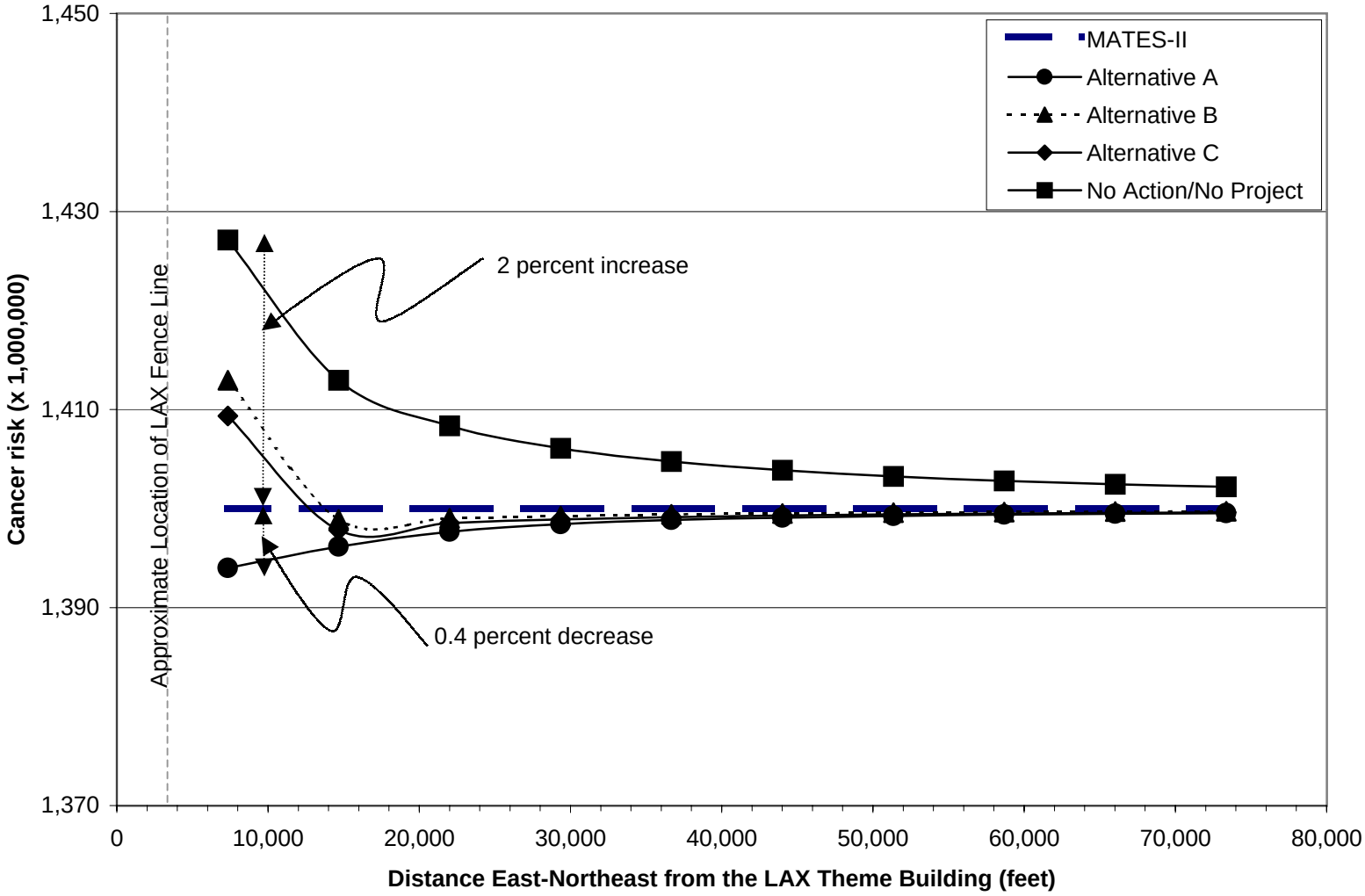
Attachment E
Risks Associated with the Build Alternatives Compared to Background

**Graph E-1 Cancer Risks, Inclusive of Environmental Baseline,
for the Build Alternatives and the No Action/No Project Alternative,
East Projection 2015 Pre-Mitigation**



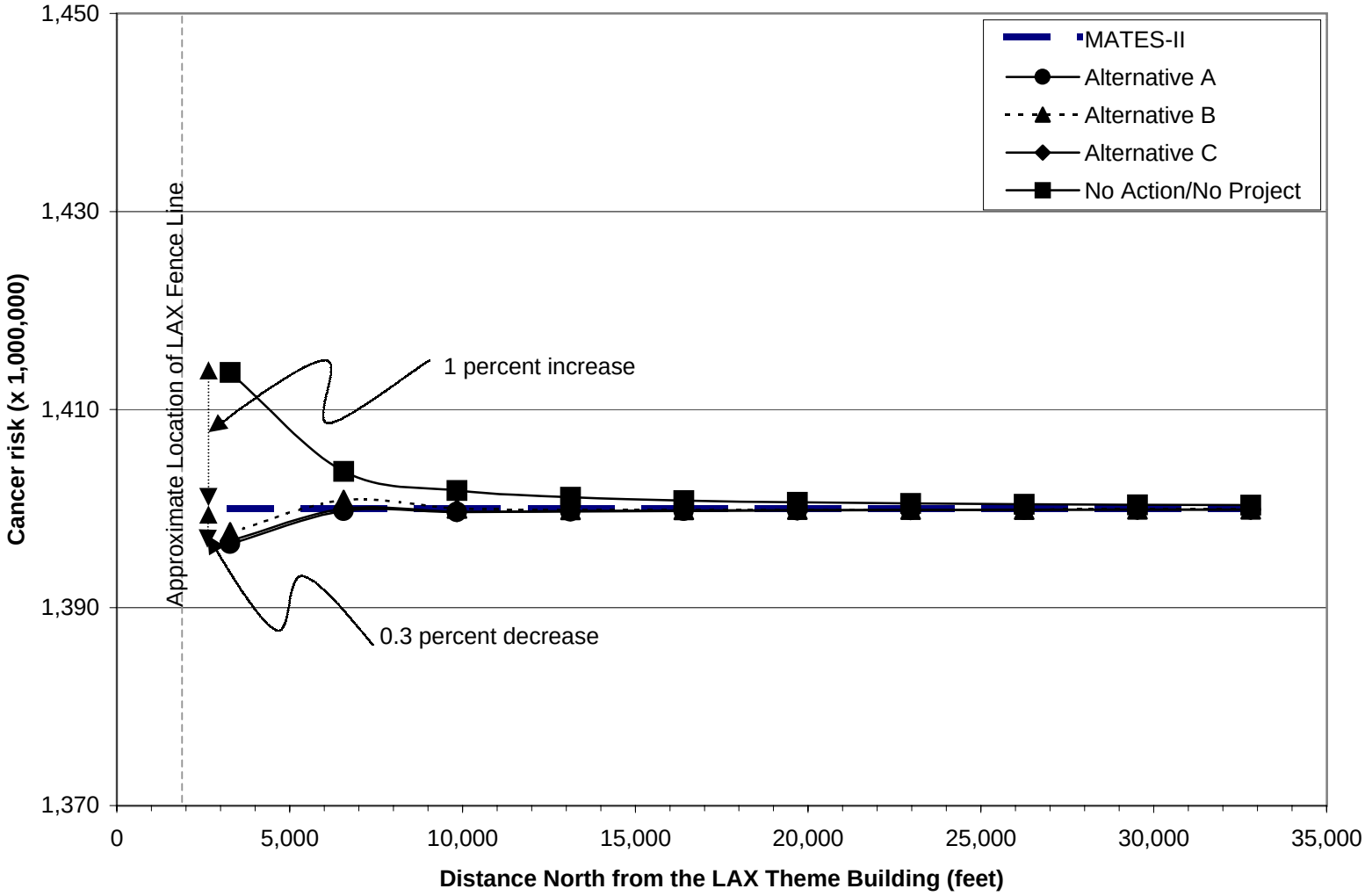
k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig East_pre

**Graph E-2 Cancer Risks, Inclusive of Environmental Baseline,
for the Build Alternatives and the No Action/No Project Alternative,
East-Northeast Projection 2015 Pre-Mitigation**



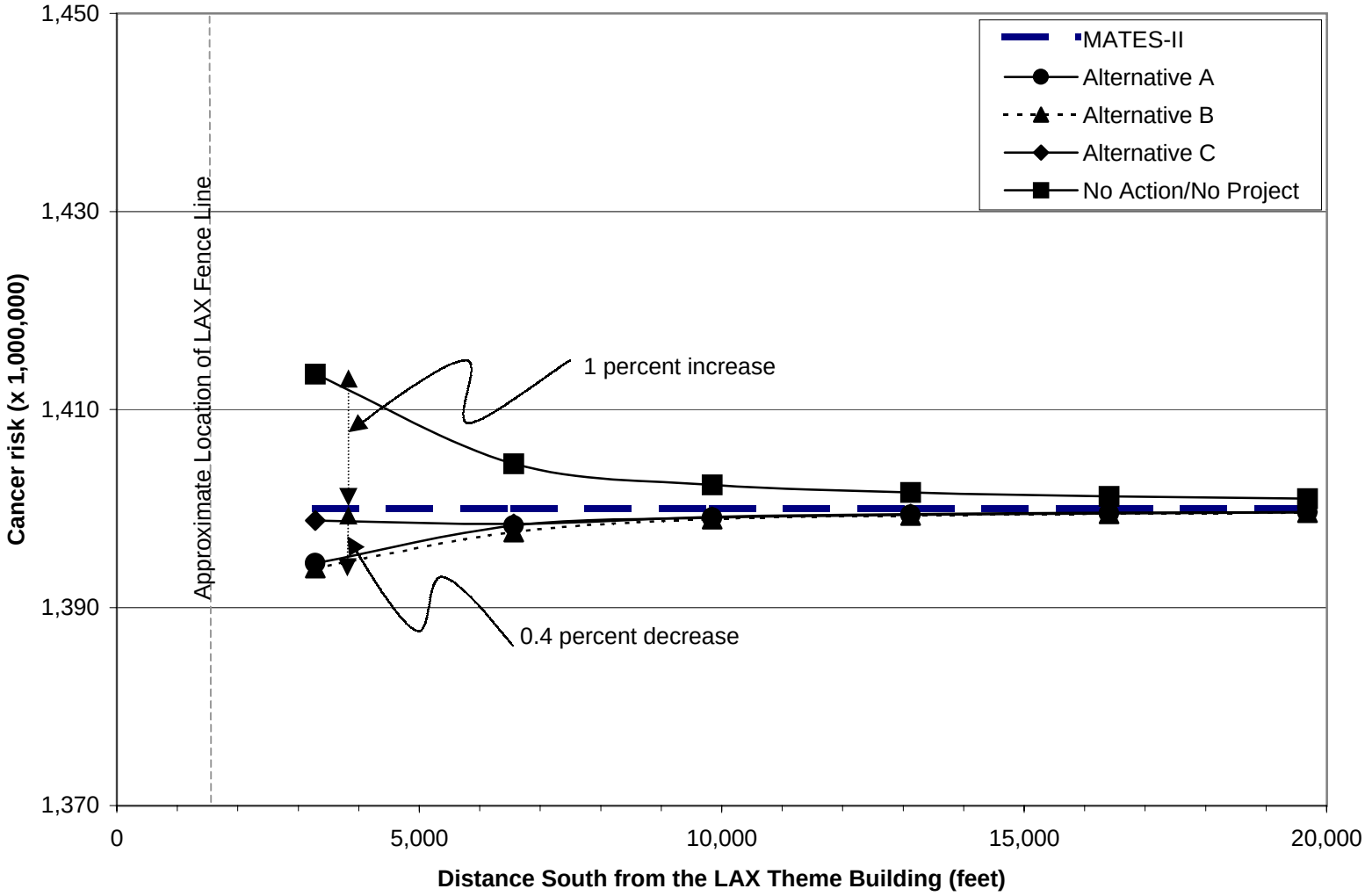
k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig ENE_pre

**Graph E-3 Cancer Risks, Inclusive of Environmental Baseline,
for the Build Alternatives and the No Action/No Project Alternative,
North Projection 2015 Pre-Mitigation**



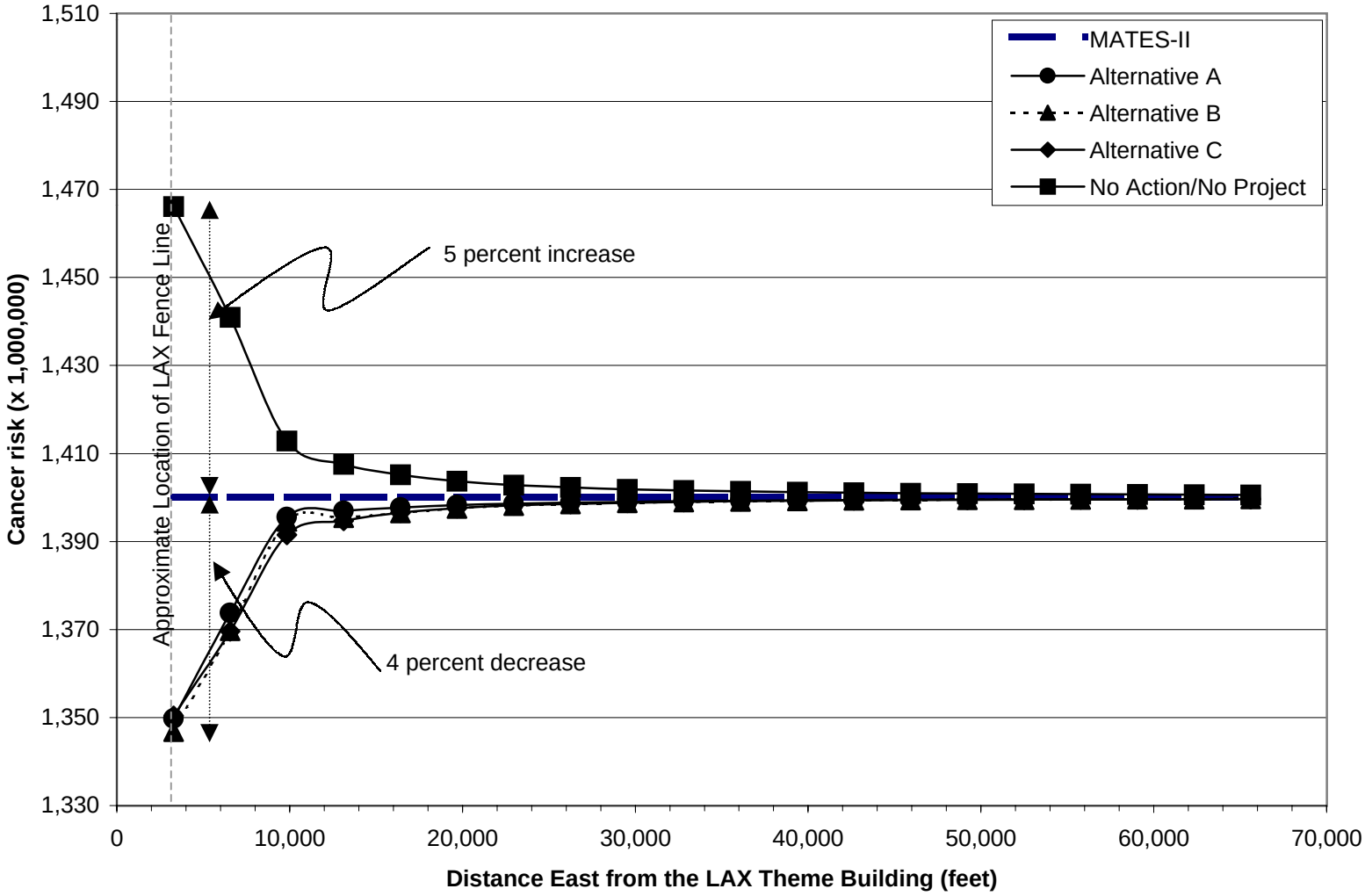
k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig North_pre

Graph E-4 Cancer Risks, Inclusive of Environmental Baseline, for the Build Alternatives and the No Action/No Project Alternative, South Projection 2015 Pre-Mitigation



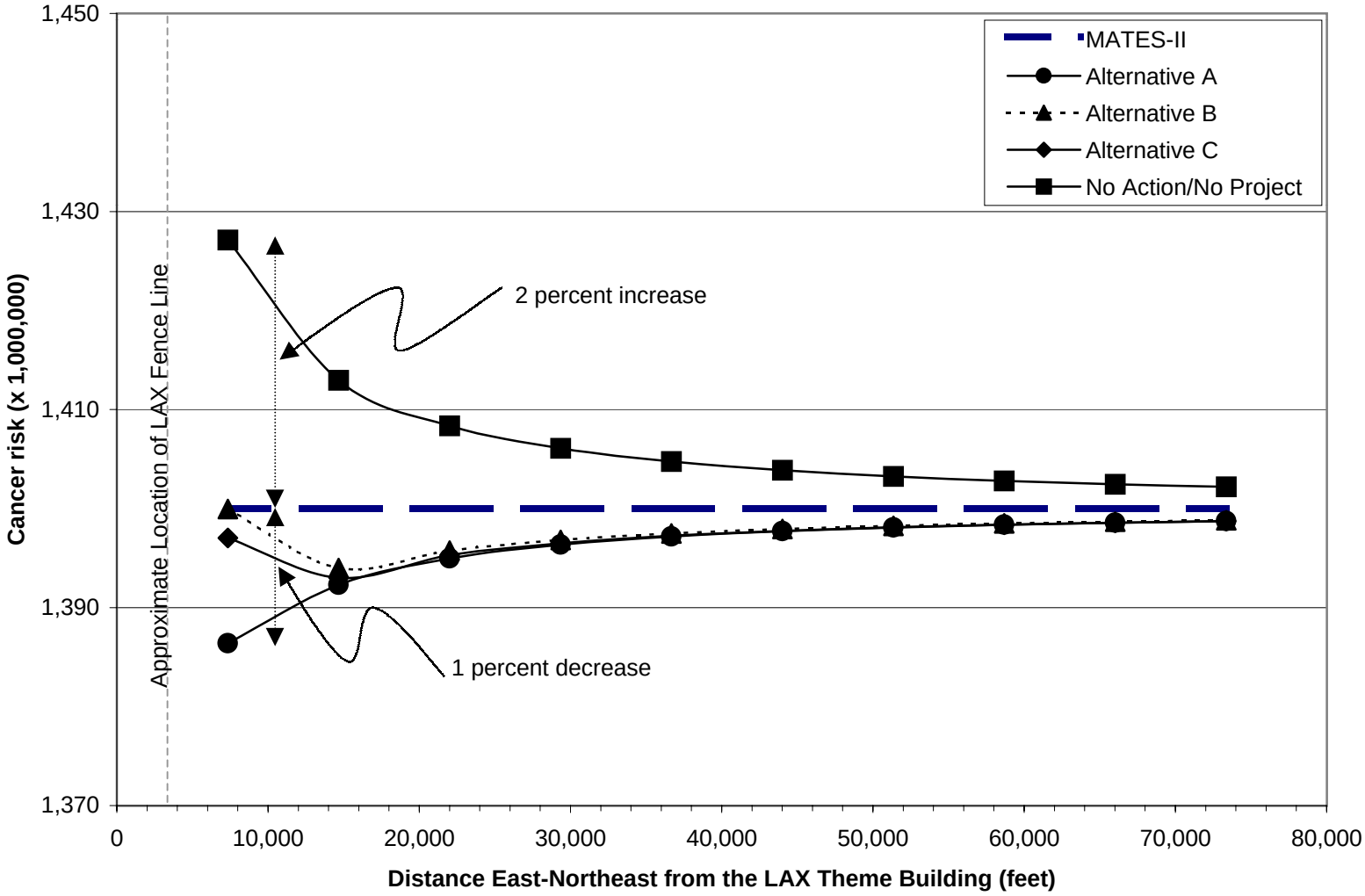
k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig South_pre

**Graph E-5 Cancer Risks, Inclusive of Environmental Baseline,
for the Build Alternatives and the No Action/No Project Alternative,
East Projection 2015 Post-Mitigation**



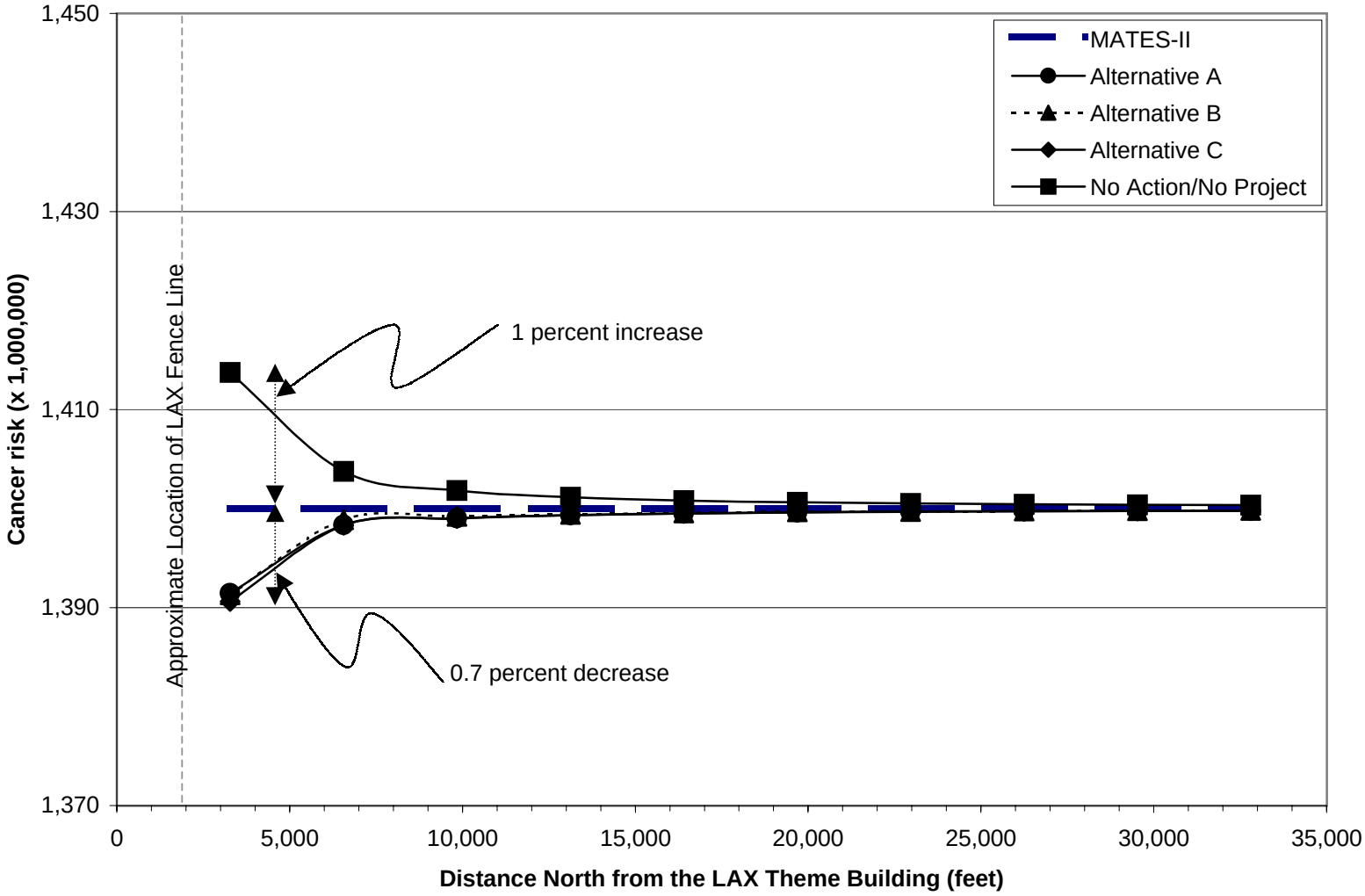
k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig East_post

**Graph E-6 Cancer Risks, Inclusive of Environmental Baseline,
for the Build Alternatives and the No Action/No Project Alternative,
East-Northeast Projection 2015 Post-Mitigation**



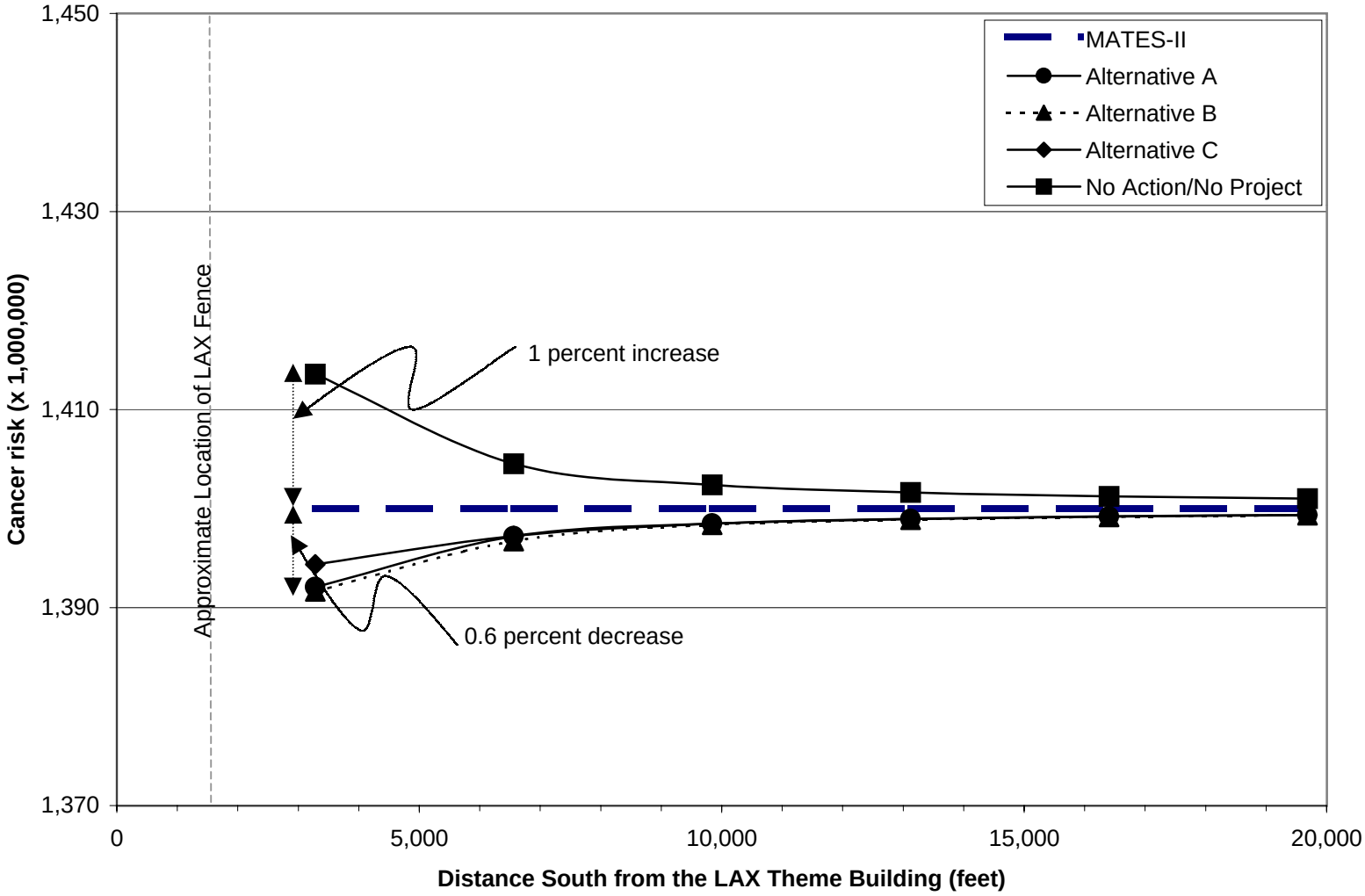
k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig ENE_post

**Graph E-7 Cancer Risks, Inclusive of Environmental Baseline,
for the Build Alternatives and the No Action/No Project Alternative,
North Projection 2015 Post-Mitigation**



k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig North_post

**Graph E-8 Cancer Risks, Inclusive of Environmental Baseline,
for the Build Alternatives and the No Action/No Project Alternative,
South Projection 2015 Post-Mitigation**



k:\8359\27571\reports_wp\health_risk\supplemental\Att E_MATES_figs\HHRAsuppattE_2015_base_figures with new titles\Fig South_post

Attachment F
Air Quality Modeling Protocol for Toxic Air Pollutants

LAX MASTER PLAN EIS/EIR
AIR QUALITY MODELING PROTOCOL FOR TOXIC AIR POLLUTANTS

1. PROJECT INTRODUCTION

The City of Los Angeles (the City) is updating the Master Plan for the Los Angeles International Airport (LAX) to identify facilities needed through the year 2015. As part of the environmental review for this project, toxic air pollutant emission inventories will be developed, dispersion modeling will be conducted, and health risks will be assessed to ensure compliance with various California environmental statutes and regulations, including AB2588 (Air Toxics "Hot Spots" Information and Assessment Act), and AB1807 (Toxic Air Contaminant Identification and Control Act of 1983), as well as the South Coast Air Quality Management District (SCAQMD) Rules 1401 (New Source Review of Toxic Air Contaminants) and 1402 (Control of Toxic Air Contaminants from Existing Sources).

This protocol identifies the assumptions and methodologies to be used in conducting the toxic air pollutant[1] health risk assessment for the proposed Master Plan combined Environmental Impact Statement/ Environmental Impact Report (EIS/EIR). Section 2 presents the approach for describing existing ambient air quality. Section 3 presents the approach for developing appropriate emission inventories. Section 4 presents the approach for conducting the air dispersion modeling. Section 5 presents the general methodology used to assess the health risks. Section 6 provides the references used to develop this protocol.

Much of the basic methodology for developing air pollutant emission inventories and conducting air dispersion modeling has been presented in the "Air Quality Modeling Protocol for Criteria Pollutants," current revision dated February 2000. In cases where the methods and assumptions for modeling toxic air pollutants are identical to those for criteria pollutants, the reader will be referred to the criteria pollutant protocol for a detailed discussion of the methods and assumptions.

2. EXISTING AMBIENT AIR QUALITY (ENVIRONMENTAL BASELINE)

The SCAQMD recently published the results of its Multiple Air Toxics Exposure Study Part II (MATES-II)[2] project which monitored and modeled toxic air pollutant concentrations at 10 fixed sites and 14 microscale sites throughout the South Coast Air Basin (SCAB) mainly during 1998. As part of the MATES-II study, microscale monitoring was conducted at the Hawthorne location (SCAQMD Monitoring Station No. 094, Southwest Coastal Los Angeles County) for 4 to 6 weeks during each of the four calendar seasons. The Hawthorne station is located approximately 2.4 miles (3.8 kilometers) southeast of the LAX Theme Building. The MATES-II data from this station will be used as the primary source for describing the existing toxic pollutant ambient air quality around LAX. This information may be supplemented by other data in the MATES-II report as well as by monitored toxic air pollutant data collected through 1996 at North Long Beach (SCAQMD Monitoring Station No. 072) and downtown Los Angeles (SCAQMD Monitoring Station No. 087) (CARB 1997a).[3] These two sites represent the multi-year air toxic monitoring stations nearest to LAX.

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1 Toxic air pollutants include those pollutants that the U.S. Environmental Protection Agency has identified as Hazardous Air Pollutants under Section 112 of the Clean Air Act, and those pollutants that the California Environmental Protection Agency has identified as Toxic Air Pollutants. This protocol will limit the extent of the health risk assessment to those toxic air pollutants for which the California Office of Environmental Health Hazard Assessment has developed unit risk factors or reference concentrations. A separate protocol titled "Air Quality Modeling Protocol for Criteria Pollutants," has been developed and was submitted separately for review and approval by SCAQMD.

2 SCAQMD, Multiple Air Toxics Study in South Coast Air Basin (Mates-II), November 1999.

3 California Air Resources Board (CARB), California Air Quality Data, Toxics Air Quality Data, http:/www.arb.ca.gove/aqd/toxics.htm [2000].

3. EMISSION MODELING AND INVENTORY PREPARATION

The following subsections present the general approach for developing toxic air pollutant emission inventories, including the sources of information, the chemicals of potential concern for airport operations, and approach for calculating emission inventories.

3.1 Emission Estimating References and Models

A variety of reference materials, primarily from California and U.S. regulatory agencies, will be used to calculate toxic air pollutant emissions from the various airport sources. Most of the organic toxic air pollutant emission factors are based on the volatile organic compound (VOC) emissions. In addition, certain elemental, semi-volatile, and non-volatile toxic emission factors are based on the particulate matter emissions. Therefore, VOC and particulate matter emissions will be estimated using methodology presented in "Air Quality Modeling Protocol for Criteria Pollutants." The references for toxic air pollutant emission factors include:

- California Air Toxics Emission Factors (CATEF) Database[4]
- VOC/PM Speciation Data System (SPECIATE) Database[5]
- U.S. EPA Memorandum, Re: Source Identification and Base Year 1990 Emission Inventory Guidance for Mobile Source HAPs on the OAQPS List of 40 Priority HAPs[6]
- Motor Vehicle-Related Air Toxics Study[7]
- CEQA Air Quality Handbook[8]
- Appropriate source test data.

In addition, emission factor data presented in peer-reviewed technical literature may be used if such data is appropriate and applicable to the LAX Master Plan alternatives, and such data will provide a more accurate estimate of LAX emissions. All such references will be discussed and emission factors justified. In cases where different emission factors in different reference documents are found for the same emission source, the reference most appropriate for operations in Southern California will be used.

3.2 Chemicals of Potential Concern

For this protocol, identification of toxic air pollutants is based on the following:

- California AB1807,
- California AB2588,
- SCAQMD Rules 1401 and 1402, and
- Clean Air Act, Section 112 (Hazardous Air Pollutants)

Air quality modeling will be conducted for those toxic air pollutants listed in the above statutes and regulations for which emission factors have been developed, and which are expected to be emitted from airport-related sources. The following processes will be used to identify sources of TAPs at LAX and to select those TAPs that may be of concern for impacts to human health.

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4 California Air Resources Board, 1997b. "California Air Toxics Emission Factors Database User's Manual, Version 1.2," 1997, CARB, Sacramento, CA.

5 U.S. Environmental Protection Agency, 1993a. "Volatile Organic Compound (VOC) / Particulate Matter (PM) Speciation Data System (SPECIATE) User's Manual, Version 1.5," February 1993, U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC.

6 U.S. Environmental Protection Agency, 1997a. Memorandum from Rich Cook to Anne Pope, Re: "Source Identification and Base year 1990 Emission Inventory Guidance for Mobile Source HAPs on the OAQPS List of 40 Priority HAPs," June 11, 1997, U.S. EPA, Office of Mobile Sources, Ann Arbor, MI.

7 U.S. Environmental Protection Agency, 1993b. "Motor Vehicle-Related Air Toxics Study," April 1993, U.S. EPA, Office of Mobile Sources, Ann Arbor, MI.

8 South Coast Air Quality Management District, 1993a. "CEQA Air Quality Handbook," November 1993, SCAQMD, Diamond Bar, CA.

Sources of TAPs

Many potential TAP emission sources are associated with current LAX conditions (**Table 1**). Each of these potential sources is associated with emissions of a variety of chemicals, many of which appear on one or more lists of TAPs identified as “of concern" by California or federal agencies. The LAX Master Plan falls under the jurisdiction of the three California statutes and regulatory programs described earlier, all of which list TAPs of concern.

Table 1

Sources of TAPs at Los Angeles International Airport

Stationary Sources (area and point)	Mobile Sources
Aircraft maintenance facilities	Aircraft
Existing and planned tank farms	On-airport vehicles
Parking facilities	Off-airport vehicles
Central Utilities Plant	Ground support equipment

Selection of TAPs of Concern

Only a subset of chemicals possibly released during airport operations will pose a threat to worker and users of the airport, or to people living, working, recreating or going to school in communities surrounding LAX. This subset of TAPs, the list of toxic air pollutants to be evaluated in detail as part of the air dispersion modeling and subsequently the Human Health Risk Assessment (HHRA), will be identified first by comparing TAPs on regulatory lists with lists of TAPs known to be released during LAX operations (determined through emissions inventories, literature searches, and projections for the future) and removing TAPs not included in the regulatory lists from further consideration.

Next, TAPs that are likely to cause the greatest impacts, based on estimated quantities released and known toxicity, will identified. Both potential carcinogens and chemicals that cause effects other than cancer will be considered in the analyses. In all cases, toxicity criteria developed by CalEPA, which in some instances are more stringent, will take precedence over federal (USEPA) criteria. Further, since inhalation is the primary route of exposure for air toxics, toxicity criteria developed from studies of inhalation exposure will take precedence over those developed from studies of oral (by mouth) exposure. TAPs of concern for the analysis will be those that contribute at least 0.1 percent to total relative impacts, as suggested in regulatory guidance.[9]

TAPs of concern will also be selected based on toxicity criteria recently proposed by CalEPA, but not yet adopted for use in risk assessment. The criteria, reference exposure levels (RELs), are based on potential for chemicals to cause non-cancer effects after exposure via inhalation. The RELs will be used in this assessment, along with established criteria from USEPA, to ensure that the HHRA will stay current if CalEPA eventually adopts RELs for use in human health risk assessment while not required regulations, this approach is thought appropriate to protect the long-term utility of the analysis.

All methods and procedures will be completely documented in a supplemental reference document for the HHRA entitled, *Screening Human Health Risk Assessment for the LAX Master Plan EIS/EIR.*

3.3 Emission Calculations

Many of the toxic pollutant emission factors are given in units of {mass of toxic per mass of VOC} or {mass of toxic per mass of particulate matter}. Even when the reference document presents the factor in absolute units, such as {mass of toxic per mile traveled} or {mass of toxic per combustion heat rate}, the factor can be converted a relative basis if the absolute VOC or particulate matter emission factor is also known. Therefore, the emission factors for toxic air pollutants will be either obtained from the references in a relative format {mass of toxic per mass of criteria pollutant} or converted to a relative format from the absolute toxic and criteria pollutant emission factors.

The toxic air pollutant emissions will be calculated by multiplying the appropriate criteria pollutant (VOC or particulate matter) emissions by the relative toxic pollutant emission factor. The criteria pollutant

[9] U.S. Environmental Protection Agency, 1989. "Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part A)". Interim Final. EPA/5401/1-89/002. December.

emissions will be determined using the methodologies presented in the "Air Quality Modeling Protocol for Criteria Pollutants."

4. DISPERSION MODELING

The following subsections provide the general approach to conducting the air dispersion modeling of toxic air pollutants, including model selection, meteorological data, source parameters, and receptor locations.

4.1 Model Selection

Dispersion modeling of pollutants generated by airport operations requires a model that can simulate emissions from multiple point, area, line, and volume sources. For this project, the Industrial Source Complex – Short Term (ISCST3) model will be used to calculate dispersion impacts from toxic air pollutant emissions produced by airport sources. The ISCST3 model is steady-state Gaussian dispersion model capable of examining impacts from multiple "complicated sources" simultaneously.[10] The FAA has approved the use of ISCST3 to assess toxic air pollutant emissions for the LAX Master Plan EIS/EIR.[11]

4.2 Meteorological Data

One year of hourly meteorological data provided by the SCAQMD will be used for the toxic air pollutant dispersion analysis. The one-year file consists of hourly surface and upper air data collected by the SCAQMD at LAX from March 1, 1996, through February 28, 1997. These are the same data used to model dispersion of criteria pollutants (see *Air Quality Modeling Protocol for Criteria Pollutants).*[12] The data set consists of hourly values of wind speed, wind direction, surface air temperature, Pasquill-Gifford stability class, and mixing heights. The data set will be used to produce conservative estimates of annual pollutant concentrations in the vicinity of LAX. For air toxic pollutants, long-term chronic exposure is appropriately evaluated for the levels of pollutants commonly observed in the LA Basin. Annual average air concentrations are appropriate for analysis of chronic exposures. The risk analysis will be similar to that developed by SCAQMD for the Mates-II.

4.3 Source Parameters

The emission rates for sources will be developed using the general approach discussed in Section 3. The source type (point, area, line or volume), size or dimension, plume rise, temporal factors, and initial dispersion coefficients will be determined using the methodology presented in the "Air Quality Modeling Protocol for Criteria Pollutants."

4.4 Receptor Locations

Pollutant concentrations produced by airport sources will be predicted at sufficient receptor locations to identify the maximum ambient air quality impacts at potential worker locations on-airport as well as at publicly accessible areas near the airport. A coarse cartesian (rectangular) coordinate grid system with a grid spacing of 250 meters over the study area will be used to develop concentration contours. In addition, fine grid systems with a grid spacing of 100 meters will be placed at locations of concentration maximums identified from the course grid modeling results. The overall extent of the modeling region will include those areas expected to have a residential toxic health risk of greater than one in one million.

Discrete receptors will be placed at specific locations of regulatory or community concern. These sensitive receptors will include schools, hospitals, nursing homes, and day-care. In addition, discrete receptors will be placed at the deposition monitoring station locations and on-site air quality monitoring station location. The sensitive or discrete receptor locations will likely include:

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[10] U.S. Environmental Protection Agency, 1995a. "User's Guide for the Industrial Source Complex (ISC3) Dispersion Models," EPA-454/B-95-003a, September 1995, U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC.

[11] Landrum & Brown, 1997a. LAX EIS/EIR Meeting Summary, November 24, 1997, FAA Headquarters, Washington DC.

[12] Camp Dresser McKee Inc., Air Quality Modeling Protocol for Criteria Pollutants, February 2000.

List of Sensitive Receptors to be Included in the HHRA Analysis

Acacia Baptist School	K-Anthony's Middle School
Arena High School	Kelso Elementary School
Bennet-Kew Elementary School	Kentwood Elementary School
Boulah Payne Elementary School	Klokke Corp.
Buford Elementary School	L.A. Southside Christian Church
C & H Health Care	Lennox Middle School
Carewest Nursing Center	Lindgren Partnership 1
Catholic Healthcare West Southern California	Loyola Village Elementary School
Center Street Elementary School	Moffet Elementary School
Centinela Elementary School	Morningside High School
Centinela Hospital Medical Center	Morningside United Church of Christ
Centinela Valley Care Center	Mount Zion Baptist Church of Los Angeles
Century Park Elementary School	Musical Hart Evangelistic Assn Inc
Chabad of the Marina	Oak Street Elementary School
Clyde Woodworth Elementary/Albert Monroe Middle School	Orville Wright Junior High School
Cowan Avenue Elementary School	Paseo del Rey Magnet School
Crippled Children's Society of	Robert F. Kennedy Medical Center
Crozier Middle School	Saint Anthony's Catholic School
Daniel Freeman Memorial Hospital	Saint Erne Healthcare Center
Desco Health Care Inc	South Bay Lutheran High School
El Segundo High School	St Eugene's Catholic School
El Segundo Middle School	St Joseph's Catholic Church School
Escuela de Montessori	St Mary's Academy of L.A.
Eucalyptus School	St. Anastasia School
Faith Lutheran Church School	St. Bernard High School
Felton Elementary School	Terrace Inglewood Brierwood
Golden West Convalesent Hospital	Trinity Lutheran Church of Hawthorne
Hawthorne Convalescent Center	Urban Healthcare Project Inc
Hawthorne High School	Visitation Catholic School
Hillcrest Continuation School	Warren Lane Elementary School
Hilltop Christian School	Washington School
Hudnall Elementary School	Westchester High School and Magnet Center
Imperial Avenue School Special Education Facility	Westchester Lutheran Church
Ingelwood Christian School	Westpoint Heights Elementary School
Inglewood High School	Whelan Elementary School
Jefferson Elementary School	Worthington Elementary School
Juan de Anza Elementary School	York School

This list of sensitive receptors was derived from a survey of the area surrounding LAX completed in 1995.[13] The HHRA will be based on an updated list of sensitive receptors.[14]

4.5 Model Output

The ISCST3 output will include annual average concentrations of the modeled toxic air pollutants for evaluation of health risks on and around the airport. Key pollutant concentrations will be presented graphically as isopleths (lines of constant concentration) interpolated from the grid point concentrations. In addition, estimated toxic air pollutant concentrations will be calculated for the discrete receptor locations described in Section 4.4 and presented in tabular format.

5. HEALTH RISK ASSESSMENT

The objective of the HHRA will be to determine the increased risk, if any, associated with the Master Plan, for people working at or using the airport, and to people living, working, going to school in communities near the airport. Existing sources of TAPs at LAX will be used as a baseline to estimate the impact of projected increases in airport activity in the future. Potential impacts of LAX development under the Master Plan and under a No Action/No Project Alternative will be assessed through a comparison of incremental air quality impacts relative to baseline conditions in 1996. This year will be used as the baseline because it is the most recent year for which adequate data are available. Further, the broader scale impacts of LAX, on air quality, both with and without implementation of the Master Plan, will be

[13] Planning consultants Research, Technical Memorandum – Schools, for the LAX Master Plan – Phase I, January 4, 1996.

[14] An updated list of sensitive receptors used in the HHRA is included in Technical Report 1, *Land Use.*

evaluated using data collected for the Multiple Air Toxics Exposure Study II (MATES II) recently completed by SCAQMD.

HHRAs can be performed at differing levels of detail, ranging from "screening" level assessments to complex formal evaluations. The approach for evaluation of potential human health risks will use both screening and formal evaluations to first focus the assessment on the most important TAPs and exposure pathways, and then to evaluate these exposures and TAPs in detail. The HHRA will follow State and Federal guidance for performance of risk assessments. The general approach to this analysis is identified below and summarized in the following subsections:

- Screening-level air dispersion modeling for initial determination of areas of potential impact
- Analysis of exposure pathways of concern for TAPs emitted during LAX operations
- Assessment of toxicity for all TAPs of concern to ensure that the most recent toxicity information is used in the HHRA.
- Characterization of potential incremental human health impacts for individual TAPs of concern, using refined air dispersion modeling to estimate potential air concentrations
- Comparisons of relative impacts of No Action/No Project alternative and Master Plan alternatives
- Evaluation of cumulative impacts within the Los Angeles basin
- Evaluation of effectiveness of mitigation on incremental human health impacts.

5.1 Analysis of Exposure Pathways and Identification of Exposure Areas

Exposure caused by breathing in toxic air pollutants will be assumed for the risk assessment to be a potentially important exposure pathway. Human health risks from inhalation exposure will be quantitatively evaluated.

Other exposure pathways involving deposition of some TAPs onto soils, and subsequent exposure via incidental ingestion of this soil, uptake from soil into homegrown vegetables, and other indirect pathways, will be analyzed in a two-step process. Only non-volatile TAPs of concern will be considered in this latter analysis because volatile chemicals, such as acrolein, benzene, and 1,3-butadiene, will not efficiently deposit onto soils.

In the first step of the process, screening-level air dispersion modeling will be used to determine potential TAP concentrations in air. In the second step, possible soil concentrations of TAPs will be estimated using a conservative deposition rate, and then these soil concentrations will be compared to urban background concentrations. Where this comparison shows that quantities of TAPs deposited onto soil would be minimal or negligible, indirect pathways associated with soil will be unlikely to contribute substantially to overall impacts of releases from LAX sources.

Areas of potential impact around LAX will be identified using the results of the selection of TAPs of concern, screening-level air dispersion modeling, and measured urban background concentrations. Benzene and 1,3-butadiene will used as representative TAPs of concern. These TAPs have been identified as chemicals likely to present a relatively high cancer risk, and for which some current background in the Los Angeles basin can be estimated from data obtained from an air sampler operating in downtown Los Angeles. Use of data from this single monitor is considered appropriate for a screening-level analysis of exposure pathways and exposure areas. More detailed analysis of exposure areas, background concentrations, and cumulative impacts will be provided in the final HHRA and will consider more comprehensive sources such as MATES-II.

The area of potential impact will be determined first by plotting the results of screening air dispersion modeling for benzene and 1,3-butadiene emissions on a map of LAX and surrounding communities. To assist in defining the air dispersion modeling domain, an initial study area will be defined as an area which extends sufficiently far from the LAX boundaries that predicted air concentrations will be only a small fraction of possible background levels at the study area boundary. "Small is not specifically defined for this analysis and professional judgement will be used. A preliminary study area will be defined only to help guide subsequent dispersion modeling, and a precisely defined area is not necessary. The study area will be reconsidered once final modeling results become available to ensure adequate analysis.

5.2 Toxicity Characterization for TAPs of Concern

Risks from exposure to TAPs will be calculated by combining estimates of potential exposure with chemical-specific toxicity criteria developed by CalEPA, USEPA, or both. The toxicity assessment initially will examine quantitative toxicity criteria for TAPs selected from regulatory lists. Appropriate criteria (mainly criteria based on inhalation exposure, unless only criteria based on ingestion are available) will then used in the selection of TAPs of concern. Subsequently, for each TAP of concern, the basis for toxicity criteria will be examined and toxicity profiles developed to provide documentation for individual criteria. Finally, for TAPs of concern likely to be associated with the highest risks (likely to be chemicals such as benzene, 1,3-butadiene, acrolein diesel particulates), additional review of the most recent toxicity information will be performed and this information added to the toxicity profiles to supplement information from regulatory agencies.

In the risk characterizations for the HHRA, toxicity criteria will be used in the calculation of quantitative risk estimates for inhalation of TAPs of concern in emissions from LAX. If needed, oral criteria will be used to evaluate potential exposure to TAPs that deposit onto soil and are subsequently ingested. Information in the toxicity profiles will also be used to assess confidence in the final assessment. Interpretation of risk results in the final HHRA will be based on both quantitative risk results and an analysis of uncertainties (confidence) in these results.

5.3 Characterization of Human Health Risks

Concentrations of TAPs of concern in air, locations of potentially exposed populations, including locations for maximally exposed individuals (MEI) exposure scenarios (worker, resident, student), and toxicity criteria will all be used to calculate incremental human health risks associated with the No Action/No Project alternative and the three Master Plan alternatives. Incremental risks will be calculated for years 2005 and 2015 using standard exposure and risk equations for estimation of inhalation risks, and for other exposure pathways if necessary. Further, incremental risks will be calculated for the existing Master Plan and for the Master Plan after mitigation measures are implemented. Risks for people recreating near the airport will be lower than those for workers, residents, and students, and no risks will be calculated for this human population. If risks are not significant as defined by CEQA thresholds for other receptor groups, risks for recreators near LAX will also be insignificant. Specific thresholds of significance for cancer and noncancer risks/hazards will be developed as part of the CEQA Environmental Impact Report (EIR).

To determine whether releases of TAPs during airport operations according to the LAX Master Plan would be significant, incremental human health risks for each of the three Alternatives will be compared to appropriate thresholds of significance identified in SCAQMD or CalEPA guidance or policy. These comparisons will focus on specific risk thresholds such as ten in one million cancer risk or a hazard index of 5. Differences in incremental human health impacts among the three Master Plan alternatives and the No Action/No Project alternative will provide a quantitative assessment of the relative impacts among build alternatives and between build and no build options. Human health impacts will also be compared with data on possible human health impacts of TAPs in the Los Angeles basin as determined in the MATES II. These latter comparisons will provide a quantitative estimate of the cumulative impacts of the Master Plan on air quality and human health risks associated with TAPs of concern within the Los Angeles Basin.

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