Arizona Department of Environmental Quality

Phoenix, Arizona

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Arizona Hazardous Air Pollution Research Program Volume 2: Findings Appendices

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Volume 2: Findings

Appendices

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CONTENTS

APPENDICES

A HAP INVENTORY REFINEMENTS
- A-1 POINT SOURCE SPECIATION PROFILES
- A-2 AREA SOURCE SPECIATION PROFILES
- A-3 SPATIAL DATA

B HAZARDOUS AIR POLLUTANT SCREENING TABLES

C ATMOSPHERIC SIMULATION MODEL ADJUSTMENT

D ESTIMATION OF ANNUAL AVERAGE HAP CONCENTRATIONS IN PAYSON

E GEOGRAPHIC DISTRIBUTIONS OF ESTIMATED COI CONCENTRATION

F RISK ASSESSMENT
- F-1 DOSE RESPONSE PROFILES FOR ELEMENTS, VOLATILE ORGANIC COMPOUNDS, POLYNUCLEAR AROMATIC HYDROCARBONS, PESTICIDES, PLUS ECAO RISK ASSESSMENT ISSUE PAPER
- F-2 PROBABILITY DISTRIBUTIONS

G EMISSION ESTIMATES BY SOURCE CATEGORY PHOENIX STUDY AREA, TUCSON STUDY AREA, CASA GRANDE STUDY AREA, AND PAYSON STUDY AREA

H APPLICATION OF THE CHEMICAL MASS BALANCE RECEPTOR MODEL TO APPORTIONMENT OF HAPS
- H-1 SOURCE CONTRIBUTION BY SPECIES

I ESTIMATION OF FUTURE HAP EMISSIONS

APPENDIX A

HAP INVENTORY REFINEMENTS

TABLE OF CONTENTS

	Page
INTRODUCTION	1
POINT SOURCE REFINEMENTS	2
ADEQ Survey Data	3
Copper Mining and Milling	3
AREA SOURCE REFINEMENTS	3
Estimation of HAP Emissions from Off-Highway Mobile Sources	6
Estimation of HAP Emissions from Degreasing	6
Estimation of HAP Emissions for Graphic Arts	7
Estimation of Chloroform Emissions from Swimming Pools	7
Estimation of Chloroform Emissions from Wastewater Treatment/POTWs	8
Estimation of Chloroform Emissions from Domestic Water Consumption	8
INVENTORY UNCERTAINTIES	8
Point Source Data Quality Limitations	9
Area Source Data Quality Limitations	10
Motor Vehicle Data Quality Limitations	11
REFERENCES	12

ATTACHMENT A-1: Point Source Speciation Profiles

ATTACHMENT A-2: Area Source Speciation Profiles

ATTACHMENT A-3: Spatial Data

INTRODUCTION

A refined emissions inventory has been developed as part of the Arizona Hazardous Air Pollution (HAP) Research Program. This appendix documents the refinements to the initial inventory. Development of the initial inventory is described in Appendix C of Volume I.

The HAP emission estimates for the Research Program were developed using currently available data with commonly accepted estimating techniques. Emissions of HAPs were estimated in one of four ways:

- Data were gathered directly from the Arizona Department of Environmental Quality (ADEQ) and county Air Pollution Control Districts (APCDs);
- HAP emission estimates were obtained from the U.S. Environmental Protection Agency (EPA) as part of the federal community-right-to-know laws;
- Activity or throughput data were used with HAP emission factors developed for specific source types; and
- Speciation profiles were applied to particulate matter (PM) or volatile organic compounds (VOC) to estimate the fraction of these emissions that are HAPs. Region-specific profiles were developed from local information, such as ambient air monitoring data. When better information was not available, average or "generic" speciation profiles for individual categories of sources were used.

Development of the final inventory consisted of refining emission estimates for certain source categories and adding new categories. These changes affected the initial emission estimates for the point and area sources. No changes were made to the on-road motor vehicle emission estimates. The changes to the point and area emissions estimates are described below, followed by a qualitative discussion of the uncertainty in the emission estimates.

This appendix also contains several attachments. Unique speciation profiles applied in the emission calculations are shown in Attachments A-1 and A-2 for point and area sources, respectively. Attachment A-3 contains numerous plots illustrating the spatial distribution of the emissions.

POINT SOURCE REFINEMENTS

Point source emissions data for this inventory were obtained from four primary sources:

- A survey of selected facilities by the Arizona Department of Environmental Quality (ADEQ);
- Permit files maintained by local air pollution control districts (APCDs);
- The community-right-to-know reporting (i.e., EPCRA Section 313) stored in the U.S. EPA's toxic release inventory (TRI) database; and
- The Grand Canyon Visibility Transport Commission (GCVTC) inventory database, developed by Radian and containing state-derived data and 1990 U.S. EPA Interim Inventory data (Dickson et al., 1995).

In general, the ADEQ and APCD data are the most detailed and up-to-date data. They were therefore preferentially used when emissions from a given facility appeared in more than one data source. The TRI database was the next preferred source. The GCVTC point source data were used in conjunction with emission factors and speciation profiles to develop HAP emission estimates for facilities with no APCD or TRI data. Furthermore, the base year for this inventory is 1993, although some of the emissions information is from other years (e.g., 1990 for GCVTC inventory data). The survey data collected by the ADEQ constitute the key refinement made to the initial point source emissions database. In addition, emission estimates were also developed for copper mining and milling operations.

Table 1 presents the distribution of point source estimation methods used in each county. The table shows the origin of emissions data for all facilities contained in each county-wide inventory.

ADEQ Survey Data

The ADEQ performed a survey of selected facilities to determine more precise estimates of HAP emissions. Many of the facilities contacted were those that have reported emissions in the TRI database. They were asked to report the actual emissions of approximately 30 compounds of interest (COI). These COI were determined from a preliminary assessment of the chemicals most likely to have impacts on human health in the Phoenix area. Special emphasis was also placed on chrome plating facilities so that emission estimates from this category of sources could be included in the inventory. Survey data were collected from 133 facilities and incorporated into the initial point source emissions database.

Copper Mining and Milling

Facility specific emission estimates were also developed for copper mining and milling operations. Emissions of HAPs from these facilities were estimated by speciating the reported PM emissions. Three separate speciation profiles were developed for copper mining and milling activities to provide more accurate estimates. The data used to develop these profiles were obtained from ADEQ. These data and the source-specific profiles used in the HAP emission estimates are included in Attachment A-1.

AREA SOURCE REFINEMENTS

The emissions database recently developed for the Grand Canyon Visibility Transport Commission (GCVTC) served as the basis for the area source emissions developed for the Arizona HAPs Research Program. In many instances, portions of the GCVTC emissions database were updated with data specific to the four study regions or with data

Table 1

Distribution of HAP Emission Estimation Methods by Number of Facilities in Each County[a]

Method	Gila	Maricopa	Pima	Pinal
ADEQ Survey	1	117	8	7
APCD Permits	0	32	1	2
U.S. EPA TRI Database	1	94	24	7
Combustion Source Emission Factor	1	7	1	4
Gasoline Speciation Profile	0	15	0	0
Copper Milling Speciation Profile	1	0	1	1
Copper Mining Speciation Profile	1	0	1	1
Generic Speciation Profiles	2	126	1	11

[a] Some facilities may have more than one emission estimation method used, due to multiple processes with different types of data reported.

believed to be of higher quality. For example, the GCVTC volatile organic compound (VOC) emissions for Phoenix were replaced with the 1990 base year ozone inventory developed by Maricopa County for the State Implementation Plan (SIP).

In addition, the GCVTC database was supplemented to account for area sources that are unique to a HAP inventory. For example, source categories were incorporated into the database to represent ethylene oxide sterilization and chloroform from chlorine use in swimming pools.

Due to the general scarcity of HAP emission factors, the HAP emissions for most area source categories were determined by applying a speciation profile to VOC or PM emission estimates. This estimation method is especially common for Pinal and Gila counties. Specific HAP emission factors were applied to a few source categories, but this usually occurred for source categories that emitted only a single HAP (e.g., chloroform from swimming pools).

Following the development of the initial emission estimates, several refinements and additions were made to the area source emissions database:

- More refined speciation data were developed for several categories of off-road mobile sources;
- The emission estimates for degreasing were refined;
- The chloroform emission estimates for swimming pools were refined;
- Chloroform emissions from wastewater treatment were estimated and added to the emissions database; and
- Chloroform emissions from domestic water use were estimated and added to the emissions database.

These changes to the initial emissions database are described below in greater detail.

Estimation of HAP Emissions from Off-Highway Mobile Sources

As described in Appendix C to Volume 1, updated VOC emissions from the Nonroad Engine Emission Inventory developed by Energy and Environmental Analysis (U.S. EPA, 1991a) were obtained and used to replace GCVTC emission estimates for gasoline (2- and 4-stroke) and diesel off-highway vehicles and equipment in Maricopa County. The GCVTC emission estimates for the other three counties were unchanged.

VOC emissions from diesel-powered farm equipment and 2- and 4-stroke gasoline-powered lawn and garden equipment were initially speciated using generic profiles. For the final emissions database, source-specific profiles were developed from engine exhaust sampling data prepared for the California Air Resources Board (ARB) (Censullo, 1991). These profiles are included in Attachment A-2 and specifically represent HAP emissions from lawn and garden equipment and diesel powered farm equipment. The remaining VOC and PM emissions from other diesel-powered equipment were speciated using source-specific speciation profiles developed by the Desert Research Institute for on-road diesel motor vehicles. VOC and PM emissions from other gasoline-powered equipment were speciated using a generic SPECIATE profile.

Estimation of HAP Emissions from Degreasing

Special refinements were also performed for degreasing solvent emissions. The refined emissions for degreasing were calculated by applying the per employee emission factor in the U.S. EPA Procedures Document (U.S. EPA, 1991b) to total employment for applicable Standard Industrial Classification (SIC) codes. Employment data for each county were obtained from *County Business Patterns*. These county-wide hydrocarbon emissions were then speciated based on national degreasing solvent usage data presented in the Procedures Document. The speciation profile for applicable HAPs can be found in Attachment A-2.

Estimation of HAP Emissions for Graphic Arts

The VOC emissions for the graphic arts area source category are based on the Maricopa County ozone SIP inventory (Maricopa County APCD, 1993), Pima County permit data (La Grange, 1995), and the GCVTC inventory. The HAPs speciation profile is based the national average composition of printing inks that is included in the U.S. EPA Procedures Document (U.S. EPA, 1991b). The percentages of mineral spirits and naphtha calculated from this speciation profile were speciated further with a mineral spirits/naphtha speciation profile that was taken from the California ARB's *Speciation Manual* (ARB, 1991). This combined speciation profile is included in Attachment A-2.

Estimation of Chloroform Emissions from Swimming Pools

Given the initially large contribution of chloroform from swimming pools, additional analysis and refinement was performed for this source category. The emission estimating technique for this source category requires the application of a surface flux value. To refine the initial estimate, both the surface area of existing pools and the flux were evaluated. The final value for total swimming pool surface area in Maricopa County was estimated by ADEQ to be approximately 1.055×10^7 square meters (ADEQ, 1995). The total swimming pool surface area in Pima County was calculated using an estimate of 32,000 pools county-wide (Ragel, 1995) and an assumed surface area of 600 square feet per pool. It was assumed that there are a minimal number of pools in Pinal and Gila Counties. A weighted chloroform flux rate of 22.3 $\mu g/m^2$-min (Rogozen et al., 1988) was then applied to the total surface area. This chloroform flux rate was developed from source sampling results in southern California, and it has been used in chloroform emission inventories by the California ARB and others. This flux also is in good agreement with unpublished data collected by Radian Corporation for a pool in northern California.

Estimation of Chloroform Emissions from Wastewater Treatment/POTWs

Chloroform emissions from wastewater treatment facilities were developed and added to the final emissions database. Chloroform was estimated by applying an emission factor to Publicly Owned Treatment Works (POTWs) wastewater throughput. Wastewater throughput data were provided by ADEQ (Broussard, 1995). Chlorination of industrial wastewater at private industrial facilities is not included in this estimate.

After aggregating wastewater throughput for each of the four counties, an annual chloroform emission factor of 12.85 lbs/million gallons per day wastewater throughput was applied. This emission factor was derived from sampling data at the Hyperion Wastewater Treatment Plant in Los Angeles (Radian, 1990). The sampling data used to derive the above emission factor were limited to chloroform emissions from the treatment plant aeration basins. Among various wastewater treatment processes, aeration basins tend to be the dominant source of chloroform emissions due to the high level of water agitation.

Estimation of Chloroform Emissions from Domestic Water Consumption

An estimate of chloroform emissions from domestic water consumption was developed and added to the final emission database. Developing this estimate required calculating a per capita daily water usage rate. The United States has an estimated water usage rate of 587 liters per capita per day of which only 2 liters is actually ingested. A weighted chloroform emission factor of 45.2 μg/liter (ARB, 1990) was then applied to the yearly water usage rates in each of the four counties.

INVENTORY UNCERTAINTIES

The emission estimates contained in the final emissions database were developed using currently available data with commonly accepted estimating techniques. Nonetheless, the resulting emission estimates should be considered approximations of the actual emissions in the four study regions. The emission estimates were developed to

facilitate the overall risk assessment conducted as part of the Arizona HAP Research Program. As discussed below, there is an important degree of uncertainty in some of the emission estimates for point, area, and motor vehicle sources.

Point Source Data Quality Limitations

Uncertainty in the point source inventory results from missing sources and the use of generic data to estimate emissions from some facilities. Much of the uncertainty in the point source inventory results in an underestimation of point source emissions, which is primarily due to the limited point source data available at this time. The point source database could be enhanced by conducting additional survey efforts to more thoroughly collect facility specific information.

Specific causes of uncertainty in the point source emission estimates include the following:

- **Underestimation of solvent emissions** - Limited data were available to assess HAP emissions from industrial, commercial, and institutional sources. We believe that reasonable estimates have been developed for combustion and fuel handling and distribution sources. Data from a limited survey, local permits, and community-right-to-know submissions were used to estimate point source solvent emissions. These three information sources only include large facilities, creating a potential underestimation bias in the point source solvent emissions.

- **Surface coating emissions** - Application of paints or coating materials may result in HAP emissions either through solvent evaporation or through overspray resulting in various pigments being emitted to the atmosphere. Many of the solvents contained in paints are considered HAPs. In addition, some of the pigments, such as hexavalent chromium, are also considered HAPs. A detailed survey would be needed to better characterize these emissions.

- **Permit and TRI data for point sources** - A significant portion of the HAP emissions from point sources in the Phoenix study region were obtained from either permit files or the U.S. EPA TRI database. In many cases, these sources of data list maximum allowable estimates, not actual emissions. The emissions of the reported chemicals from these data sources are therefore likely to be overestimates of the actual emissions.

- **Use of speciation profiles** - For many source types, criteria pollutant emissions (i.e., PM and VOC) were speciated to yield estimates of the HAP emissions. Regional-specific profiles were available for this study for fugitive emissions of gasoline and copper mining and milling activities. The region-specific speciation approach should provide reasonable estimates of HAPs. For other source types, generic profiles were applied. Our confidence in the emission estimates generated from the generic profiles is much less than it is for emissions estimated using either emission factors or region-specific profiles. In addition, not all of the source types included in this inventory have profiles available. If a profile was not available for a point source, no emissions were estimated.

Area Source Data Quality Limitations

During the development of the area source emission estimates, significant effort was made to accurately depict emissions. Despite these efforts, however, some degree of uncertainty inevitably remains in these emission estimates. The following discussion highlights some of the key areas of uncertainty in the area source emission estimates:

- **Use of speciation profiles** - Numerous Arizona-specific speciation profiles were developed specifically for this inventory, but in several instances generic speciation profiles were the only source of available data. Application of generic profiles creates additional uncertainty in the HAP emission estimates.

- **Miscellaneous industrial solvent use** - The HAP emissions for this category appear quite high, especially for methanol. There are two possible reasons for this result. The first is that industrial solvent use in Arizona may be different from typical nationwide industrial solvent use. The second reason is that the per employee emission factors were based upon 1982 employment data and 1985 solvent use data. Current conditions could be quite different from the conditions that are represented by these data.

- **Chloroform emissions** - Chloroform emissions from swimming pools were based upon a chloroform flux rate that was derived from source testing performed on a single residential pool (Rogozen et al., 1988). However, the chemistry of chloroform formation is extremely complex and is affected by many factors. These factors include water temperature, water pH, ammonia concentration, precursor type and concentration, chlorine dose, reaction time, and location of chlorine

addition (Chou, 1991). Subsequent research has prompted the California Air Resources Board to establish a range of swimming pool chloroform emissions in their state inventory, with emissions calculated using the Rogozen flux rate as the upper end of the range (ARB, 1990). Therefore, actual chloroform emissions may be lower than those contained in the Arizona HAPS Research Program database. Additional sampling and research would be needed to reduce the uncertainty associated with this source category, including the seasonal variation in emissions.

- **Off-Road mobile sources** - For this analysis, readily available information that could be used to estimate polycyclic organic matter emissions from gasoline powered off-road mobile sources was not available. Including POM emissions for off-road sources would further increase the overall contribution of this category of sources to HAPs emitted in the study region.

- **Cooling towers** - Standard estimating techniques were used to develop hexavalent chromium emissions from industrial cooling towers. A complete phase-out of chromium as a corrosion inhibitor will take place in the near future as a result of U.S. EPA rules. Discussions with Maricopa County Bureau of Air Pollution Control staff indicate that little hexavalent chromium is currently being used in cooling towers. The current estimating technique, therefore, likely overestimates the emissions of this HAP from cooling towers.

It should also be noted there is an important element of uncertainty in the spatial allocation of the area source emissions. Area source emissions are estimated at the county level and then placed into approximate grid cell locations according to population, traffic, commercial, or industrial land use characteristics. The spatial disaggregation process approximates the actual spatial distribution of emissions and contains an inherent level of uncertainty.

Motor Vehicle Data Quality Limitations

There are also data quality limitations associated with the motor vehicle data used in this inventory. Possible sources of uncertainty include the following:

- **Inherent problems with MOBILE5a** - It is commonly believed that MOBILE5a tends to underestimate VOC emissions. For example,

MOBILE5a emission factors are based primarily on Federal Test Procedure (FTP) data. However, typical driving behavior does not conform with the FTP cycle. Therefore, MOBILE5a is generally unable to estimate emissions that occur outside of FTP conditions, particularly emissions during hard accelerations. Other examples of uncertainties include unregistered vehicles and "gross emitters."

- **Inherent problems with PART5** - Compared with MOBILE5a, PART5 is in the early stages of development. In the current version of the model, vehicle PM emissions are not affected by changes in vehicle speeds, temperatures, or the existence of I/M or anti-tampering programs. The inability of PART5 to model PM emissions under these variable situations suggests a higher level of imprecision.

- **Application of winter VOC speciation profile for gasoline-powered vehicles** - The sales period for oxygenated fuels was identified as October 1-March 31. The winter VOC speciation profile developed by Desert Research Institute for Phoenix includes the effects of oxygenated fuels. However, winter (as defined in this inventory) covers only the months of November through January. Thus, the non-oxygenated speciation profile is being applied to a portion of the oxygenated fuel sales period.

- **Lack of detailed transportation data for Pinal and Gila Counties** - Because of the lack of detailed transportation data, it was not possible to run MOBILE5a and PART5 for these two counties. It is thought that use of these two models with local data would give better emission estimates than the emissions contained in the GCTVC inventory database.

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ATTACHMENT A-1

POINT SOURCE SPECIATION PROFILES

Gasoline VOC Speciation Profile[a]

HAP	CAS No.	Weight Fraction of VOC
Benzene	71-43-2	0.003391
Cyclohexane	110-82-7	0.002288
Ethanol	64-17-5	0.003783
Ethylbenzene	100-41-4	0.000462
n-Hexane	110-54-3	0.009375
Naphthalene	91-20-3	0.000002
Toluene	108-88-3	0.006670
m-Xylene	108-38-3	0.001551
o-Xylene	95-47-6	0.000435
Cumene (Isopropylbenzene)	98-82-8	0.000027
Methyl tert Butyl Ether	1634-04-4	0.006923
Other Hydrocarbons		0.965093

[a] Profile developed from data supplied by DRI (1995).

Copper Mining PM_{10} Speciation Profile[a]

HAP	CAS No.	Weight Fraction of PM_{10}
Antimony compounds	7440-36-0	2.60×10^{-5}
Arsenic compounds (inorganic including arsine)	7440-38-2	1.54×10^{-4}
Beryllium compounds	7440-41-7	6.84×10^{-6}
Barium	7440-39-3	1.30×10^{-4}
Cadmium compounds	7440-43-9	2.63×10^{-5}
Cobalt compounds	7440-48-4	2.62×10^{-4}
Lead compounds	7439-92-1	5.20×10^{-4}
Manganese compounds	7439-96-5	2.00×10^{-4}
Nickel compounds	7440-02-0	7.31×10^{-5}
Selenium compounds	7782-49-2	5.60×10^{-4}
Zinc	7440-66-6	4.27×10^{-3}

[a] Profile developed from data supplied by ADEQ (1995).

Copper Milling PM_{10} Speciation Profile[a]

HAP	CAS No.	Weight Fraction (PM10)
Antimony compounds	7440-36-0	4.31×10^{-4}
Arsenic compounds (inorganic including arsine)	7440-38-2	2.06×10^{-4}
Beryllium compounds	7440-41-7	8.14×10^{-6}
Barium	7440-39-3	6.24×10^{-4}
Chromium compounds	7440-47-3	7.86×10^{-5}
Cadmium compounds	7440-43-9	7.37×10^{-5}
Cobalt compounds	7440-48-4	3.04×10^{-4}
Lead compounds	7439-92-1	4.17×10^{-3}
Manganese compounds	7439-96-5	7.50×10^{-4}
Mercury compounds	7439-97-6	1.97×10^{-6}
Nickel compounds	7440-02-0	2.18×10^{-4}
Selenium compounds	7782-49-2	1.84×10^{-3}
Zinc	7440-66-6	6.73×10^{-3}

[a] Profile developed from data supplied by ADEQ (1995).

Copper Milling VOC Speciation Profile[a]

HAP	CAS No.	Weight Fraction (VOC)
Glycol Ethers	52286-19-8	2.19×10^{-4}
Methylene Chloride (Dichloromethane)	75-09-2	2.19×10^{-4}
Methyl Ethyl Ketone (2-Butanone)	78-93-3	5.48×10^{-5}
Methyl Isobutyl Ketone (Hexone)	108-10-1	1.10×10^{-5}
Hexane	110-54-3	1.92×10^{-4}
Chlorine	7782-50-5	2.74×10^{-4}

[a] Profile developed from data supplied by ADEQ (1995).

		Profile:	Mining (PM10 only)	
		Company:	MAGMA San Manual (Mill & Concentrator)	
		Operation:	Primary Crushing Plant	
		Emissions:	Total (tpy)	fraction of PM10
HAP	Name	CAS No.		
VOC				
TSP			1.64E+01	
PM10			4.74E+00	
Sb	ANTIMONY COMPOUNDS	7440-36-0	1.23E-04	2.60E-05
AS	ARSENIC COMPOUNDS (INORGANIC INCLUDING AR	7440-38-2	7.30E-04	1.54E-04
Be	BERYLLIUM COMPOUNDS	7440-41-7	3.25E-05	6.84E-06
Ba	BARIUM	7440-39-3	6.14E-04	1.30E-04
Cr	CHROMIUM COMPOUNDS	7440-47-3	0.00E+00	0.00E+00
Cd	CADMIUM COMPOUNDS	7440-43-9	1.25E-04	2.63E-05
Cobalt	COBALT COMPOUNDS	7440-48-4	1.24E-03	2.62E-04
Pb	LEAD COMPOUNDS	7439-92-1	2.47E-03	5.20E-04
Mn	MANGANESE COMPOUNDS	7439-96-5	9.46E-04	2.00E-04
Hg	MERCURY COMPOUNDS	7439-97-6	0.00E+00	0.00E+00
Ni	NICKEL COMPOUNDS	7440-02-0	3.47E-04	7.31E-05
Se	SELENIUM COMPOUNDS	7782-49-2	2.66E-03	5.60E-04
Zn	ZINC	7440-66-6	2.02E-02	4.27E-03
AZ0009	GLYCOL ETHERS	52286-19-8	0.00E+00	
75092	METHYLENE CHLORIDE (DICHLOROMETHANE)	75-09-2	0.00E+00	
78933	METHYL ETHYL KETONE (2-BUTANONE)	78-93-3	0.00E+00	
108101	METHYL ISOBUTYL KETONE (HEXONE)	108-10-1	0.00E+00	
110543	HEXANE	110-54-3	0.00E+00	
7782505	CHLORINE	7782-50-5	0.00E+00	

		Profile:	Mill PM10	
		Company:	MAGMA San Manual (Mill & Concentrator)	
		Operation:	ALL	
		Emissions:	Total	fraction of PM10
HAP	Name	CAS No.		
VOC				
TSP			5.16E+01	
PM10			1.50E+01	
Sb	ANTIMONY COMPOUNDS	7440-36-0	4.51E-04	3.01E-05
AS	ARSENIC COMPOUNDS (INORGANIC INCLUDING AR	7440-38-2	2.66E-03	1.78E-04
Be	BERYLLIUM COMPOUNDS	7440-41-7	1.22E-04	8.14E-06
Ba	BARIUM	7440-39-3	2.22E-03	1.49E-04
Cr	CHROMIUM COMPOUNDS	7440-47-3		0.00E+00
Cd	CADMIUM COMPOUNDS	7440-43-9	9.74E-04	6.51E-05
Cobalt	COBALT COMPOUNDS	7440-48-4	4.56E-03	3.04E-04
Pb	LEAD COMPOUNDS	7439-92-1	9.04E-03	6.04E-04
Mn	MANGANESE COMPOUNDS	7439-96-5	3.50E-03	2.34E-04
Hg	MERCURY COMPOUNDS	7439-97-6	0.00E+00	0.00E+00
Ni	NICKEL COMPOUNDS	7440-02-0	1.65E-03	1.10E-04
Se	SELENIUM COMPOUNDS	7782-49-2	9.66E-03	6.45E-04
Zn	ZINC	7440-66-6	7.37E-02	4.92E-03
AZ0009	GLYCOL ETHERS	52286-19-8		
75092	METHYLENE CHLORIDE (DICHLOROMETHANE)	75-09-2		
78933	METHYL ETHYL KETONE (2-BUTANONE)	78-93-3		
108101	METHYL ISOBUTYL KETONE (HEXONE)	108-10-1		
110543	HEXANE	110-54-3		
7782505	CHLORINE	7782-50-5		

		Profile:			
		Company:	ASARCO HAYDEN CONCENTRATOR		
		Operation:	ALL		
		Emissions:	Total	fraction of PM10	Average fraction
HAP	Name	CAS No.			
VOC			0.00E+00		
TSP			2.01E+02		
PM10			6.36E+01		
Sb	ANTIMONY COMPOUNDS	7440-36-0	5.10E-02	8.02E-04	4.31E-04
AS	ARSENIC COMPOUNDS (INORGANIC INCLUDING AR	7440-38-2	3.63E-03	5.71E-05	2.06E-04
Be	BERYLLIUM COMPOUNDS	7440-41-7	0.00E+00	0.00E+00	8.14E-06
Ba	BARIUM	7440-39-3	6.05E-02	9.51E-04	6.24E-04
Cr	CHROMIUM COMPOUNDS	7440-47-3	1.00E-02	1.57E-04	7.86E-05
Cd	CADMIUM COMPOUNDS	7440-43-9	1.10E-03	1.73E-05	7.37E-05
Cobalt	COBALT COMPOUNDS	7440-48-4	0.00E+00	0.00E+00	3.04E-04
Pb	LEAD COMPOUNDS	7439-92-1	4.54E-01	7.14E-03	4.17E-03
Mn	MANGANESE COMPOUNDS	7439-96-5	6.57E-02	1.03E-03	7.50E-04
Hg	MERCURY COMPOUNDS	7439-97-6	2.50E-04	3.93E-06	1.97E-06
Ni	NICKEL COMPOUNDS	7440-02-0	1.36E-02	2.14E-04	2.18E-04
Se	SELENIUM COMPOUNDS	7782-49-2	1.52E-01	2.40E-03	1.84E-03
Zn	ZINC	7440-66-6	2.29E-01	3.61E-03	6.73E-03
AZ0009	GLYCOL ETHERS	52286-19-8			
75092	METHYLENE CHLORIDE (DICHLOROMETHANE)	75-09-2			
78933	METHYL ETHYL KETONE (2-BUTANONE)	78-93-3			
108101	METHYL ISOBUTYL KETONE (HEXONE)	108-10-1			
110543	HEXANE	110-54-3			
7782505	CHLORINE	7782-50-5			

		Profile:	Mill VOC	
		Company:	CYPRUS BAGDAD MINE & MILL	
		Operation:	ALL	
		Emissions:	Total	fraction of VOC
HAP	Name	CAS No.		
VOC			1.89E+01	
TSP			9.62E+02	
PM10			3.65E+02	
Sb	ANTIMONY COMPOUNDS	7440-36-0	0.00E+00	
AS	ARSENIC COMPOUNDS (INORGANIC INCLUDING AR	7440-38-2	0.00E+00	
Be	BERYLLIUM COMPOUNDS	7440-41-7	0.00E+00	
Ba	BARIUM	7440-39-3	0.00E+00	
Cr	CHROMIUM COMPOUNDS	7440-47-3		
Cd	CADMIUM COMPOUNDS	7440-43-9	0.00E+00	
Cobalt	COBALT COMPOUNDS	7440-48-4	3.00E-01	
Pb	LEAD COMPOUNDS	7439-92-1	0.00E+00	
Mn	MANGANESE COMPOUNDS	7439-96-5	0.00E+00	
Hg	MERCURY COMPOUNDS	7439-97-6	0.00E+00	
Ni	NICKEL COMPOUNDS	7440-02-0	0.00E+00	
Se	SELENIUM COMPOUNDS	7782-49-2	0.00E+00	
Zn	ZINC	7440-66-6	0.00E+00	
AZ0009	GLYCOL ETHERS	52286-19-8	8.00E-02	2.19E-04
75092	METHYLENE CHLORIDE (DICHLOROMETHANE)	75-09-2	8.00E-02	2.19E-04
78933	METHYL ETHYL KETONE (2-BUTANONE)	78-93-3	2.00E-02	5.48E-05
108101	METHYL ISOBUTYL KETONE (HEXONE)	108-10-1	4.00E-03	1.10E-05
110543	HEXANE	110-54-3	7.00E-02	1.92E-04
7782505	CHLORINE	7782-50-5	1.00E-01	2.74E-04

ATTACHMENT A-2

AREA SOURCE SPECIATION PROFILES

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Winter)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Highway Vehicles - Gasoline	VOC			
Light Duty Gasoline Vehicles (LDGV)				
Total: All Road Types				
		1,2-XYLENE	95-47-6	0.020893
		1,3-BUTADIENE	106-99-0	0.003692
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.021195
		ACETALDEHYDE	75-07-0	0.005654
		ACETONE	67-64-1	0.005315
		ACROLEIN	107-02-8	0.000678
		BENZENE	71-43-2	0.032135
		CROTONALDEHYDE	123-73-9	0.000452
		ETHYLBENZENE	100-41-4	0.016390
		FORMALDEHYDE	50-00-0	0.012438
		HEXANE	110-54-3	0.023858
		METHYL ETHYL KETONE	78-93-3	0.001922
		METHYLCYCLOPENTANE	96-37-7	0.016392
		NAPHTHALENE	91-20-3	0.001948
		NONANE	111-84-2	0.002442
		POLYCYCLIC ORGANIC MATTER		0.003802
		PROPIONALDEHYDE	123-38-6	0.000678
		STYRENE	100-42-5	0.002639
		TOLUENE	108-88-3	0.098304
		XYLENES (MIXED ISOMERS)	1330-20-7	0.059526
			Total	0.330353

Arizona HAPS

Source Specific Speciation Profiles (Spring)

11/22/95

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Highway Vehicles - Gasoline	VOC			
Light Duty Gasoline Vehicles (LDGV)				
Total: All Road Types				
		1,2-XYLENE	95-47-6	0.019107
		1,3-BUTADIENE	106-99-0	0.002375
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.015974
		ACETALDEHYDE	75-07-0	0.005653
		ACETONE	67-64-1	0.005314
		ACROLEIN	107-02-8	0.000678
		BENZENE	71-43-2	0.027197
		CROTONALDEHYDE	123-73-9	0.000452
		ETHYLBENZENE	100-41-4	0.014123
		FORMALDEHYDE	50-00-0	0.012437
		HEXANE	110-54-3	0.043132
		METHYL ETHYL KETONE	78-93-3	0.001922
		METHYLCYCLOPENTANE	96-37-7	0.016212
		NAPHTHALENE	91-20-3	0.002251
		NONANE	111-84-2	0.002266
		POLYCYCLIC ORGANIC MATTER		0.005004
		PROPIONALDEHYDE	123-38-6	0.000678
		STYRENE	100-42-5	0.002366
		TOLUENE	108-88-3	0.098873
		XYLENES (MIXED ISOMERS)	1330-20-7	0.055214
			Total	0.331228

Arizona HAPS

Source Specific Speciation Profiles (Summer)

11/22/95

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Highway Vehicles - Gasoline	VOC			
Light Duty Gasoline Vehicles (LDGV)				
Total: All Road Types				
		1,2-XYLENE	95-47-6	0.019107
		1,3-BUTADIENE	106-99-0	0.002375
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.015974
		ACETALDEHYDE	75-07-0	0.005653
		ACETONE	67-64-1	0.005314
		ACROLEIN	107-02-8	0.000678
		BENZENE	71-43-2	0.027197
		CROTONALDEHYDE	123-73-9	0.000452
		ETHYLBENZENE	100-41-4	0.014123
		FORMALDEHYDE	50-00-0	0.012437
		HEXANE	110-54-3	0.043132
		METHYL ETHYL KETONE	78-93-3	0.001922
		METHYLCYCLOPENTANE	96-37-7	0.016212
		NAPHTHALENE	91-20-3	0.002251
		NONANE	111-84-2	0.002266
		POLYCYCLIC ORGANIC MATTER		0.005004
		PROPIONALDEHYDE	123-38-6	0.000678
		STYRENE	100-42-5	0.002366
		TOLUENE	108-88-3	0.098873
		XYLENES (MIXED ISOMERS)	1330-20-7	0.055214
			Total	0.331228

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Fall)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Highway Vehicles - Gasoline	VOC			
Light Duty Gasoline Vehicles (LDGV)				
Total: All Road Types				
		1,2-XYLENE	95-47-6	0.019107
		1,3-BUTADIENE	106-99-0	0.002375
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.015974
		ACETALDEHYDE	75-07-0	0.005653
		ACETONE	67-64-1	0.005314
		ACROLEIN	107-02-8	0.000678
		BENZENE	71-43-2	0.027197
		CROTONALDEHYDE	123-73-9	0.000452
		ETHYLBENZENE	100-41-4	0.014123
		FORMALDEHYDE	50-00-0	0.012437
		HEXANE	110-54-3	0.043132
		METHYL ETHYL KETONE	78-93-3	0.001922
		METHYLCYCLOPENTANE	96-37-7	0.016212
		NAPHTHALENE	91-20-3	0.002251
		NONANE	111-84-2	0.002266
		POLYCYCLIC ORGANIC MATTER		0.005004
		PROPIONALDEHYDE	123-38-6	0.000678
		STYRENE	100-42-5	0.002366
		TOLUENE	108-88-3	0.098873
		XYLENES (MIXED ISOMERS)	1330-20-7	0.055214
			Total	0.331228

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Highway Vehicles - Diesel	VOC			
Light Duty Diesel Vehicles (LDDV)				
Total: All Road Types				
		1,2-XYLENE	95-47-6	0.010279
		1,3-BUTADIENE	106-99-0	0.004162
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.007131
		ACETALDEHYDE	75-07-0	0.006600
		BENZENE	71-43-2	0.019333
		BIPHENYL	92-52-4	0.000834
		ETHYLBENZENE	100-41-4	0.005457
		FORMALDEHYDE	50-00-0	0.086100
		HEXANE	110-54-3	0.011080
		METHYLCYCLOPENTANE	96-37-7	0.006134
		NAPHTHALENE	91-20-3	0.002469
		NONANE	111-84-2	0.004948
		POLYCYCLIC ORGANIC MATTER		0.016463
		STYRENE	100-42-5	0.016296
		TOLUENE	108-88-3	0.037129
		XYLENES (MIXED ISOMERS)	1330-20-7	0.020347
			Total	0.254762

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Off-Highway Vehicle Gasoline, 2-Stroke	TOG			
Lawn & Garden Equipment				
Total				
		1,2-XYLENE	95-47-6	0.030629
		1,3-BUTADIENE	106-99-0	0.002533
		1,3-XYLENE	108-38-3	0.069275
		1,4-XYLENE	106-42-3	0.013764
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.010154
		ACETALDEHYDE	75-07-0	0.000515
		ACETONE	67-64-1	0.000912
		BENZALDEHYDE	100-52-7	0.000090
		BENZENE	71-43-2	0.039200
		CYCLOHEXANONE	108-94-1	0.000013
		ETHYLBENZENE	100-41-4	0.023657
		FORMALDEHYDE	50-00-0	0.000645
		HEXANE	110-54-3	0.016388
		METHANOL	67-56-1	0.000106
		METHYL ETHYL KETONE	78-93-3	0.000212
		METHYLCYCLOPENTANE	96-37-7	0.023646
		NONANE	111-84-2	0.005220
		PENTANAL	110-62-3	0.000015
		STYRENE	100-42-5	0.003452
		TOLUENE	108-88-3	0.112134
			Total	0.352560

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Off-Highway Vehicle Gasoline, 4-Stroke	TOG			
Lawn & Garden Equipment				
Total				
		1,2-XYLENE	95-47-6	0.020262
		1,3-BUTADIENE	106-99-0	0.013541
		1,3-XYLENE	108-38-3	0.043569
		1,4-XYLENE	106-42-3	0.007961
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.004841
		ACETALDEHYDE	75-07-0	0.001619
		ACETONE	67-64-1	0.001923
		BENZALDEHYDE	100-52-7	0.000193
		BENZENE	71-43-2	0.054577
		CYCLOHEXANONE	108-94-1	0.000078
		ETHYLBENZENE	100-41-4	0.016249
		FORMALDEHYDE	50-00-0	0.013085
		HEXANE	110-54-3	0.011075
		METHANOL	67-56-1	0.001393
		METHYL ETHYL KETONE	78-93-3	0.000354
		METHYLCYCLOPENTANE	96-37-7	0.009759
		NAPHTHALENE	91-20-3	0.001952
		NONANE	111-84-2	0.005807
		STYRENE	100-42-5	0.002699
		TOLUENE	108-88-3	0.072519
			Total	0.283456

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Off-Highway Vehicle Diesel	TOG			
Farm Equipment				
Total				
		1,2-XYLENE	95-47-6	0.002501
		1,3-BUTADIENE	106-99-0	0.000152
		1,3-XYLENE	108-38-3	0.003937
		1,4-XYLENE	106-42-3	0.000755
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.002866
		ACETALDEHYDE	75-07-0	0.079715
		ACETONE	67-64-1	0.077553
		BENZALDEHYDE	100-52-7	0.005175
		BENZENE	71-43-2	0.013343
		BIPHENYL	92-52-4	0.000834
		CYCLOHEXANONE	108-94-1	0.001417
		ETHYLBENZENE	100-41-4	0.002108
		FORMALDEHYDE	50-00-0	0.153175
		HEXANE	110-54-3	0.001416
		METHANOL	67-56-1	0.000171
		METHYL ETHYL KETONE	78-93-3	0.014267
		METHYLCYCLOPENTANE	96-37-7	0.001094
		NAPHTHALENE	91-20-3	0.002469
		NONANE	111-84-2	0.002361
		PENTANAL	110-62-3	0.001458
		POLYCYCLIC ORGANIC MATTER		0.016463
		STYRENE	100-42-5	0.000651
		TOLUENE	108-88-3	0.008138
			Total	0.392019

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Surface Coating	TOG			
Architectural Coatings				
Total: All Solvent Types				
		ACETONE	67-64-1	0.032000
		CELLOSOLVE ACETATE	111-15-9	0.013000
		DIMETHYLFORMAMIDE, N,N-	68-12-2	0.005000
		ETHYLBENZENE	100-41-4	0.043000
		ETHYLENE GLYCOL	107-21-1	0.006000
		HEXANE	110-54-3	0.207000
		ISOBUTYL ACETATE	110-19-0	0.015000
		ISOBUTYL ALCOHOL	78-83-1	0.006000
		METHANOL	67-56-1	0.039000
		METHYL ETHYL KETONE	78-93-3	0.056000
		METHYL ISOBUTYL KETONE	108-10-1	0.006000
		N-BUTYL ACETATE	138-22-7	0.025000
		N-BUTYL ALCOHOL	71-36-3	0.016000
		PROPYLENE GLYCOL	57-55-6	0.008000
		TOLUENE	108-88-3	0.052000
		XYLENES (MIXED ISOMERS)	1330-20-7	0.026000
			Total	0.555000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Surface Coating	TOG			
Architectural Coatings				
Total: All Solvent Types				
		BENZENE	71-43-2	0.003000
		CHLOROETHANE	75-00-3	0.006000
		ETHYLENE GLYCOL	107-21-1	0.005000
		HEXYLENE GLYCOL	107-41-5	0.014000
		METHYL CHLORIDE	74-87-3	0.005000
		METHYLENE CHLORIDE	75-09-2	0.055000
		N-BUTYL ALCOHOL	71-36-3	0.200000
			Total	0.288000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Stationary Source Fuel Combustion				
Residential	VOC			
Wood				
Total: Woodstoves & Fireplaces				
		1,2-XYLENE	95-47-6	0.000986
		1,3-BUTADIENE	106-99-0	0.004977
		2,2,4-TRIMETHYLPENTANE	540-84-1	0.015099
		ACETALDEHYDE	75-07-0	0.002400
		BENZENE	71-43-2	0.034235
		BIPHENYL	92-52-4	0.001296
		ETHYLBENZENE	100-41-4	0.000766
		FORMALDEHYDE	50-00-0	0.029100
		NAPHTHALENE	91-20-3	0.045288
		NONANE	111-84-2	0.000485
		POLYCYCLIC ORGANIC MATTER		0.096358
		STYRENE	100-42-5	0.013179
		XYLENES (MIXED ISOMERS)	1330-20-7	0.002150
			Total	0.246319

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Surface Coating	TOG			
Auto Refinishing: SIC 7532				
Total: All Solvent Types				
		ACETONE	67-64-1	0.012000
		ETHYL ACETATE	141-78-6	0.020000
		ETHYLBENZENE	100-41-4	0.005000
		METHYL ETHYL KETONE	78-93-3	0.005000
		METHYL ISOBUTYL KETONE	108-10-1	0.003000
		N-BUTYL ACETATE	138-22-7	0.095000
		N-PROPYL ACETATE	109-60-4	0.012000
		TOLUENE	108-88-3	0.378000
		TRIMETHYLBENZENE	25551-13-7	0.001000
		XYLENES (MIXED ISOMERS)	1330-20-7	0.081000
			Total	0.612000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Degreasing	TOG			
All Processes/All Industries				
Total: All Solvent Types				
		1,1,1-TRICHLOROETHANE	71-55-6	0.237000
		DIMETHYLAMINE	124-40-3	0.098000
		METHYLENE CHLORIDE	75-09-2	0.043000
		TETRACHLOROETHYLENE	127-18-4	0.023000
		TRICHLOROETHYLENE	79-01-6	0.083000
			Total	0.484000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Dry Cleaning	TOG			
All Processes				
Perchloroethylene				
		TETRACHLOROETHYLENE	127-18-4	1.000000
			Total	1.000000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Graphic Arts	TOG			
All Processes				
Total: All Solvent Types				
		ACETONE	67-64-1	0.065000
		ETHYL ACETATE	141-78-6	0.033000
		ETHYLBENZENE	100-41-4	0.006200
		GLYCOL ETHERS (MISC.)	52286-19-8	0.177000
		GLYCOLS		0.018000
		METHYL ETHYL KETONE	78-93-3	0.036000
		N-BUTYL ACETATE	138-22-7	0.022000
		N-PROPYL ACETATE	109-60-4	0.036000
		N-PROPYL ALCOHOL	71-23-8	0.018000
		NONANE	111-84-2	0.002800
		TOLUENE	108-88-3	0.117300
		TRIMETHYLBENZENE	25551-13-7	0.014300
		XYLENES (MIXED ISOMERS)	1330-20-7	0.096900
			Total	0.642500

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Commercial	TOG			
Cutback Asphalt				
Total: All Solvent Types				
		ETHYLBENZENE	100-41-4	0.023000
		NONANE	111-84-2	0.104000
		TOLUENE	108-88-3	0.064000
		TRIMETHYLBENZENE	25551-13-7	0.053000
		XYLENES (MIXED ISOMERS)	1330-20-7	0.122000
			Total	0.366000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Commercial	TOG			
Emulsified Asphalt				
Total: All Solvent Types				
		NAPHTHALENE	91-20-3	0.065000
		TRIMETHYLBENZENE	25551-13-7	0.089000
			Total	0.154000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Commercial	TOG			
Asphalt Roofing				
Total: All Solvent Types				
		NAPHTHALENE	91-20-3	0.065000
		TRIMETHYLBENZENE	25551-13-7	0.089000
			Total	0.154000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Consumer	TOG			
Personal Care Products				
Total: All Solvent Types				
		METHYLENE CHLORIDE	75-09-2	0.041800
			Total	0.041800

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Consumer Household Products	TOG			
Total: All Solvent Types				
		METHYLENE CHLORIDE	75-09-2	0.130100
			Total	0.130100

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Consumer Household Products	TOG			
Total: All Solvent Types				
		METHANOL	67-56-1	0.141900
			Total	0.141900

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Consumer	TOG			
Household Products				
Total: All Solvent Types				
		1,4-DICHLOROBENZENE	106-46-7	1.000000
			Total	1.000000

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Consumer	TOG			
Automotive Aftermarket Products				
Total: All Solvent Types				
		METHANOL	67-56-1	0.141900
			Total	0.141900

Arizona HAPS

Source Specific Speciation Profiles (Annual)

11/22/95

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Solvent Utilization				
Miscellaneous Non-Industrial: Consumer	TOG			
Adhesives and Sealants				
Total: All Solvent Types				
		ETHYLENE GLYCOL	107-21-1	0.199000
		TOLUENE	108-88-3	0.723900
			Total	0.922900

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Storage & Transport				
Petroleum & Petroleum Product Storage	TOG			
Bulk Stations/Terminals: Breathing Loss				
Gasoline				
		1,2-XYLENE	95-47-6	0.000435
		1,3-XYLENE	108-38-3	0.001551
		BENZENE	71-43-2	0.003391
		CUMENE	98-82-8	0.000027
		ETHYLBENZENE	100-41-4	0.000462
		HEXANE	110-54-3	0.009375
		METHYL TERT BUTYL ETHER	1634-04-4	0.006923
		NAPHTHALENE	91-20-3	0.000002
		TOLUENE	108-88-3	0.006670
			Total	0.028836

Arizona HAPS

Source Specific Speciation Profiles (Annual)

11/22/95

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Highway Vehicles - Gasoline	PM10			
Light Duty Gasoline Vehicles (LDGV)				
Total: All Road Types				
		ANTIMONY	7440-36-0	0.000189
		ARSENIC	7740-38-2	0.000021
		BARIUM	7440-39-3	0.000580
		CADMIUM	7440-43-9	0.000106
		CHLORINE	7782-50-5	0.006400
		CHROMIUM	7440-47-3	0.000151
		COBALT	7440-48-4	0.000025
		COPPER	7440-50-8	0.000739
		ELEMENTAL CARBON		0.135021
		INDIUM	7440-74-6	0.000035
		LEAD	7439-92-1	0.001553
		MANGANESE	7439-96-5	0.001048
		MERCURY	7439-97-6	0.000018
		MOLYBDENUM	7439-98-7	0.000058
		NICKEL	7440-02-0	0.000094
		ORGANIC CARBON		0.300752
		POTASSIUM	7440-09-7	0.003861
		SELENIUM	7782-49-2	0.000010
		SILVER	7440-22-4	0.000042
		STRONTIUM	7440-24-6	0.000070
		THALLIUM	7440-28-0	0.000020
		TIN	7440-31-5	0.000149
		YTTRIUM	7440-65-5	0.000017
		ZINC	7440-66-6	0.002727
		ZIRCONIUM	7440-67-7	0.000038
			Total	0.453724

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Highway Vehicles - Diesel	PM10			
Light Duty Diesel Vehicles (LDDV)				
Total: All Road Types				
		ANTIMONY	7440-36-0	0.000108
		ARSENIC	7740-38-2	0.000009
		BARIUM	7440-39-3	0.001397
		CADMIUM	7440-43-9	0.000098
		CHLORINE	7782-50-5	0.016204
		CHROMIUM	7440-47-3	0.000039
		COBALT	7440-48-4	0.000026
		COPPER	7440-50-8	0.000132
		ELEMENTAL CARBON		0.329189
		INDIUM	7440-74-6	0.000047
		LEAD	7439-92-1	0.000147
		MANGANESE	7439-96-5	0.000082
		MERCURY	7439-97-6	0.000014
		MOLYBDENUM	7439-98-7	0.000019
		NICKEL	7440-02-0	0.000026
		ORGANIC CARBON		0.400956
		POTASSIUM	7440-09-7	0.003876
		SELENIUM	7782-49-2	0.000009
		SILVER	7440-22-4	0.000026
		STRONTIUM	7440-24-6	0.000018
		THALLIUM	7440-28-0	0.000031
		TIN	7440-31-5	0.000118
		YTTRIUM	7440-65-5	0.000029
		ZINC	7440-66-6	0.000699
		ZIRCONIUM	7440-67-7	0.000019
			Total	0.753318

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Paved Roads	PM10			
All Paved Roads				
Total: Fugitives				
		ANTIMONY	7440-36-0	0.000015
		ARSENIC	7740-38-2	0.000006
		BARIUM	7440-39-3	0.000753
		CADMIUM	7440-43-9	0.000029
		CHLORINE	7782-50-5	0.002188
		CHROMIUM	7440-47-3	0.000277
		COBALT	7440-48-4	0.000096
		COPPER	7440-50-8	0.000324
		ELEMENTAL CARBON		0.006823
		INDIUM	7440-74-6	0.000011
		LEAD	7439-92-1	0.000865
		MANGANESE	7439-96-5	0.001136
		MERCURY	7439-97-6	0.000011
		MOLYBDENUM	7439-98-7	0.000018
		NICKEL	7440-02-0	0.000107
		ORGANIC CARBON		0.101649
		POTASSIUM	7440-09-7	0.019882
		SELENIUM	7782-49-2	0.000007
		SILVER	7440-22-4	0.000001
		STRONTIUM	7440-24-6	0.001027
		THALLIUM	7440-28-0	0.000022
		TIN	7440-31-5	0.000004
		YTTRIUM	7440-65-5	0.000035
		ZINC	7440-66-6	0.001238
		ZIRCONIUM	7440-67-7	0.000161
			Total	0.136685

Arizona HAPS

Source Specific Speciation Profiles (Annual)

11/22/95

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Mobile Sources				
Unpaved Roads	PM10			
All Unpaved Roads				
Total: Fugitives				
		ANTIMONY	7440-36-0	0.000003
		ARSENIC	7740-38-2	0.000028
		BARIUM	7440-39-3	0.000638
		CADMIUM	7440-43-9	0.000020
		CHLORINE	7782-50-5	0.001234
		CHROMIUM	7440-47-3	0.000206
		COBALT	7440-48-4	0.000060
		COPPER	7440-50-8	0.000102
		INDIUM	7440-74-6	0.000039
		LEAD	7439-92-1	0.000494
		MANGANESE	7439-96-5	0.000976
		MERCURY	7439-97-6	0.000022
		MOLYBDENUM	7439-98-7	0.000031
		NICKEL	7440-02-0	0.000068
		ORGANIC CARBON		0.052807
		POTASSIUM	7440-09-7	0.016299
		SELENIUM	7782-49-2	0.000004
		SILVER	7440-22-4	0.000002
		STRONTIUM	7440-24-6	0.000579
		THALLIUM	7440-28-0	0.000018
		TIN	7440-31-5	0.000005
		YTTRIUM	7440-65-5	0.000034
		ZINC	7440-66-6	0.000963
		ZIRCONIUM	7440-67-7	0.000140
			Total	0.074772

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Natural Sources				
Geogenic	PM10			
Wind Erosion				
Dust Devils				
		ANTIMONY	7440-36-0	0.000005
		ARSENIC	7740-38-2	0.000009
		BARIUM	7440-39-3	0.000701
		CADMIUM	7440-43-9	0.000022
		CHLORINE	7782-50-5	0.000499
		CHROMIUM	7440-47-3	0.000275
		COBALT	7440-48-4	0.000142
		COPPER	7440-50-8	0.000138
		INDIUM	7440-74-6	0.000018
		LEAD	7439-92-1	0.000141
		MANGANESE	7439-96-5	0.001540
		MERCURY	7439-97-6	0.000012
		MOLYBDENUM	7439-98-7	0.000005
		NICKEL	7440-02-0	0.000077
		ORGANIC CARBON		0.029922
		POTASSIUM	7440-09-7	0.021470
		SELENIUM	7782-49-2	0.000002
		SILVER	7440-22-4	0.000006
		STRONTIUM	7440-24-6	0.000359
		THALLIUM	7440-28-0	0.000012
		YTTRIUM	7440-65-5	0.000048
		ZINC	7440-66-6	0.000305
		ZIRCONIUM	7440-67-7	0.000205
			Total	0.055913

Arizona HAPS

11/22/95

Source Specific Speciation Profiles (Annual)

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Miscellaneous Area Sources				
Agriculture Production - Crops	PM10			
Agriculture - Crops				
Tilling				
		ANTIMONY	7440-36-0	0.000008
		ARSENIC	7740-38-2	0.000016
		BARIUM	7440-39-3	0.000649
		CADMIUM	7440-43-9	0.000044
		CHLORINE	7782-50-5	0.000768
		CHROMIUM	7440-47-3	0.000243
		COBALT	7440-48-4	0.000094
		COPPER	7440-50-8	0.000167
		INDIUM	7440-74-6	0.000017
		LEAD	7439-92-1	0.000127
		MANGANESE	7439-96-5	0.001448
		MERCURY	7439-97-6	0.000016
		MOLYBDENUM	7439-98-7	0.000020
		NICKEL	7440-02-0	0.000080
		ORGANIC CARBON		0.038952
		POTASSIUM	7440-09-7	0.021290
		SELENIUM	7782-49-2	0.000006
		SILVER	7440-22-4	0.000015
		STRONTIUM	7440-24-6	0.000398
		THALLIUM	7440-28-0	0.000016
		TIN	7440-31-5	0.000035
		YTTRIUM	7440-65-5	0.000043
		ZINC	7440-66-6	0.000252
		ZIRCONIUM	7440-67-7	0.000171
			Total	0.064875

Arizona HAPS

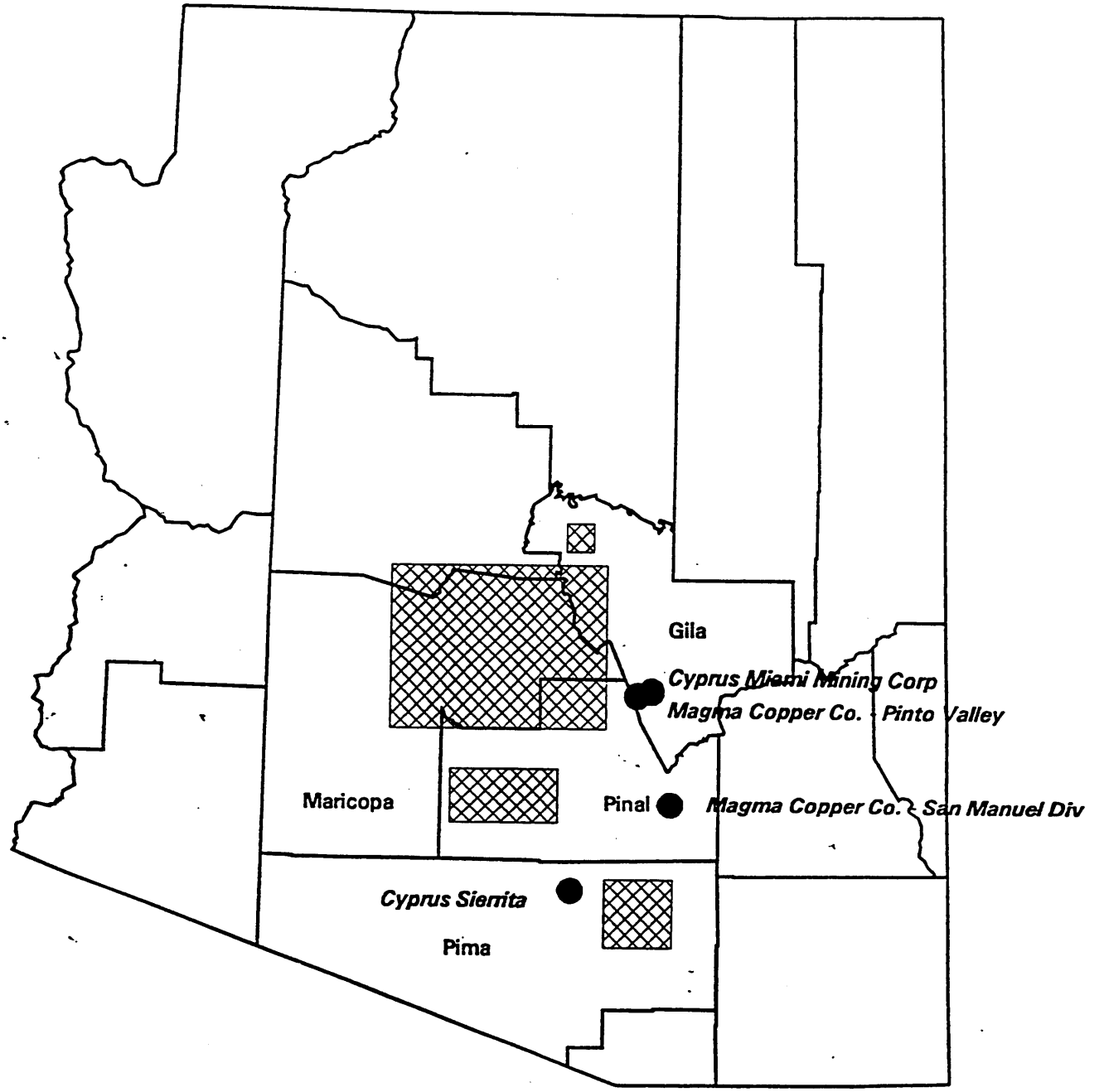
Source Specific Speciation Profiles (Annual)

11/22/95

Area Source Category	Profile Type	Material Name	CAS Number	Weight Fraction
Industrial Processes				
Construction: SIC 15 - 17	PM10			
All Processes				
Wind Erosion				
		ARSENIC	7740-38-2	0.000006
		BARIUM	7440-39-3	0.000614
		CADMIUM	7440-43-9	0.000011
		CHLORINE	7782-50-5	0.000750
		CHROMIUM	7440-47-3	0.000204
		COBALT	7440-48-4	0.000126
		COPPER	7440-50-8	0.000130
		INDIUM	7440-74-6	0.000005
		LEAD	7439-92-1	0.000194
		MANGANESE	7439-96-5	0.001086
		MERCURY	7439-97-6	0.000008
		MOLYBDENUM	7439-98-7	0.000012
		NICKEL	7440-02-0	0.000072
		ORGANIC CARBON		0.049128
		POTASSIUM	7440-09-7	0.015960
		SELENIUM	7782-49-2	0.000008
		SILVER	7440-22-4	0.000016
		STRONTIUM	7440-24-6	0.000495
		THALLIUM	7440-28-0	0.000008
		YTTRIUM	7440-65-5	0.000042
		ZINC	7440-66-6	0.000355
		ZIRCONIUM	7440-67-7	0.000171
			Total	0.069401

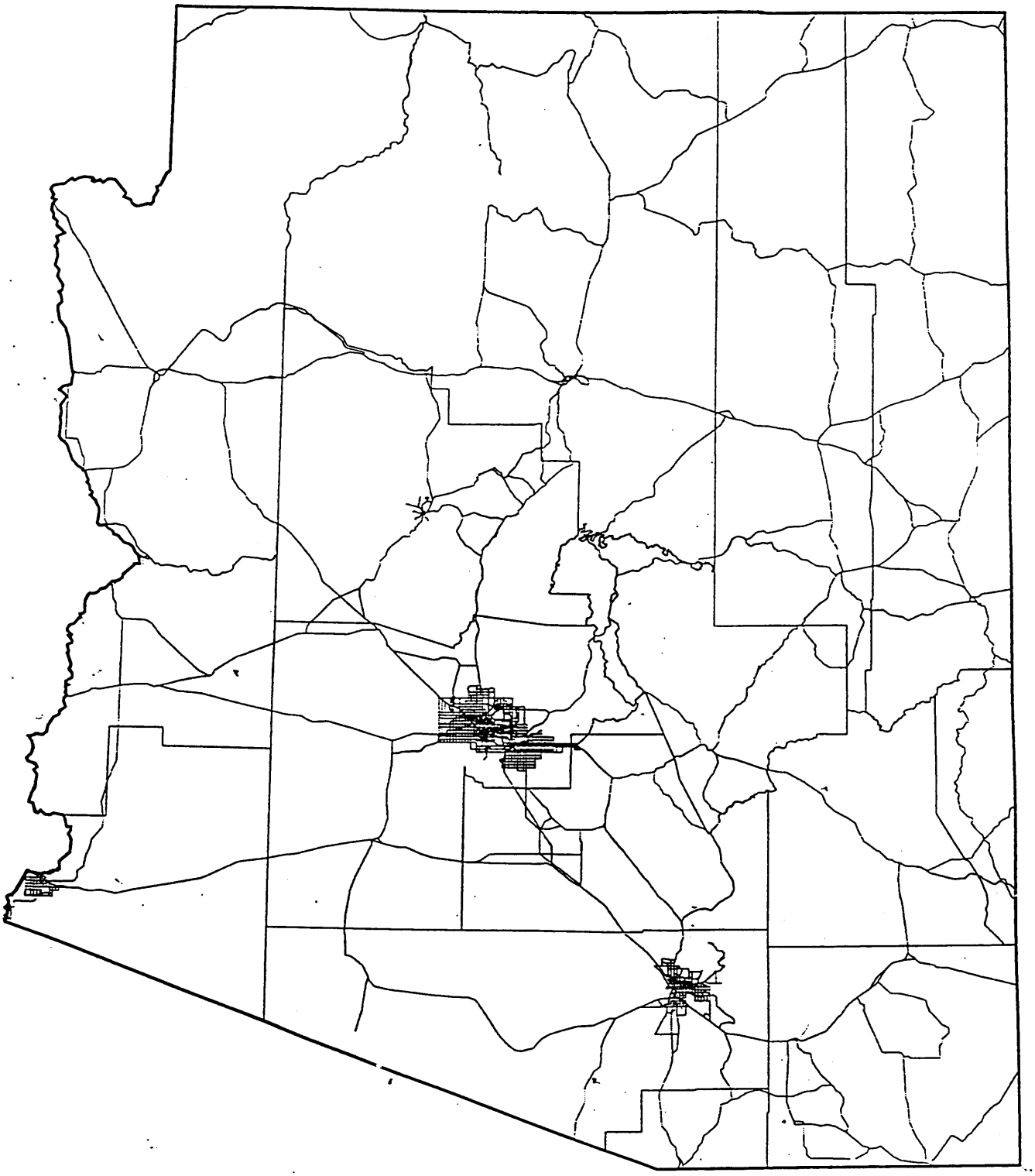
ATTACHMENT A-3

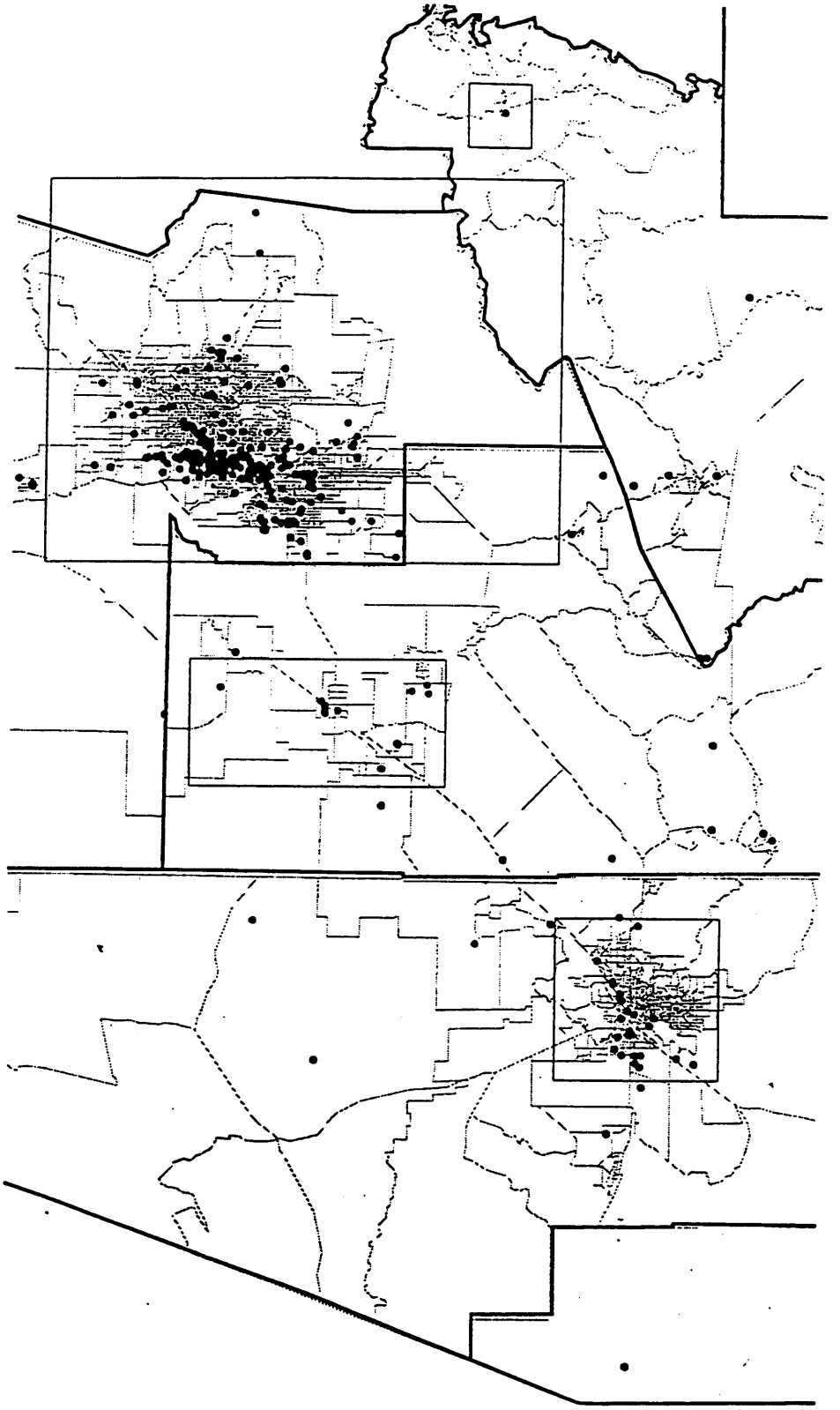
SPATIAL DATA



Counties and Study Regions Included in the Inventory
Mining and Milling Operations

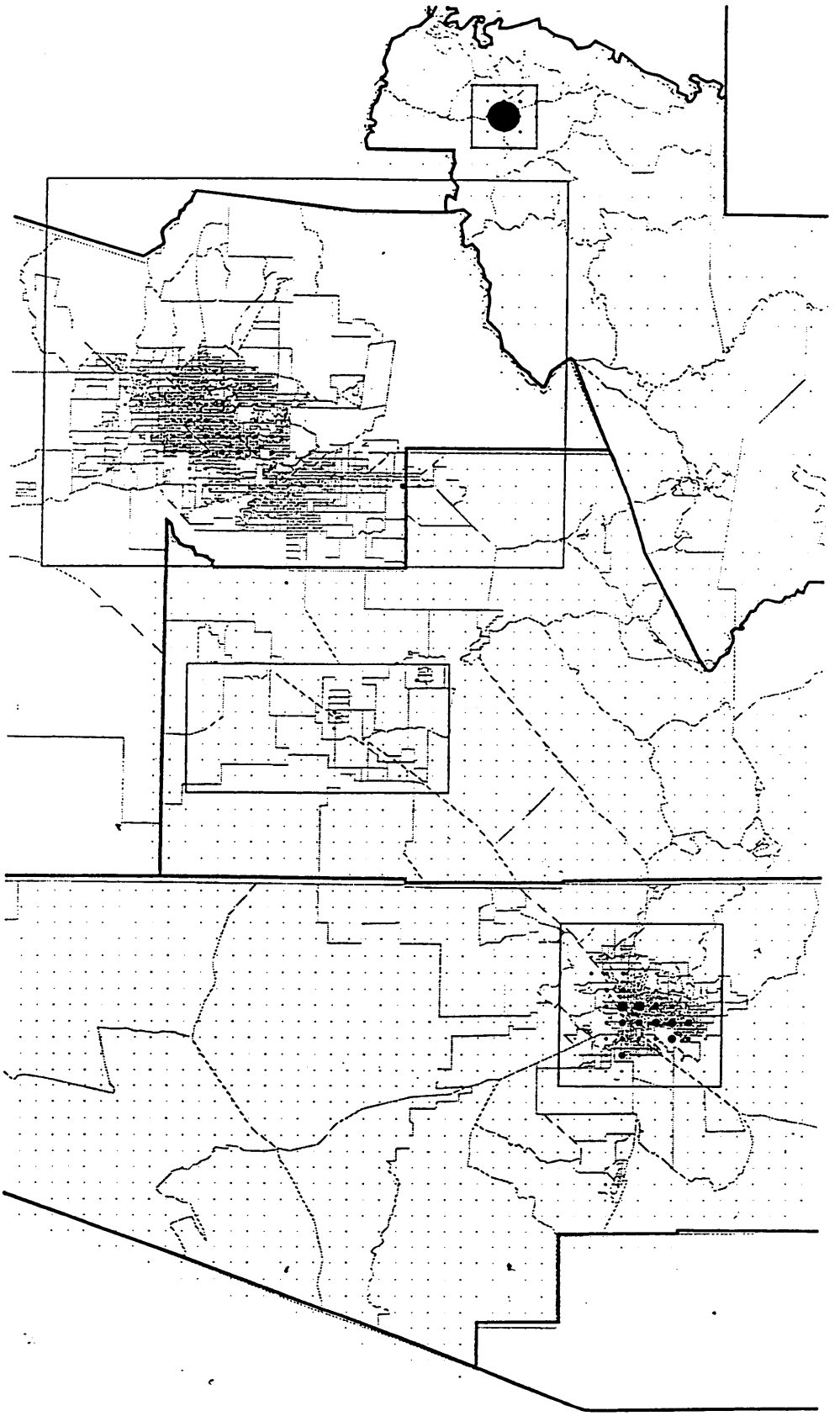
State of Arizona
Major Roads





Counties and Study Regions Included in the Inventory
Point Sources

Emissions Modeling Domain
Chrysene Emissions - Area Sources - Fall



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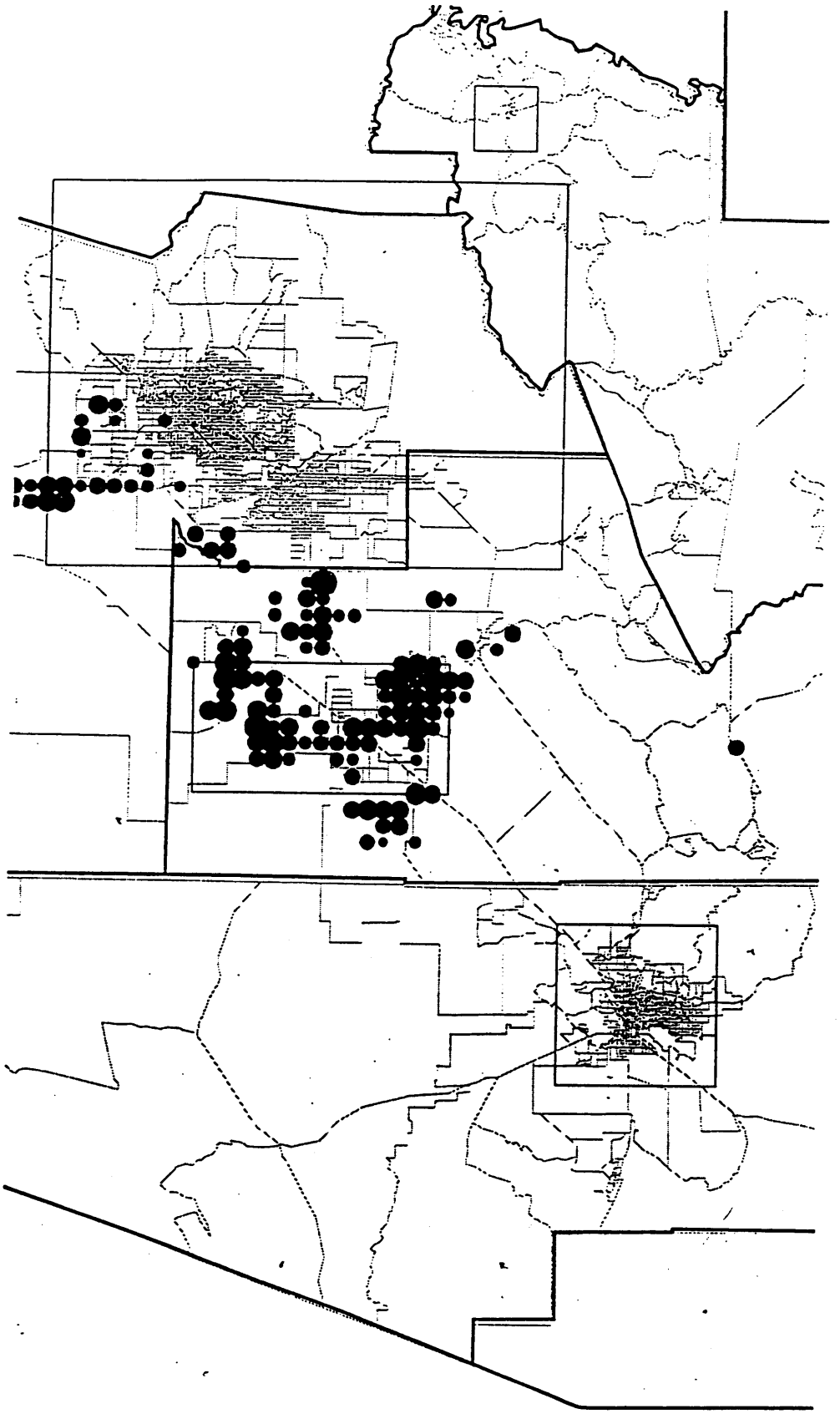
Emissions Modeling Domain
Methyl Parathion Emissions - Fall



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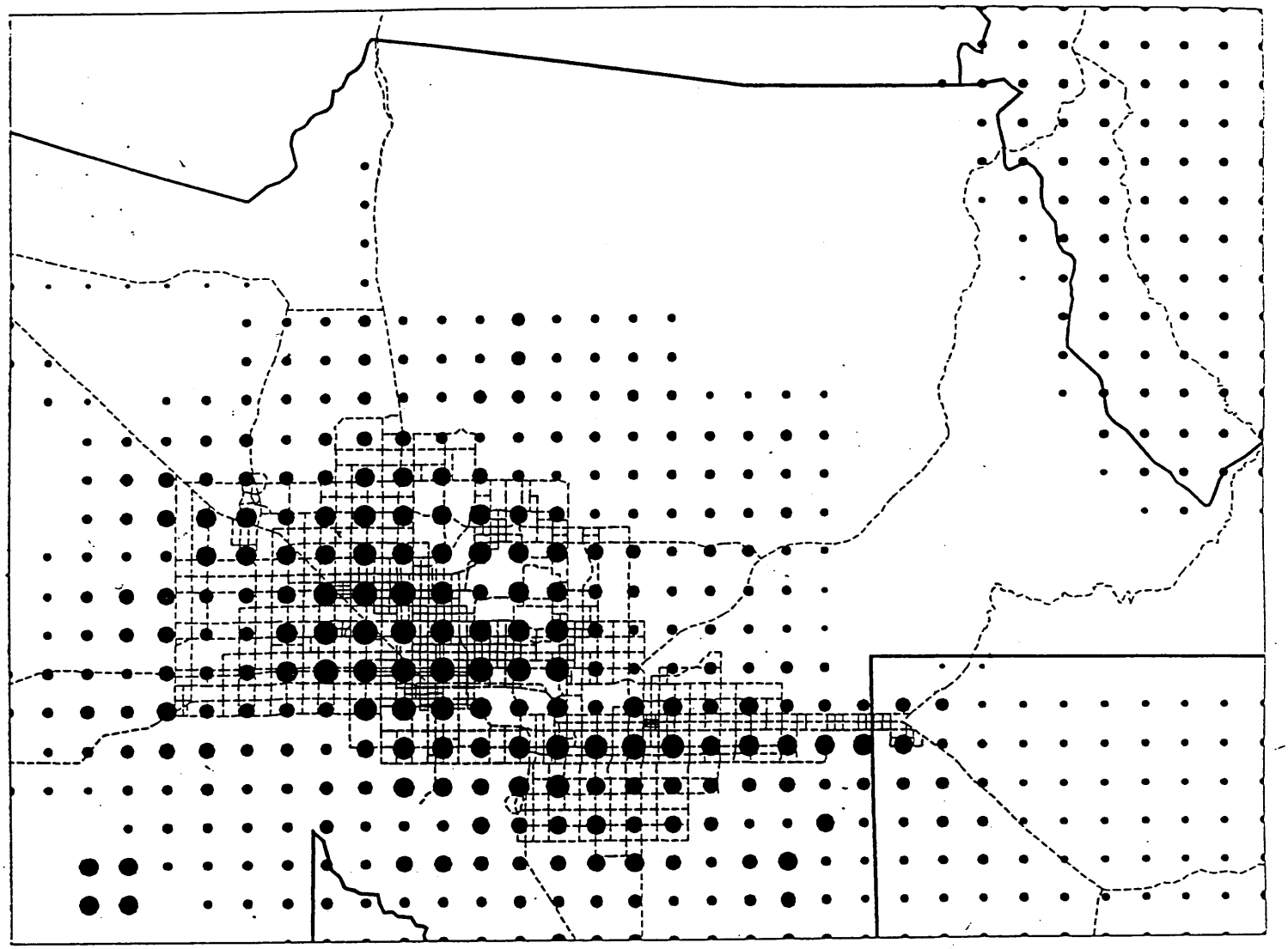
Phoenix Study Region
1,3 Butadiene Emissions - Area Sources - Fall



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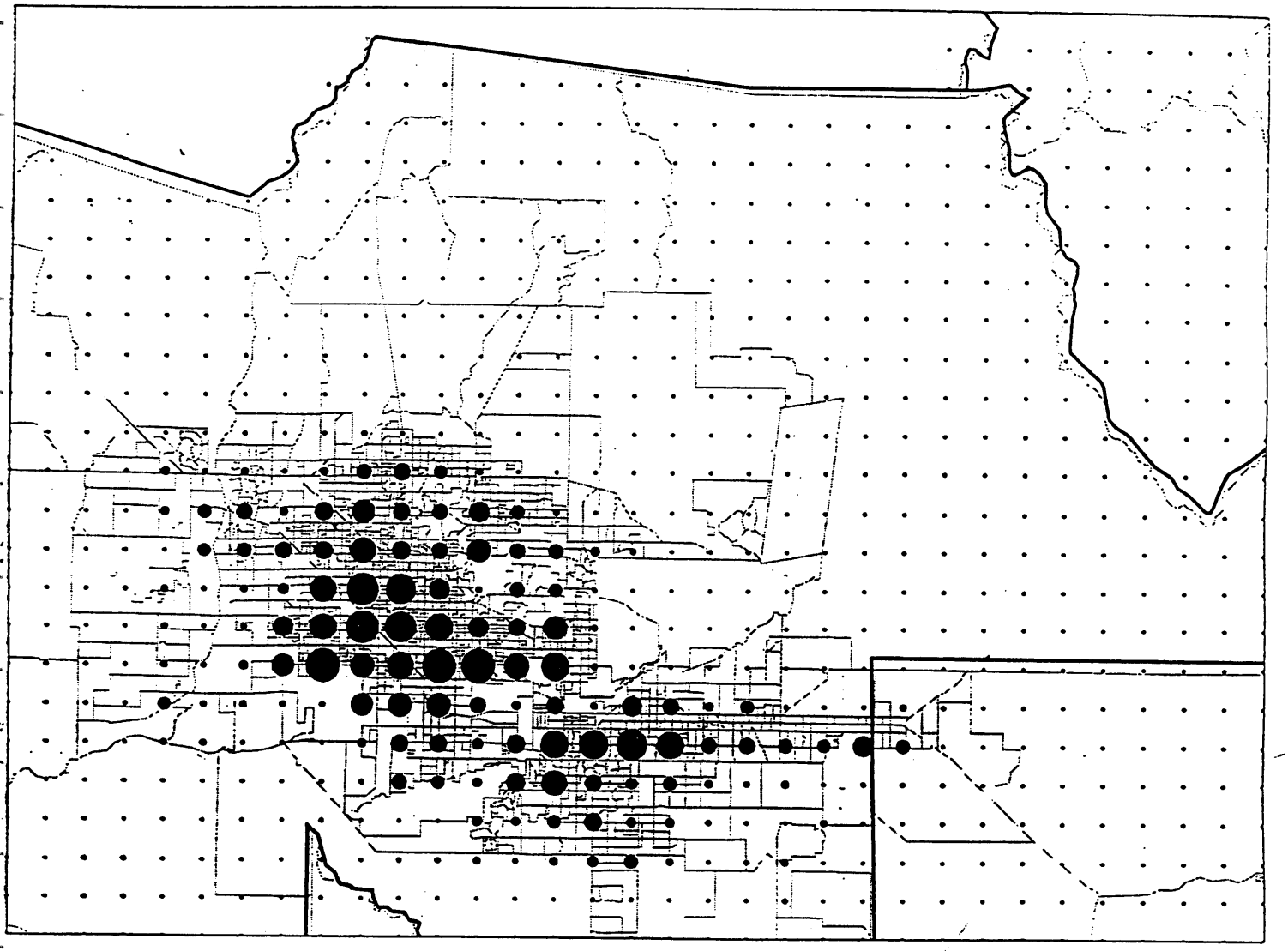
Phoenix Study Region
Benzene Emissions - Area Sources - Fall



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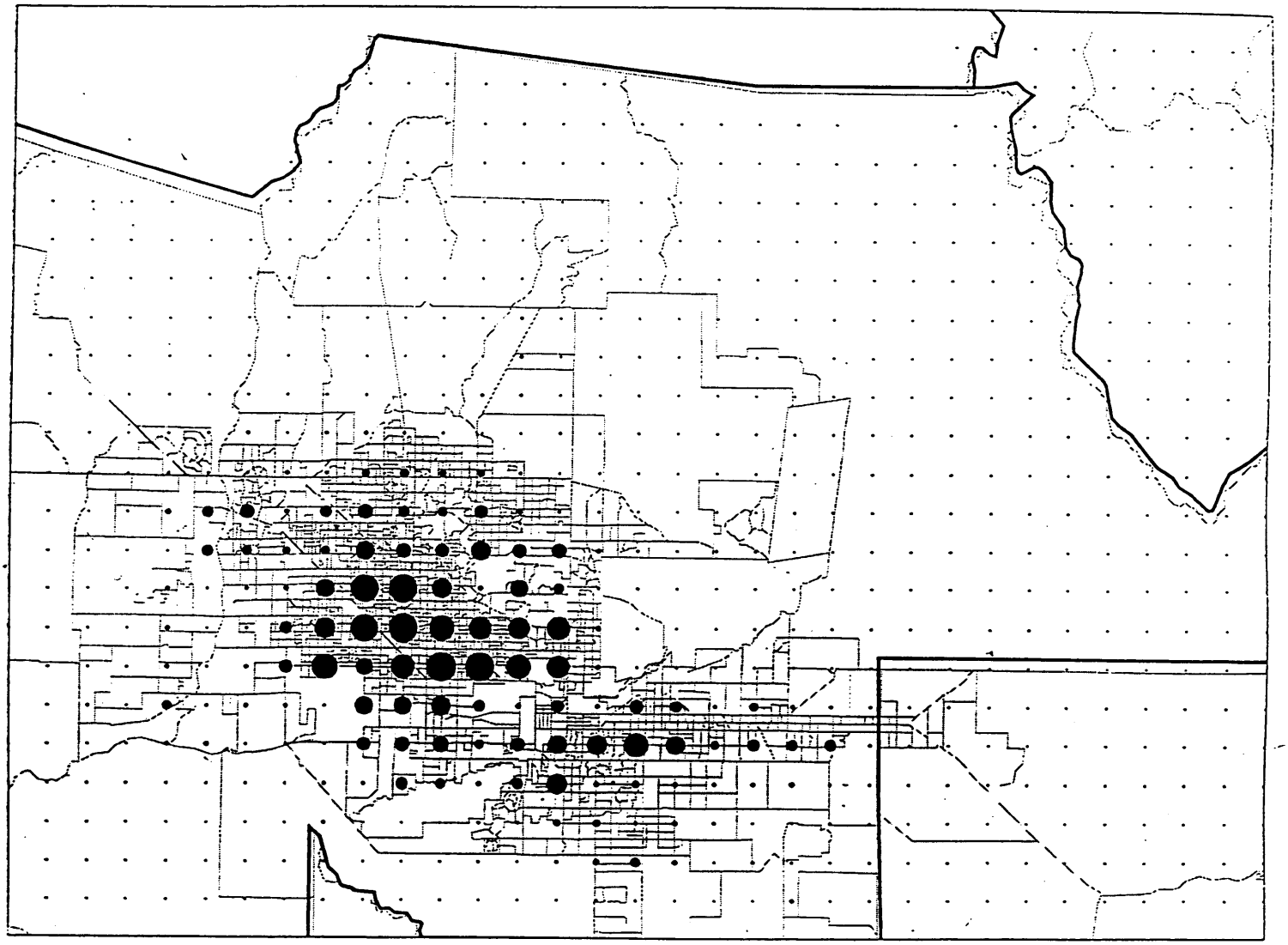
Phoenix Study Region
Chloroform Emissions - Area Sources - Fall



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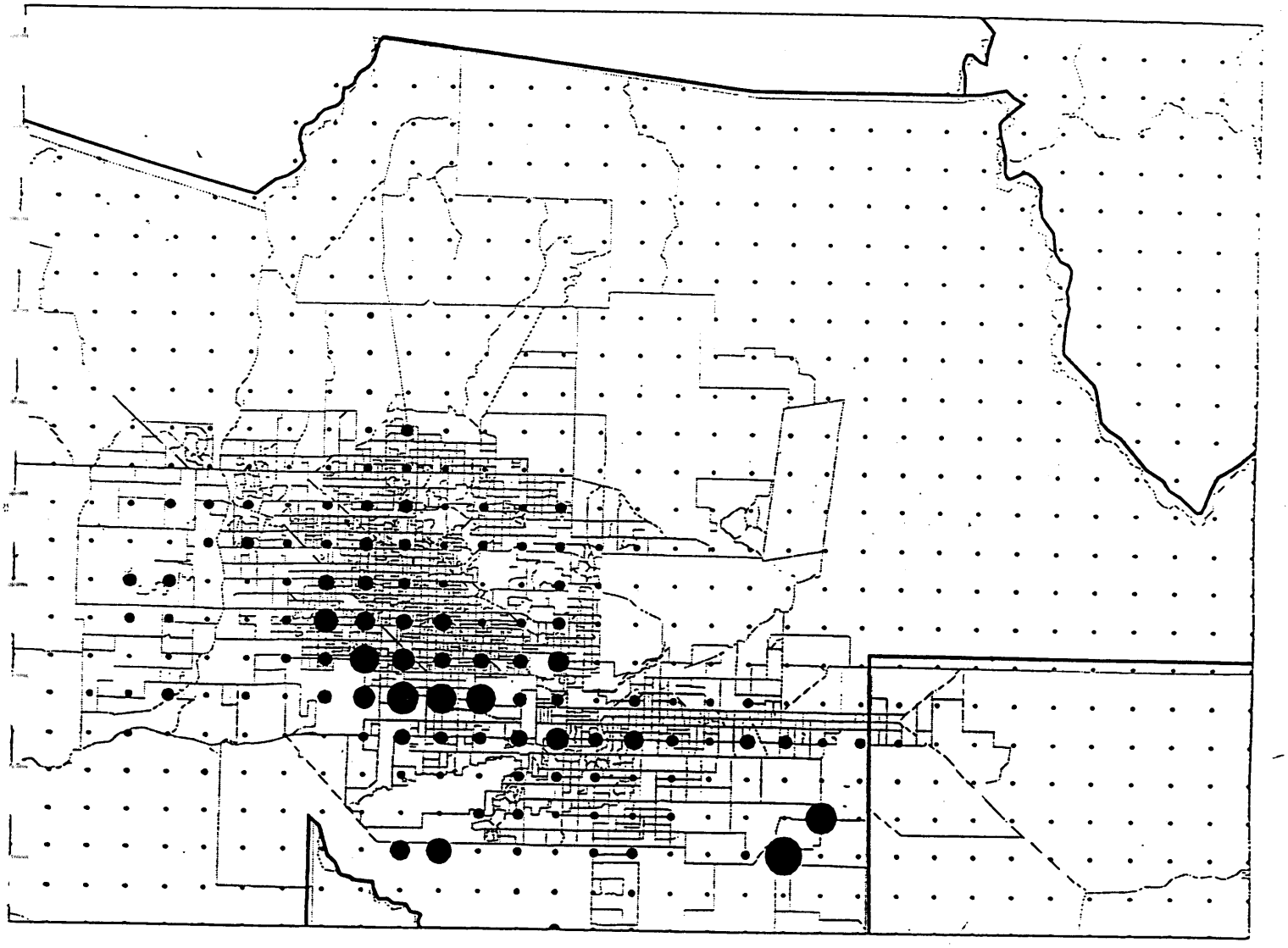
Phoenix Study Region
Chromium Emissions (tpy)- Area Sources - Fall



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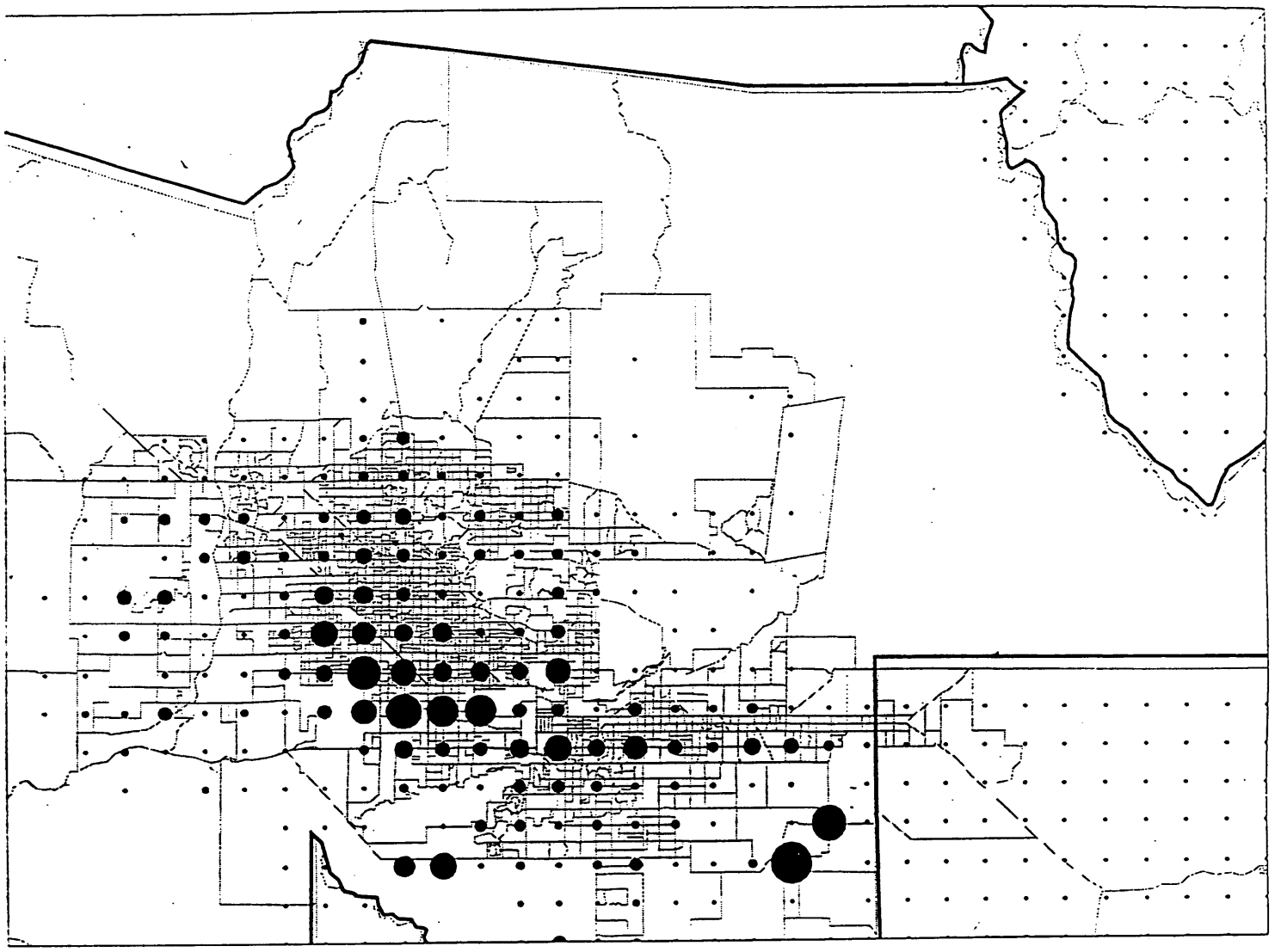
Phoenix Study Region
Dichloromethane Emissions - Area Sources - Fall



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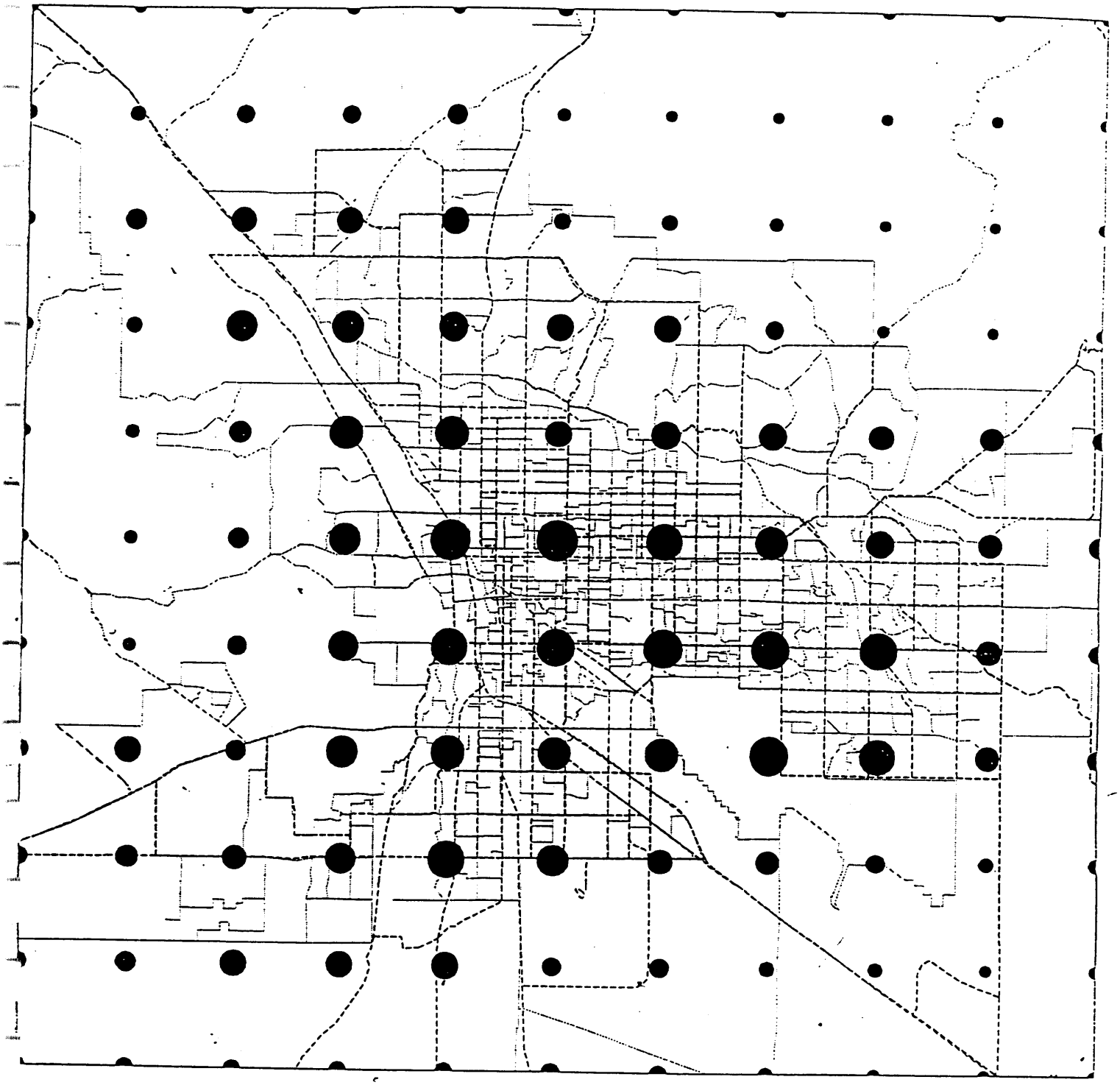
Tucson Study Region
1,3 Butadiene Emissions - Area Sources - Fall



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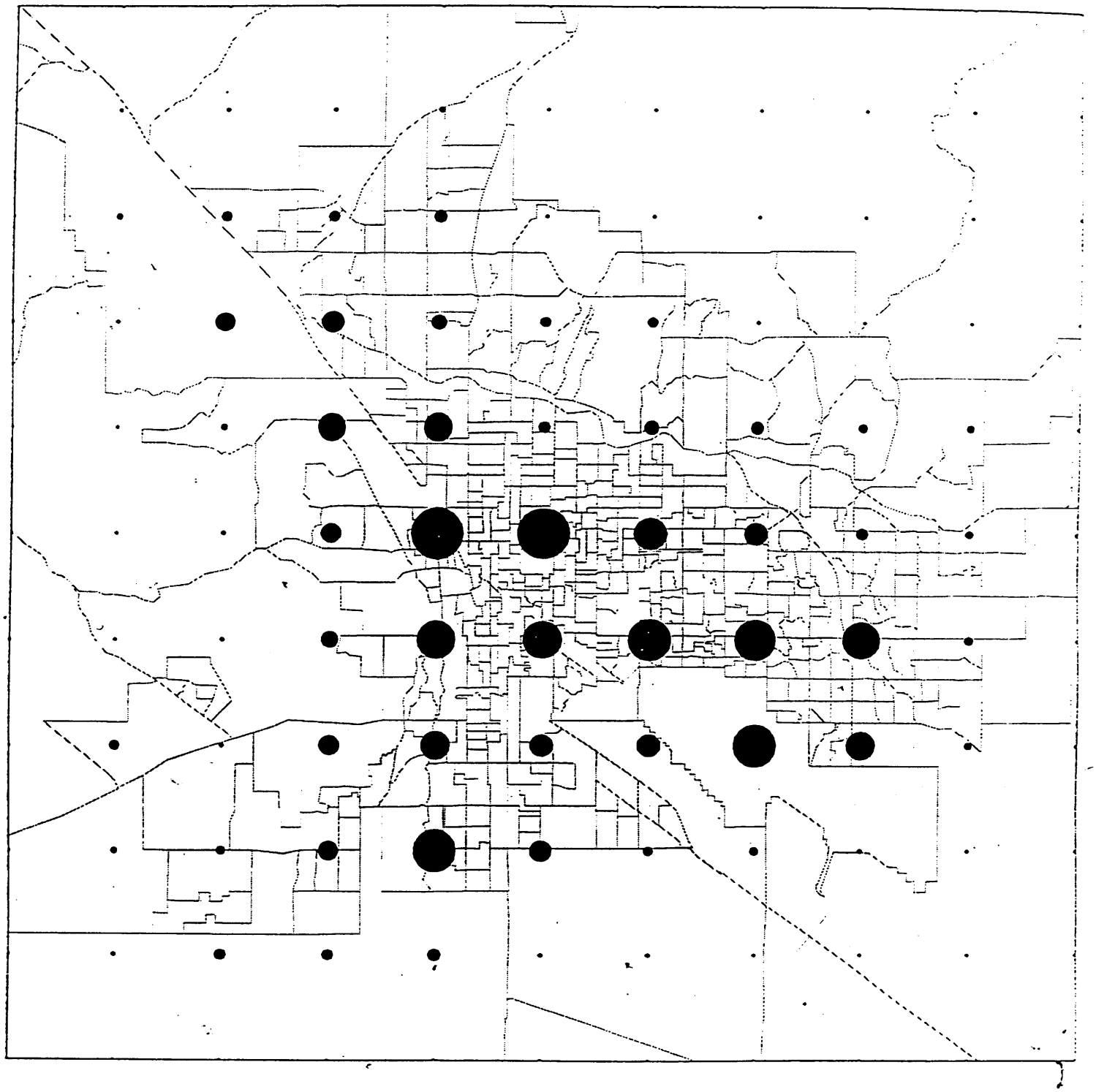
Tucson Study Region
Benzene Emissions - Area Sources - Fall



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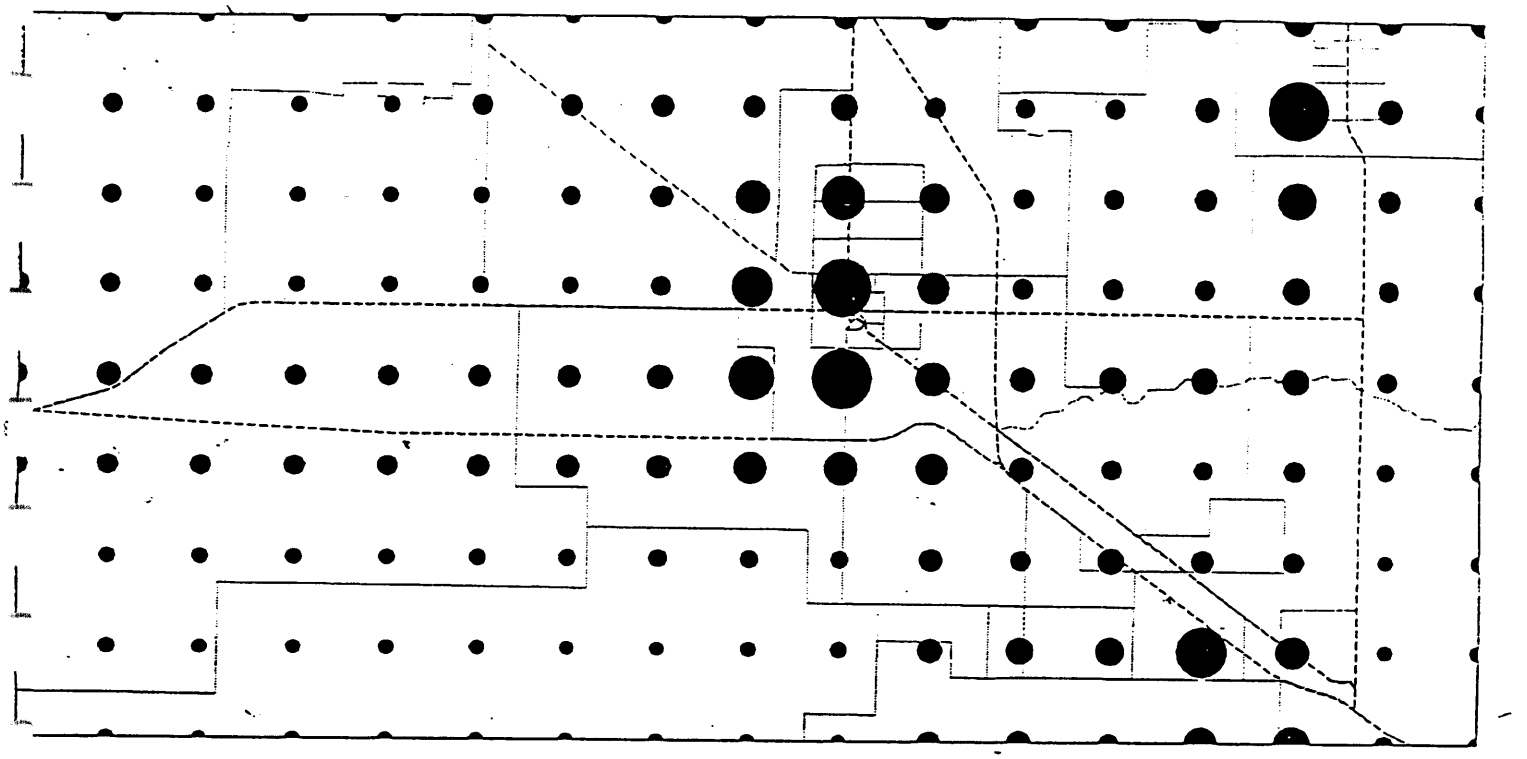
Casa Grande Study Region
1,3 Butadiene Emissions - Area Sources - Fall



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APPENDIX B

HAZARDOUS AIR POLLUTANT SCREENING TABLES

APPENDIX B

HAZARDOUS AIR POLLUTANT SCREENING TABLES

This appendix contains tables that list the data that were examined to identify hazardous substances that are likely to be present in the atmosphere of each of the four study regions. The substances that were identified by this examination as having the potential to cause adverse health effects in each of the regions are called chemicals of interest (COI). The screening process is described further in Section 2 of this document and in Section 4 of Volume 1.

There are two tables for each geographic study region. The first table contains data for all of the substances on the Research HAPs List with an available carcinogenic dose-response value and either an estimate of its emissions or measurements of its concentration in the region. The second table contains data for all substances on the Research HAPs list with an available non-carcinogenic dose-response value and either an estimate of its emissions or measurements of its concentration.

Each table entry includes the following:

- The chemical species' Chemical Abstract Service (CAS) number
- The species' name
- The estimated annual emissions within the study region, in English tons per year
- The average concentration measured at the residential neighborhood sampling site during the research program, in micrograms per cubic meter of air. These average concentrations include all values measured through August 1995. A value of zero was included in the average when the species was not detected in a sample. Values below the lower quantifiable limit, defined as the lowest value with an uncertainty of ±50%, were included as reported in the laboratory analysis (i.e., these values were not set to a fraction of the lower quantifiable limit).
- The number of measurement that were made
- The number of samples in which the species was detected
- The number of samples in which the concentration was above the lower quantifiable limit
- The average lower quantifiable limit, if any concentrations were both detected and below the lower quantifiable limit
- The carcinogenic emission risk indicator (ERI), which is the estimated annual emissions multiplied by the screening cancer unit risk

- The screening excess lifetime cancer risk (ELCR), which is the average measured concentration multiplied by the screening cancer unit risk, in units of 10^{-6}.
- The non-carcinogenic ERI, which is the estimated annual emissions divided by the screening chronic reference concentration.
- The screening chronic hazard quotient, which is the average measured concentration divided by the screening chronic reference concentration

The first table for each region is sorted first by decreasing carcinogenic ERI and then by decreasing screening ELCR. The second table for each region is sorted by decreasing non-carcinogenic ERI and then by decreasing screening hazard quotient. Entries are included for species with available screening chronic reference concentrations and either estimated emissions or average measured concentrations.

PHOENIX REGION
Screening for Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
106-99-0	BUTADIENE, 1,3-	2.93E+02	7.17E-01	59	53	53		821.5	200.80		
71-43-2	BENZENE (INCLUDING BENZENE FROM GASOLINE)	1.98E+03	7.69E+00	59	59	59		164.7	63.87	331.62	1.286
50-00-0	FORMALDEHYDE	1.11E+03	4.42E+00	59	59	59		144.4	57.47	1.59	0.006
7440-38-2	ARSENIC COMPOUNDS (INORGANIC INCLUDING ARSINE)	1.16E+00	1.85E-03	60	51	6	6.62E-03	50.0	7.94	1.11	0.002
7440-43-9	CADMIUM COMPOUNDS	2.76E+00	3.95E-03	60	57	0	1.72E-02	49.7	7.11	1.58	0.002
67-66-3	CHLOROFORM	1.61E+02	3.51E-01	59	58	49	1.95E-01	37.1	8.08	4.60	0.010
7440-02-0	NICKEL COMPOUNDS	1.14E+01	1.88E-03	60	57	14	2.84E-03	27.4	0.45	0.16	0.000
106-46-7	DICHLOROBENZENE, P-	1.64E+02	2.83E-01	59	33	2	9.61E-01	18.1	3.11	0.21	0.000
18540-29-9	CHROMIUM (VI)	1.09E-01						13.0		0.01	
75-21-8	ETHYLENE OXIDE	1.27E+01						12.7			
79-01-6	TRICHLOROETHYLENE	7.04E+02	2.38E-01	59	58	30	2.15E-01	12.1	0.41	33.52	0.011
75-07-0	ACETALDEHYDE	2.98E+02	5.12E+00	59	59	59		6.5	11.27	33.08	0.569
75-09-2	METHYLENE CHLORIDE (DICHLOROMETHANE)	1.14E+03	5.61E+00	59	59	59		5.3	2.63	0.38	0.002
127-18-4	TETRACHLOROETHYLENE (PERCHLOROETHYLENE)	7.51E+02	1.16E+00	59	59	57	2.71E-01	4.4	0.67	21.45	0.033
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)	1.36E+00						3.0		6.79	
107-13-1	ACRYLONITRILE	2.99E+00						2.0		1.50	
50-32-8	BENZO(A)PYRENE	1.62E-01	6.12E-04	59	49	19	7.32E-04	1.8	0.67	0.00	0.000
75-01-4	VINYL CHLORIDE	2.01E+00						1.7			
100-44-7	BENZYL CHLORIDE	1.36E+00						0.7			
75-27-4	BROMODICHLOROMETHANE	1.18E+00						0.4		0.02	
62-53-3	ANILINE	2.37E+01						0.4		0.24	
107-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)	9.02E-01						0.2		0.09	
56-23-5	CARBON TETRACHLORIDE	1.56E+00	7.51E-01	59	59	59		0.2	11.27	0.78	0.376
140-88-5	ETHYL ACRYLATE	1.49E+00						0.2			
1582-09-8	TRIFLURALIN	5.29E+00						0.1		0.20	
7440-41-7	BERYLLIUM COMPOUNDS	4.44E-03						0.1		0.00	
75-56-9	PROPYLENE OXIDE	2.83E+00						0.1		0.09	
79-34-5	TETRACHLOROETHANE, 1,1,2,2-	1.20E-01						0.1			
1746-01-6	TETRACHLORODIBENZO-P-DIOXIN	1.10E-07						0.0			
75-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)	6.98E-02	0.00E+00	59	0	0		0.0	0.00	0.00	0.000
106-89-8	EPICHLOROHYDRIN (1-CHLORO-2,3-EPOXYPROPANE)	1.36E+00						0.0		1.36	
218-01-9	CHRYSENE	1.37E-01	7.02E-04	59	50	43	3.41E-04	0.0	0.01	0.00	0.000
101-77-9	METHYLENEDIANILINE, 4,4-	3.14E-03						0.0			
78-87-5	PROPYLENE DICHLORIDE (1,2-DICHLOROPROPANE)	6.98E-02						0.0		0.02	

PHOENIX REGION
Screening for Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
79-00-5	TRICHLOROETHANE 1,1,2-	4.98E-02	0.00E+00	59	0	0		0.0	0.00	0.00	0.000
123-91-1	DIOXANE (1,4-DIETHYLENEOXIDE)	7.94E-02						0.0			
1897-45-6	CHLOROTHALONIL	3.30E-02						0.0		0.00	
74-87-3	METHYL CHLORIDE (CHLOROMETHANE)	0.00E+00						0.0			
53-70-3	DIBENZ(A,H)ANTHRACENE		1.20E-04	59	26	0	8.63E-04		0.25		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		1.20E-04	59	26	0	8.63E-04		0.25		0.000
205-99-2	BENZO(B)FLUORANTHENE		1.03E-03	59	51	35	3.81E-04		0.11		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.03E-03	59	51	35	3.81E-04		0.11		0.000
193-39-5	INDENO(123-CD)PYRENE		5.06E-04	59	54	20	5.93E-04		0.06		0.000
56-55-3	BENZO(A)ANTHRACENE		3.60E-04	59	40	13	6.49E-04		0.04		0.000

PHOENIX REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
107-02-8	ACROLEIN	3.97E+01	7.59E-02	5	5	5				1987.27	3.795
7439-96-5	MANGANESE COMPOUNDS	1.51E+02	2.06E-02	60	60	56	3.70E-03			376.63	0.052
71-43-2	BENZENE (INCLUDING BENZENE FROM GASOLINE)	1.98E+03	7.69E+00	59	59	59		164.7	63.87	331.62	1.286
7440-39-3	BARIUM	9.04E+01	3.32E-02	60	56	4	9.79E-02			180.84	0.066
25551-13-7	TRIMETHYLBENZENE	2.16E+02								154.11	
7439-92-1	LEAD COMPOUNDS	6.45E+01	1.98E-02	60	59	56	3.47E-03			43.01	0.013
79-01-6	TRICHLOROETHYLENE	7.04E+02	2.38E-01	59	58	30	2.15E-01	12.1	0.41	33.52	0.011
75-07-0	ACETALDEHYDE	2.98E+02	5.12E+00	59	59	59		6.5	11.27	33.08	0.569
108-90-7	CHLOROBENZENE	6.25E+02	2.65E-02	59	14	10	9.20E-02			31.25	0.001
127-18-4	TETRACHLOROETHYLENE (PERCHLOROETHYLENE)	7.51E+02	1.16E+00	59	59	57	2.71E-01	4.4	0.67	21.45	0.033
108-88-3	TOLUENE	8.46E+03	2.02E+01	59	59	59				21.16	0.051
110-54-3	HEXANE, N-	2.36E+03	6.02E+00	59	59	59				11.79	0.030
7439-97-6	MERCURY COMPOUNDS	2.89E+00	3.10E-04	60	30	0	3.84E-03			9.63	0.001
67-56-1	METHANOL	1.25E+04								7.13	
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)	1.36E+00						3.0		6.79	
7440-28-0	THALLIUM	1.61E+00	2.15E-04	60	35	0	3.72E-03			5.76	0.001
7783-06-4	HYDROGEN SULFIDE	4.35E+00								4.83	
67-66-3	CHLOROFORM	1.61E+02	3.51E-01	59	58	49	1.95E-01	37.1	8.08	4.60	0.010
1330-20-7	XYLENES (ISOMERS & MIXTURE)	2.91E+03	1.27E+01	59	59	59				4.16	0.018
71-55-6	METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)	3.50E+03	1.40E+00	59	59	59				3.50	0.001
298-04-4	DISULFOTON	4.75E-01								3.39	
108-10-1	METHYL ISOBUTYL KETONE (HEXONE)	2.04E+02								2.55	
67-64-1	ACETONE	7.20E+02	1.53E+00	59	56	24	1.43E+00			2.06	0.004
95-47-6	XYLENES, O-	1.41E+03	4.64E+00	59	59	59				2.01	0.007
7647-01-0	HYDROCHLORIC ACID (HYDROGEN CHLORIDE (GAS ONLY))	1.37E+01								1.96	
298-00-0	METHYL PARATHION	1.71E+00								1.96	
2921-88-2	CHLORPYRIFOS	1.85E+01								1.77	
7664-41-7	AMMONIA	1.65E+02								1.65	
50-00-0	FORMALDEHYDE	1.11E+03	4.42E+00	59	59	59		144.4	57.47	1.59	0.006
7440-43-9	CADMIUM COMPOUNDS	2.76E+00	3.95E-03	60	57	0	1.72E-02	49.7	7.11	1.58	0.002
107-13-1	ACRYLONITRIL	2.99E+00						2.0		1.50	
91-20-3	NAPHTHALENE	2.03E+02	8.26E-01	59	59	59				1.45	0.006
79-10-7	ACRYLIC ACID	1.36E+00								1.36	
106-89-8	EPICHLOROHYDRIN (1-CHLORO-2,3-EPOXYPROPANE)	1.36E+00						0.0		1.36	

PHOENIX REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
71-36-3	BUTANOL, N-	4.71E+02								1.34	
108-38-3	XYLENES, M-	8.04E+02								1.15	
7440-38-2	ARSENIC COMPOUNDS (INORGANIC INCLUDING ARSINE)	1.16E+00	1.85E-03	60	51	6	6.62E-03	50.0	7.94	1.11	0.002
78-93-3	METHYL ETHYL KETONE (2-BUTANONE)	1.04E+03	9.94E-01	59	49	39	5.25E-01			1.04	0.001
100-41-4	ETHYL BENZENE	1.00E+03	3.45E+00	59	59	59				1.00	0.003
56-23-5	CARBON TETRACHLORIDE	1.56E+00	7.51E-01	59	59	59		0.2	11.27	0.78	0.376
101-68-8	METHYLENE DIPHENYL DIISOCYANATE (MDI)	1.44E-02								0.72	
95-50-1	DICHLOROBENZENE, O-	1.39E+02	0.00E+00	59	0	0				0.69	0.000
98-95-3	NITROSOBENZENE	1.36E+00								0.68	
7440-36-0	ANTIMONY COMPOUNDS	9.49E-01	1.31E-03	60	24	0	3.10E-02			0.68	0.001
106-42-3	XYLENES, P-	1.96E+02								0.65	
7782-50-5	CHLORINE	2.09E+02	2.49E-01	60	60	55	1.58E-02			0.60	0.001
123-73-9	CROTONALDEHYDE	1.95E+01	2.44E-02	5	1	1				0.56	0.001
75-09-2	METHYLENE CHLORIDE (DICHLOROMETHANE)	1.14E+03	5.61E+00	59	59	59		5.3	2.63	0.38	0.002
98-01-1	FURFURAL	1.42E+01								0.28	
100-42-5	STYRENE	2.77E+02	1.27E+00	59	59	59				0.28	0.001
62-53-3	ANILINE	2.37E+01						0.4		0.24	
68-12-2	DIMETHYL FORMAMIDE	6.28E+00								0.21	
106-46-7	DICHLOROBENZENE, P-	1.64E+02	2.83E-01	59	33	2	9.61E-01	18.1	3.11	0.21	0.000
1582-09-8	TRIFLURALIN	5.29E+00						0.1		0.20	
7440-50-8	COPPER COMPOUNDS	2.53E+01	2.93E-02	60	59	57	2.60E-03			0.19	0.000
98-82-8	CUMENE	1.73E+00								0.19	
298-02-2	PHORATE	1.22E-01								0.17	
7440-02-0	NICKEL COMPOUNDS	1.14E+01	1.88E-03	60	57	14	2.84E-03	27.4	0.45	0.16	0.000
333-41-5	DIAZINON	4.31E-01								0.14	
7439-98-7	MOLYBDENUM	1.97E+00	8.48E-04	60	52	1	3.93E-03			0.11	0.000
7440-22-4	SILVER COMPOUNDS	1.80E+00	3.77E-03	60	52	2	1.61E-02			0.10	0.000
116-06-3	ALDICARB	3.48E-01								0.10	
115-29-7	ENDOSULFAN	2.00E+00								0.10	
75-56-9	PROPYLENE OXIDE	2.83E+00						0.1		0.09	
107-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)	9.02E-01						0.2		0.09	
7664-93-9	SULFURIC ACID	5.80E+00								0.08	
7440-66-6	ZINC	8.64E+01	3.17E-02	60	60	57	2.80E-03			0.08	0.000
7440-48-4	COBALT COMPOUNDS	1.65E+01	1.63E-04	60	28	0	1.77E-02			0.08	0.000

PHOENIX REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
141-78-6	ETHYL ACETATE	2.18E+02								0.07	
7782-49-2	SELENIUM COMPOUNDS	1.15E+00	5.38E-04	60	48	6	1.80E-03			0.07	0.000
126-99-8	CHLOROPRENE	3.81E-01								0.05	
80-62-6	METHYL METHACRYLATE	1.49E+01								0.05	
1563-66-2	CARBOFURAN	7.22E-01								0.04	
7440-24-6	STRONTIUM	8.24E+01	9.19E-03	60	57	56	1.40E-03			0.04	0.000
156-59-2	DICHLOROETHENE, 1,2-,CIS-	1.36E+00	0.00E+00	59	0	0				0.04	0.000
75-15-0	CARBON DISULFIDE	3.70E-01								0.04	
26628-22-8	SODIUM AZIDE	4.78E-01								0.03	
75-71-8	DICHLORODIFLUOROMETHANE	6.78E+00								0.03	
92-52-4	BIPHENYL	4.22E+00	1.99E-02	59	59	59				0.02	0.000
100-52-7	BENZALDEHYDE	8.12E+00	1.79E+00	59	50	47	1.40E-01			0.02	0.005
7440-42-8	BORON	4.57E-01								0.02	
121-75-5	MALATHION	1.33E+00								0.02	
78-87-5	PROPYLENE DICHLORIDE (1,2-DICHLOROPROPANE)	6.98E-02						0.0		0.02	
75-27-4	BROMODICHLOROMETHANE	1.18E+00						0.4		0.02	
57-55-6	PROPANEDIOL, 1,2-	1.03E+03								0.01	
07-21-1	ETHYLENE GLYCOL	9.75E+01								0.01	
6752-77-5	METHOMYL	1.17E+00								0.01	
96-33-3	METHYL ACRYLATE	1.36E+00								0.01	
11-15-9	ETHOXY ETHYL ACETATE, 2-	1.21E+01								0.01	
634-04-4	METHYL TERT BUTYL ETHER	2.79E+01								0.01	
440-47-3	CHROMIUM, TOTAL	3.06E+01	2.77E-03	60	58	7	6.03E-03			0.01	0.000
08-95-2	PHENOL	1.59E+01								0.01	
664-39-3	HYDROGEN FLUORIDE (HYDROFLUORIC ACID)	1.48E+00								0.01	
8-83-1	ISOBUTYL ALCOHOL	6.87E+00								0.01	
8540-29-9	CHROMIUM (VI)	1.09E-01						13.0		0.01	
782-41-4	FLUORINE (AS FLUORIDE)	1.22E+00								0.01	
0-29-7	ETHYL ETHER	3.13E+00								0.00	
7-12-5	CYANIDE COMPOUNDS (AS HYDROGEN CYANIDE)	1.25E-02								0.00	
9-00-5	TRICHLOROETHANE 1,1,2-	4.98E-02	0.00E+00	59	0	0		0.0	0.00	0.00	0.000
08-05-4	VINYL ACETATE	6.23E-01								0.00	
4-74-2	DIBUTYLPHTHALATE	9.82E-01								0.00	
5-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)	6.98E-02	0.00E+00	59	0	0		0.0	0.00	0.00	0.000

PHOENIX REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
85-44-9	PHTHALIC ANHYDRIDE	2.63E-01								0.00	
50-32-8	BENZO(A)PYRENE	1.62E-01	6.12E-04	59	49	19	7.32E-04	1.8	0.67	0.00	0.000
108-94-1	CYCLOHEXANONE	2.67E+01								0.00	
75-34-3	ETHYLIDENE DICHLORIDE (1,1-DICHLOROETHANE)	7.18E-01								0.00	
218-01-9	CHRYSENE	1.37E-01	7.02E-04	59	50	43	3.41E-04	0.0	0.01	0.00	0.000
21087-64-9	METRIBUZIN	8.09E-02								0.00	
63-25-2	CARBARYL	2.96E-01								0.00	
75-00-3	ETHYL CHLORIDE (CHLOROETHANE)	7.33E+00								0.00	
1897-45-6	CHLOROTHALONIL	3.30E-02						0.0		0.00	
79-20-9	METHYL ACETATE	1.91E+00								0.00	
85-68-7	BUTYL BENZYL PHTHALATE	3.57E-01								0.00	
206-44-0	FLUORANTHENE	6.32E-02	6.62E-03	59	59	59				0.00	0.000
7440-31-5	TIN AND OXIDE COMPOUNDS	8.90E-01	2.75E-03	60	49	0	2.53E-02			0.00	0.000
94-75-7	D 2,4-, SALTS AND ESTERS	1.09E-02								0.00	
7440-41-7	BERYLLIUM COMPOUNDS	4.44E-03						0.1		0.00	
108-31-6	MALEIC ANHYDRIDE	7.53E-02								0.00	
64-18-6	FORMIC ACID	1.38E+00								0.00	
23950-58-5	PRONAMIDE	1.19E-02								0.00	
7439-89-6	IRON (SOLUBLE COMPOUNDS)	1.32E-02	8.25E-01	60	60	58	3.20E-03			0.00	0.001
65-85-0	BENZOIC ACID	4.28E-02								0.00	
131-11-3	DIMETHYL PHTHALATE	2.06E-02								0.00	
74-83-9	METHYL BROMIDE (BROMOMETHANE)	0.00E+00	4.60E-03	59	2	0	3.88E-01			0.00	0.001
17804-35-2	BENOMYL	0.00E+00								0.00	
1910-42-5	PARAQUAT	0.00E+00								0.00	
91-57-6	2-METHYLNAPHTHALENE (NAPHTHALENE AS SURROGATE)		4.62E-01	59	59	59					0.003
MNAPH1	1-METHYLNAPHTHALENE, 1-		2.06E-01	59	59	59					0.001
DM1367	DIMETHYLNAPHTHALENE, 1,7+1,3+1,6-		8.80E-02	59	59	59					0.001
DMN267	2,6+2,7-DIMETHYLNAPHTHALENE (NAPHTHALENE AS SURROGATE)		5.48E-02	59	59	59					0.000
7429-90-5	ALUMINUM		1.34E+00	60	60	59	1.88E-02				0.000
D14523	DIMETHYLNAPHTHALENE, 2,3+1,4+1,5-		2.71E-02	59	59	58	2.42E-03				0.000
208-96-8	ACENAPHTHYLENE		1.47E-02	59	53	44	1.45E-03				0.000
DMN12	DIMETHYLNAPHTHALENE, 1,2-		8.71E-03	59	48	47	2.17E-03				0.000
83-32-9	ACENAPHTHENE		9.35E-03	59	59	58	5.46E-04				0.000
129-00-0	PYRENE		4.08E-03	59	59	59					0.000

PHOENIX REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
85-01-8	PHENANTHRENE		2.76E-02	59	59	59					0.000
191-24-2	BENZO(GHI)PERYLENE		1.17E-03	59	56	31	7.78E-04				0.000
205-99-2	BENZO(B)FLUORANTHENE		1.03E-03	59	51	35	3.81E-04		0.11		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.03E-03	59	51	35	3.81E-04		0.11		0.000
193-39-5	INDENO(123-CD)PYRENE		5.06E-04	59	54	20	5.93E-04		0.06		0.000
DMN18	DIMETHYLNAPHTHALENE, 1,8-		5.23E-04	59	16	11	1.82E-03				0.000
56-55-3	BENZO(A)ANTHRACENE		3.60E-04	59	40	13	6.49E-04		0.04		0.000
120-12-7	ANTHRACENE		2.48E-03	59	57	57					0.000
53-70-3	DIBENZ(A,H)ANTHRACENE		1.20E-04	59	26	0	8.63E-04		0.25		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		1.20E-04	59	26	0	8.63E-04		0.25		0.000
BBCHRN	BENZO(B)CHRYSENE		1.14E-04	59	20	5	4.59E-04				0.000
86-73-7	FLUORENE		1.34E-02	59	59	59					0.000

TUCSON REGION
Screening for Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
106-99-0	BUTADIENE, 1,3-	8.81E+01	2.14E-01	23	23	23		246.7	60.01		
71-43-2	BENZENE (INCLUDING BENZENE FROM GASOLINE)	6.29E+02	2.99E+00	23	23	23		52.2	24.79	105.09	0.499
50-00-0	FORMALDEHYDE	3.27E+02	8.43E-01	25	25	25		42.6	10.96	0.47	0.001
7440-38-2	ARSENIC COMPOUNDS (INORGANIC INCLUDING ARSINE)	3.83E-01	1.07E-03	3	3	0	3.20E-03	16.5	4.59	0.36	0.001
7440-43-9	CADMIUM COMPOUNDS	8.04E-01	0.00E+00	3	0	0		14.5	0.00	0.46	0.000
7440-02-0	NICKEL COMPOUNDS	3.69E+00	3.17E-03	3	3	3		8.9	0.76	0.05	0.000
67-66-3	CHLOROFORM	2.79E+01	2.59E-01	23	22	9	1.95E-01	6.4	5.95	0.80	0.007
106-46-7	DICHLOROBENZENE, P-	3.90E+01	2.87E-02	23	1	0	9.61E-01	4.3	0.32	0.05	0.000
18540-29-9	CHROMIUM (VI)	2.54E-02						3.0		0.00	
79-01-6	TRICHLOROETHYLENE	1.25E+02	2.31E-01	23	20	5	2.15E-01	2.1	0.40	5.97	0.011
75-07-0	ACETALDEHYDE	9.28E+01	2.39E+00	25	25	25		2.0	5.25	10.31	0.265
75-21-8	ETHYLENE OXIDE	1.96E+00						2.0			
75-09-2	METHYLENE CHLORIDE (DICHLOROMETHANE)	2.40E+02	4.05E+00	23	23	23		1.1	1.90	0.08	0.001
127-18-4	TETRACHLOROETHYLENE (PERCHLOROETHYLENE)	1.80E+02	5.33E-01	23	22	17	2.71E-01	1.0	0.31	5.13	0.015
50-32-8	BENZO(A)PYRENE	5.81E-02	1.21E-04	25	12	1	8.26E-04	0.6	0.13	0.00	0.000
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)	2.46E-02	0.00E+00	10	0	0		0.1	0.00	0.12	0.000
107-13-1	ACRYLONITRILE	2.46E-02						0.0		0.01	
75-56-9	PROPYLENE OXIDE	4.13E-01						0.0		0.01	
100-44-7	BENZYL CHLORIDE	2.46E-02						0.0			
56-23-5	CARBON TETRACHLORIDE	7.80E-02	8.56E-01	23	23	23		0.0	12.83	0.04	0.428
218-01-9	CHRYSENE	1.06E-01	2.60E-04	25	21	7	4.07E-04	0.0	0.00	0.00	0.000
140-88-5	ETHYL ACRYLATE	2.46E-02						0.0			
62-53-3	ANILINE	2.46E-02						0.0		0.00	
106-89-8	EPICHLOROHYDRIN (1-CHLORO-2,3-EPOXYPROPANE)	2.46E-02						0.0		0.02	
7440-41-7	BERYLLIUM COMPOUNDS	0.00E+00						0.0		0.00	
74-87-3	METHYL CHLORIDE (CHLOROMETHANE)	0.00E+00						0.0			
75-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)		1.21E-02	23	1	0	3.96E-01		0.60		0.000
79-00-5	TRICHLOROETHANE 1,1,2-		2.13E-02	23	1	0	5.45E-01		0.34		0.002
205-99-2	BENZO(B)FLUORANTHENE		1.39E-04	25	10	4	4.36E-04		0.02		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.39E-04	25	10	4	4.36E-04		0.02		0.000
56-55-3	BENZO(A)ANTHRACENE		5.90E-05	25	18	0	7.61E-04		0.01		0.000
193-39-5	INDENO(123-CD)PYRENE		4.70E-05	25	11	0	7.02E-04		0.01		0.000
53-70-3	DIBENZ(A,H)ANTHRACENE		0.00E+00	25	0	0			0.00		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		0.00E+00	25	0	0			0.00		0.000

TUCSON REGION
Screening for Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
107-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)		0.00E+00	10	0	0			0.00		0.000

TUCSON REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
107-02-8	ACROLEIN	1.13E+01	1.35E-02	25	9	3	5.23E-02			563.15	0.677
71-43-2	BENZENE (INCLUDING BENZENE FROM GASOLINE)	6.29E+02	2.99E+00	23	23	23		52.2	24.79	105.09	0.499
7439-96-5	MANGANESE COMPOUNDS	4.15E+01	1.53E-02	3	3	3				103.71	0.038
7440-39-3	BARIUM	2.58E+01	1.83E-02	3	3	0	9.54E-02			51.58	0.037
25551-13-7	TRIMETHYLBENZENE	3.22E+01								23.03	
7439-92-1	LEAD COMPOUNDS	2.00E+01	6.10E-03	3	3	3				13.34	0.004
75-07-0	ACETALDEHYDE	9.28E+01	2.39E+00	25	25	25		2.0	5.25	10.31	0.265
108-88-3	TOLUENE	2.67E+03	6.11E+00	23	23	23				6.67	0.015
108-90-7	CHLOROBENZENE	1.22E+02	2.60E-02	23	5	3	9.20E-02			6.11	0.001
79-01-6	TRICHLOROETHYLENE	1.25E+02	2.31E-01	23	20	5	2.15E-01	2.1	0.40	5.97	0.011
127-18-4	TETRACHLOROETHYLENE (PERCHLOROETHYLENE)	1.80E+02	5.33E-01	23	22	17	2.71E-01	1.0	0.31	5.13	0.015
110-54-3	HEXANE, N-	6.92E+02	3.02E+00	23	23	23				3.46	0.015
7439-97-6	MERCURY COMPOUNDS	8.17E-01	2.33E-04	3	2	0	3.70E-03			2.72	0.001
7440-28-0	THALLIUM	5.45E-01	1.67E-04	3	1	0	3.40E-03			1.95	0.001
67-56-1	METHANOL	2.54E+03								1.45	
1330-20-7	XYLENES (ISOMERS & MIXTURE)	9.40E+02	3.96E+00	23	23	23				1.34	0.006
67-66-3	CHLOROFORM	2.79E+01	2.59E-01	23	22	9	1.95E-01	6.4	5.95	0.80	0.007
71-55-6	METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)	6.88E+02	9.50E-01	23	23	23				0.69	0.001
95-47-6	XYLENES, O-	4.54E+02	1.57E+00	23	23	23				0.65	0.002
67-64-1	ACETONE	1.86E+02	6.01E-01	25	14	4	1.47E+00			0.53	0.002
7647-01-0	HYDROCHLORIC ACID (HYDROGEN CHLORIDE (GAS ONLY))	3.53E+00								0.50	
108-10-1	METHYL ISOBUTYL KETONE (HEXONE)	3.90E+01								0.49	
50-00-0	FORMALDEHYDE	3.27E+02	8.43E-01	25	25	25		42.6	10.96	0.47	0.001
7440-43-9	CADMIUM COMPOUNDS	8.04E-01	0.00E+00	3	0	0		14.5	0.00	0.46	0.000
7440-38-2	ARSENIC COMPOUNDS (INORGANIC INCLUDING ARSINE)	3.83E-01	1.07E-03	3	3	0	3.20E-03	16.5	4.59	0.36	0.001
108-38-3	XYLENES, M-	2.36E+02								0.34	
91-20-3	NAPHTHALENE	4.69E+01	3.02E-01	25	25	25				0.33	0.002
100-41-4	ETHYL BENZENE	3.19E+02	1.23E+00	23	23	23				0.32	0.001
71-36-3	BUTANOL, N-	1.09E+02								0.31	
7440-36-0	ANTIMONY COMPOUNDS	3.73E-01	5.33E-04	3	1	0	2.56E-02			0.27	0.000
78-93-3	METHYL ETHYL KETONE (2-BUTANONE)	2.23E+02	9.93E-01	25	25	21	5.30E-01			0.22	0.001
7664-41-7	AMMONIA	2.02E+01								0.20	
7782-50-5	CHLORINE	6.72E+01	7.55E-01	3	3	3				0.19	0.002
06-42-3	XYLENES, P-	5.34E+01								0.18	

TUCSON REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
123-73-9	CROTONALDEHYDE	6.05E+00	2.10E-01	25	19	11	6.65E-02			0.17	0.006
95-50-1	DICHLOROBENZENE, O-	2.74E+01	0.00E+00	23	0	0				0.14	0.000
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)	2.46E-02	0.00E+00	10	0	0		0.1	0.00	0.12	0.000
75-09-2	METHYLENE CHLORIDE (DICHLOROMETHANE)	2.40E+02	4.05E+00	23	23	23		1.1	1.90	0.08	0.001
100-42-5	STYRENE	7.48E+01	1.55E+00	23	23	23				0.07	0.002
7440-50-8	COPPER COMPOUNDS	8.99E+00	1.44E-02	3	3	3				0.07	0.000
75-15-0	CARBON DISULFIDE	6.29E-01								0.06	
7440-02-0	NICKEL COMPOUNDS	3.69E+00	3.17E-03	3	3	3		8.9	0.76	0.05	0.000
7664-93-9	SULFURIC ACID	3.65E+00								0.05	
106-46-7	DICHLOROBENZENE, P-	3.90E+01	2.87E-02	23	1	0	9.61E-01	4.3	0.32	0.05	0.000
68-12-2	DIMETHYL FORMAMIDE	1.24E+00								0.04	
56-23-5	CARBON TETRACHLORIDE	7.80E-02	8.56E-01	23	23	23		0.0	12.83	0.04	0.428
7439-98-7	MOLYBDENUM	5.84E-01	1.77E-03	3	3	1	3.40E-03			0.03	0.000
7440-66-6	ZINC	3.02E+01								0.03	
79-10-7	ACRYLIC ACID	2.46E-02								0.02	
06-89-8	EPICHLOROHYDRIN (1-CHLORO-2,3-EPOXYPROPANE)	2.46E-02						0.0		0.02	
41-78-6	ETHYL ACETATE	7.24E+01								0.02	
7440-22-4	SILVER COMPOUNDS	3.93E-01	1.83E-03	3	2	0	1.49E-02			0.02	0.000
7782-49-2	SELENIUM COMPOUNDS	3.72E-01	6.70E-05	3	1	0	1.80E-03			0.02	0.000
7440-48-4	COBALT COMPOUNDS	4.41E+00	4.00E-04	3	3	0	1.67E-02			0.02	0.000
98-82-8	CUMENE	1.28E-01								0.01	
75-56-9	PROPYLENE OXIDE	4.13E-01						0.0		0.01	
7440-24-6	STRONTIUM	2.73E+01	7.93E-03	3	3	3				0.01	0.000
107-13-1	ACRYLONITRILE	2.46E-02						0.0		0.01	
98-95-3	NITROSOBENZENE	2.46E-02								0.01	
2921-88-2	CHLORPYRIFOS	9.49E-02								0.01	
634-04-4	METHYL TERT BUTYL ETHER	2.29E+01								0.01	
92-52-4	BIPHENYL	1.32E+00	9.66E-03	25	25	25				0.01	0.000
100-52-7	BENZALDEHYDE	1.53E+00	5.67E-01	25	24	21	1.28E-01			0.00	0.002
107-21-1	ETHYLENE GLYCOL	2.29E+01								0.00	
111-15-9	ETHOXY ETHYL ACETATE, 2-	3.23E+00								0.00	
7440-47-3	CHROMIUM, TOTAL	9.05E+00	2.77E-03	3	3	1	5.20E-03			0.00	0.000
57-55-6	PROPANEDIOL, 1,2-	1.57E+02								0.00	
7782-41-4	FLUORINE (AS FLUORIDE)	3.47E-01								0.00	

TUCSON REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
18540-29-9	CHROMIUM (VI)	2.54E-02						3.0		0.00	
78-83-1	ISOBUTYL ALCOHOL	1.52E+00								0.00	
218-01-9	CHRYSENE	1.06E-01	2.60E-04	25	21	7	4.07E-04	0.0	0.00	0.00	0.000
80-62-6	METHYL METHACRYLATE	1.68E-01								0.00	
50-32-8	BENZO(A)PYRENE	5.81E-02	1.21E-04	25	12	1	8.26E-04	0.6	0.13	0.00	0.000
7440-42-8	BORON	1.04E-02								0.00	
206-44-0	FLUORANTHENE	4.76E-02	5.58E-03	25	25	25				0.00	0.000
108-94-1	CYCLOHEXANONE	5.13E+00								0.00	
62-53-3	ANILINE	2.46E-02						0.0		0.00	
96-33-3	METHYL ACRYLATE	2.46E-02								0.00	
75-00-3	ETHYL CHLORIDE (CHLOROETHANE)	1.95E+00								0.00	
16752-77-5	METHOMYL	1.37E-02								0.00	
7440-31-5	TIN AND OXIDE COMPOUNDS	2.58E-01	3.53E-03	3	3	0	2.25E-02			0.00	0.000
108-95-2	PHENOL	2.38E-01								0.00	
116-06-3	ALDICARB	2.64E-04								0.00	
60-29-7	ETHYL ETHER	2.46E-02								0.00	
1563-66-2	CARBOFURAN	3.00E-04								0.00	
79-20-9	METHYL ACETATE	2.46E-02								0.00	
64-18-6	FORMIC ACID	2.46E-02								0.00	
7440-41-7	BERYLLIUM COMPOUNDS	0.00E+00						0.0		0.00	
84-74-2	DIBUTYLPHTHALATE	0.00E+00								0.00	
1910-42-5	PARAQUAT	0.00E+00								0.00	
74-83-9	METHYL BROMIDE (BROMOMETHANE)		1.57E-01	23	2	2					0.031
79-00-5	TRICHLOROETHANE 1,1,2-		2.13E-02	23	1	0	5.45E-01		0.34		0.002
91-57-6	2-METHYLNAPHTHALENE (NAPHTHALENE AS SURROGATE)		1.78E-01	25	25	25					0.001
7439-89-6	IRON (SOLUBLE COMPOUNDS)		6.40E-01	3	3	3					0.001
7429-90-5	ALUMINUM		2.11E+00	3	3	3					0.001
MNAPH1	1-METHYLNAPHTHALENE, 1-		8.11E-02	25	25	25					0.001
75-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)		1.21E-02	23	1	0	3.96E-01		0.60		0.000
DM1367	DIMETHYLNAPHTHALENE, 1,7+1,3+1,6-		4.43E-02	25	25	25					0.000
DMN267	2,6+2,7-DIMETHYLNAPHTHALENE (NAPHTHALENE AS SURROGATE)		2.12E-02	25	25	25					0.000
D14523	DIMETHYLNAPHTHALENE, 2,3+1,4+1,5-		1.11E-02	25	25	23	4.05E-03				0.000
DMN12	DIMETHYLNAPHTHALENE, 1,2-		8.30E-03	25	25	20	2.30E-03				0.000
83-32-9	ACENAPHTHENE		6.10E-03	25	23	23					0.000

TUCSON REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
129-00-0	PYRENE		2.67E-03	25	25	25					0.000
85-01-8	PHENANTHRENE		1.80E-02	25	25	25					0.000
208-96-8	ACENAPHTHYLENE		2.44E-03	25	23	12	1.70E-03				0.000
120-12-7	ANTHRACENE		3.23E-03	25	23	23					0.000
191-24-2	BENZO(GHI)PERYLENE		2.93E-04	25	22	0	9.24E-04				0.000
DMN18	DIMETHYLNAPHTHALENE, 1,8-		3.09E-04	25	10	2	1.45E-03				0.000
205-99-2	BENZO(B)FLUORANTHENE		1.39E-04	25	10	4	4.36E-04		0.02		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.39E-04	25	10	4	4.36E-04		0.02		0.000
86-73-7	FLUORENE		8.50E-03	25	25	25					0.000
56-55-3	BENZO(A)ANTHRACENE		5.90E-05	25	18	0	7.61E-04		0.01		0.000
BBCHRN	BENZO(B)CHRYSENE		5.20E-05	25	7	0	5.32E-04				0.000
193-39-5	INDENO(123-CD)PYRENE		4.70E-05	25	11	0	7.02E-04		0.01		0.000
53-70-3	DIBENZ(A,H)ANTHRACENE		0.00E+00	25	0	0			0.00		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		0.00E+00	25	0	0			0.00		0.000
56-59-2	DICHLOROETHENE, 1,2-,CIS-		0.00E+00	23	0	0					0.000
07-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)		0.00E+00	10	0	0			0.00		0.000

CASA GRANDE REGION
Screening for Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
106-99-0	BUTADIENE, 1,3-	1.20E+01	1.40E-01	25	24	24		33.7	39.12		
71-43-2	BENZENE (INCLUDING BENZENE FROM GASOLINE)	9.89E+01	1.46E+00	25	24	24		8.2	12.14	16.52	0.244
50-00-0	FORMALDEHYDE	6.28E+01	6.23E-01	25	24	22	1.80E-01	8.2	8.10	0.09	0.001
18540-29-9	CHROMIUM (VI)	4.10E-02						4.9		0.00	
7440-38-2	ARSENIC COMPOUNDS (INORGANIC INCLUDING ARSINE)	7.29E-02	2.43E-04	14	9	0	3.11E-03	3.1	1.04	0.07	0.000
7440-43-9	CADMIUM COMPOUNDS	1.55E-01	6.86E-04	14	8	0	1.54E-02	2.8	1.23	0.09	0.000
7440-02-0	NICKEL COMPOUNDS	4.37E-01	1.69E-03	14	14	3	2.64E-03	1.0	0.41	0.01	0.000
50-32-8	BENZO(A)PYRENE	8.41E-02	2.45E-04	24	12	1	8.11E-04	0.9	0.27	0.00	0.000
106-46-7	DICHLOROBENZENE, P-	8.36E+00	0.00E+00	25	0	0		0.9	0.00	0.01	0.000
82-68-8	PENTACHLORONITROBENZENE	8.26E-01						0.6		0.08	
75-07-0	ACETALDEHYDE	1.64E+01	2.30E+00	25	25	25		0.4	5.06	1.83	0.255
79-01-6	TRICHLOROETHYLENE	1.42E+01	6.19E-01	25	25	25		0.2	1.06	0.68	0.029
67-66-3	CHLOROFORM	5.74E-01	1.54E-01	25	24	8	1.95E-01	0.1	3.55	0.02	0.004
75-09-2	METHYLENE CHLORIDE (DICHLOROMETHANE)	2.33E+01	3.05E+00	25	25	25		0.1	1.43	0.01	0.001
1582-09-8	TRIFLURALIN	2.41E+00						0.1		0.09	
127-18-4	TETRACHLOROETHYLENE (PERCHLOROETHYLENE)	7.42E+00	1.74E-01	25	21	7	2.71E-01	0.0	0.10	0.21	0.005
75-21-8	ETHYLENE OXIDE	2.11E-02						0.0			
7440-41-7	BERYLLIUM COMPOUNDS	5.40E-04						0.0		0.00	
75-56-9	PROPYLENE OXIDE	5.84E-02						0.0		0.00	
218-01-9	CHRYSENE	1.31E-02	2.36E-04	24	18	5	3.70E-04	0.0	0.00	0.00	0.000
56-23-5	CARBON TETRACHLORIDE	8.04E-03	7.12E-01	25	25	25		0.0	10.67	0.00	0.356
74-87-3	METHYL CHLORIDE (CHLOROMETHANE)	0.00E+00						0.0			
79-00-5	TRICHLOROETHANE 1,1,2-		1.66E-01	25	12	2	5.45E-01		2.65		0.012
75-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)		4.44E-02	25	5	0	3.96E-01		2.22		0.001
205-99-2	BENZO(B)FLUORANTHENE		1.74E-04	24	7	1	4.24E-04		0.02		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.74E-04	24	7	1	4.24E-04		0.02		0.000
53-70-3	DIBENZ(A,H)ANTHRACENE		7.00E-06	24	1	0	1.01E-03		0.01		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		7.00E-06	24	1	0	1.01E-03		0.01		0.000
193-39-5	INDENO(123-CD)PYRENE		8.40E-05	24	10	1	6.55E-04		0.01		0.000
56-55-3	BENZO(A)ANTHRACENE		8.10E-05	24	19	0	6.95E-04		0.01		0.000
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)		0.00E+00	10	0	0			0.00		0.000
107-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)		0.00E+00	10	0	0			0.00		0.000

CASA GRANDE REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
107-02-8	ACROLEIN	1.78E+00	8.00E-03	25	9	0	5.68E-02			89.03	0.400
71-43-2	BENZENE (INCLUDING BENZENE FROM GASOLINE)	9.89E+01	1.46E+00	25	24	24		8.2	12.14	16.52	0.244
298-00-0	METHYL PARATHION	1.25E+01								14.28	
7439-96-5	MANGANESE COMPOUNDS	4.81E+00	1.79E-02	14	14	13	2.60E-03			12.03	0.045
7440-39-3	BARIUM	2.93E+00	9.26E-03	14	11	0	9.30E-02			5.86	0.019
298-04-4	DISULFOTON	8.04E-01								5.74	
7439-92-1	LEAD COMPOUNDS	3.28E+00	4.06E-03	14	14	11	3.13E-03			2.19	0.003
75-07-0	ACETALDEHYDE	1.64E+01	2.30E+00	25	25	25		0.4	5.06	1.83	0.255
25551-13-7	TRIMETHYLBENZENE	2.56E+00								1.83	
7664-41-7	AMMONIA	1.36E+02								1.36	
108-88-3	TOLUENE	5.32E+02	5.86E+00	25	25	25				1.33	0.015
2921-88-2	CHLORPYRIFOS	1.36E+01								1.29	
67-64-1	ACETONE	2.65E+02	6.57E-01	25	15	5	1.59E+00			0.76	0.002
79-01-6	TRICHLOROETHYLENE	1.42E+01	6.19E-01	25	25	25		0.2	1.06	0.68	0.029
108-90-7	CHLOROBENZENE	1.24E+01	2.58E-02	25	4	2	9.20E-02			0.62	0.001
110-54-3	HEXANE, N-	1.15E+02	5.67E+00	25	25	25				0.58	0.028
7439-97-6	MERCURY COMPOUNDS	9.37E-02	4.64E-04	14	13	0	3.52E-03			0.31	0.002
7440-28-0	THALLIUM	8.14E-02	3.57E-04	14	11	0	3.24E-03			0.29	0.001
1330-20-7	XYLENES (ISOMERS & MIXTURE)	1.61E+02	2.91E+00	25	25	25				0.23	0.004
127-18-4	TETRACHLOROETHYLENE (PERCHLOROETHYLENE)	7.42E+00	1.74E-01	25	21	7	2.71E-01	0.0	0.10	0.21	0.005
116-06-3	ALDICARB	7.20E-01								0.21	
67-56-1	METHANOL	2.53E+02								0.14	
121-75-5	MALATHION	8.57E+00								0.12	
115-29-7	ENDOSULFAN	2.49E+00								0.12	
78-93-3	METHYL ETHYL KETONE (2-BUTANONE)	1.06E+02	9.45E-01	25	25	21	5.28E-01			0.11	0.001
298-02-2	PHORATE	6.47E-02								0.09	
1582-09-8	TRIFLURALIN	2.41E+00						0.1		0.09	
95-47-6	XYLENES, O-	6.36E+01	1.21E+00	25	25	25				0.09	0.002
50-00-0	FORMALDEHYDE	6.28E+01	6.23E-01	25	24	22	1.80E-01	8.2	8.10	0.09	0.001
7440-43-9	CADMIUM COMPOUNDS	1.55E-01	6.86E-04	14	8	0	1.54E-02	2.8	1.23	0.09	0.000
7440-36-0	ANTIMONY COMPOUNDS	1.16E-01	1.19E-03	14	6	0	2.58E-02			0.08	0.001
82-68-8	PENTACHLORONITROBENZENE	8.26E-01						0.6		0.08	
71-55-6	METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)	7.08E+01	6.83E+00	25	25	25				0.07	0.007
7440-38-2	ARSENIC COMPOUNDS (INORGANIC INCLUDING ARSINE)	7.29E-02	2.43E-04	14	9	0	3.11E-03	3.1	1.04	0.07	0.000

CASA GRANDE REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
71-36-3	BUTANOL, N-	2.02E+01								0.06	
91-20-3	NAPHTHALENE	7.57E+00	2.39E-01	24	24	24				0.05	0.002
100-41-4	ETHYL BENZENE	4.60E+01	8.19E-01	25	25	25				0.05	0.001
108-10-1	METHYL ISOBUTYL KETONE (HEXONE)	3.53E+00								0.04	
123-73-9	CROTONALDEHYDE	1.11E+00	9.82E-02	25	20	12	7.32E-02			0.03	0.003
7782-50-5	CHLORINE	1.05E+01	4.48E-01	14	14	14				0.03	0.001
108-38-3	XYLENES, M-	1.95E+01								0.03	
7440-50-8	COPPER COMPOUNDS	2.87E+00	4.67E-02	14	14	14				0.02	0.000
106-42-3	XYLENES, P-	5.19E+00								0.02	
67-66-3	CHLOROFORM	5.74E-01	1.54E-01	25	24	8	1.95E-01	0.1	3.55	0.02	0.004
95-50-1	DICHLOROBENZENE, O-	2.77E+00	0.00E+00	25	0	0				0.01	0.000
333-41-5	DIAZINON	4.23E-02								0.01	
16752-77-5	METHOMYL	1.12E+00								0.01	
100-42-5	STYRENE	1.19E+01	1.50E+00	25	25	25				0.01	0.002
106-46-7	DICHLOROBENZENE, P-	8.36E+00	0.00E+00	25	0	0		0.9	0.00	0.01	0.000
1563-66-2	CARBOFURAN	1.59E-01								0.01	
75-09-2	METHYLENE CHLORIDE (DICHLOROMETHANE)	2.33E+01	3.05E+00	25	25	25		0.1	1.43	0.01	0.001
7440-02-0	NICKEL COMPOUNDS	4.37E-01	1.69E-03	14	14	3	2.64E-03	1.0	0.41	0.01	0.000
7439-98-7	MOLYBDENUM	9.75E-02	4.93E-04	14	12	0	3.55E-03			0.01	0.000
7440-66-6	ZINC	4.93E+00								0.00	
141-78-6	ETHYL ACETATE	1.31E+01								0.00	
56-23-5	CARBON TETRACHLORIDE	8.04E-03	7.12E-01	25	25	25		0.0	10.67	0.00	0.356
7664-93-9	SULFURIC ACID	2.55E-01								0.00	
68-12-2	DIMETHYL FORMAMIDE	1.00E-01								0.00	
7782-49-2	SELENIUM COMPOUNDS	5.76E-02	1.64E-04	14	10	0	1.62E-03			0.00	0.000
98-82-8	CUMENE	2.78E-02								0.00	
18540-29-9	CHROMIUM (VI)	4.10E-02						4.9		0.00	
7440-48-4	COBALT COMPOUNDS	4.42E-01	3.50E-04	14	12	0	1.93E-02			0.00	0.000
7440-22-4	SILVER COMPOUNDS	3.61E-02	2.76E-03	14	14	0	1.44E-02			0.00	0.000
75-56-9	PROPYLENE OXIDE	5.84E-02						0.0		0.00	
92-52-4	BIPHENYL	2.75E-01	9.04E-03	24	24	24				0.00	0.000
7440-24-6	STRONTIUM	3.22E+00	8.07E-03	14	14	14				0.00	0.000
57-55-6	PROPANEDIOL, 1,2-	1.01E+02								0.00	
1634-04-4	METHYL TERT BUTYL ETHER	2.76E+00								0.00	

CASA GRANDE REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
50-32-8	BENZO(A)PYRENE	8.41E-02	2.45E-04	24	12	1	8.11E-04	0.9	0.27	0.00	0.000
7782-41-4	FLUORINE (AS FLUORIDE)	9.48E-02								0.00	
100-52-7	BENZALDEHYDE	1.55E-01	7.39E-01	25	20	19	1.52E-01			0.00	0.002
7440-47-3	CHROMIUM, TOTAL	1.07E+00	1.81E-03	14	14	1	5.37E-03			0.00	0.000
107-21-1	ETHYLENE GLYCOL	1.87E+00								0.00	
7440-42-8	BORON	4.99E-03								0.00	
111-15-9	ETHOXY ETHYL ACETATE, 2-	2.60E-01								0.00	
63-25-2	CARBARYL	6.13E-02								0.00	
21087-64-9	METRIBUZIN	1.18E-02								0.00	
218-01-9	CHRYSENE	1.31E-02	2.36E-04	24	18	5	3.70E-04	0.0	0.00	0.00	0.000
78-83-1	ISOBUTYL ALCOHOL	1.20E-01								0.00	
80-62-6	METHYL METHACRYLATE	2.53E-02								0.00	
85-00-7	DIQUAT	3.77E-04								0.00	
7440-31-5	TIN AND OXIDE COMPOUNDS	9.54E-02	1.41E-03	14	11	0	2.16E-02			0.00	0.000
206-44-0	FLUORANTHENE	5.96E-03	5.33E-03	24	24	24				0.00	0.000
7440-41-7	BERYLLIUM COMPOUNDS	5.40E-04						0.0		0.00	
108-94-1	CYCLOHEXANONE	5.13E-01								0.00	
75-00-3	ETHYL CHLORIDE (CHLOROETHANE)	1.57E-01								0.00	
108-95-2	PHENOL	1.23E-02								0.00	
7647-01-0	HYDROCHLORIC ACID (HYDROGEN CHLORIDE (GAS ONLY))	0.00E+00								0.00	
1910-42-5	PARAQUAT	0.00E+00								0.00	
74-83-9	METHYL BROMIDE (BROMOMETHANE)		6.83E-02	25	3	2	3.88E-01				0.014
79-00-5	TRICHLOROETHANE 1,1,2-		1.66E-01	25	12	2	5.45E-01		2.65		0.012
75-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)		4.44E-02	25	5	0	3.96E-01		2.22		0.001
91-57-6	2-METHYLNAPHTHALENE (NAPHTHALENE AS SURROGATE)		1.50E-01	24	24	24					0.001
7439-89-6	IRON (SOLUBLE COMPOUNDS)		7.14E-01	14	14	14					0.001
MNAPH1	1-METHYLNAPHTHALENE, 1-		6.82E-02	24	24	24					0.000
7429-90-5	ALUMINUM		1.55E+00	14	14	14					0.000
DM1367	DIMETHYLNAPHTHALENE, 1,7+1,3+1,6-		3.40E-02	24	24	24					0.000
DMN267	2,6+2,7-DIMETHYLNAPHTHALENE (NAPHTHALENE AS SURROGATE)		1.76E-02	24	24	24					0.000
D14523	DIMETHYLNAPHTHALENE, 2,3+1,4+1,5-		1.00E-02	24	24	24					0.000
DMN12	DIMETHYLNAPHTHALENE, 1,2-		9.40E-03	24	24	23	2.16E-03				0.000
83-32-9	ACENAPHTHENE		7.79E-03	24	24	24					0.000
129-00-0	PYRENE		2.66E-03	24	24	24					0.000

CASA GRANDE REGION
Screening for Non-Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
85-01-8	PHENANTHRENE		1.80E-02	24	24	24					0.000
208-96-8	ACENAPHTHYLENE		1.84E-03	24	20	13	1.59E-03				0.000
120-12-7	ANTHRACENE		2.94E-03	24	24	24					0.000
191-24-2	BENZO(GHI)PERYLENE		2.78E-04	24	22	1	8.30E-04				0.000
205-99-2	BENZO(B)FLUORANTHENE		1.74E-04	24	7	1	4.24E-04		0.02		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.74E-04	24	7	1	4.24E-04		0.02		0.000
193-39-5	INDENO(123-CD)PYRENE		8.40E-05	24	10	1	6.55E-04		0.01		0.000
56-55-3	BENZO(A)ANTHRACENE		8.10E-05	24	19	0	6.95E-04		0.01		0.000
86-73-7	FLUORENE		8.93E-03	24	24	24					0.000
BBCHRN	BENZO(B)CHRYSENE		4.70E-05	24	7	0	4.70E-04				0.000
DMN18	DIMETHYLNAPHTHALENE, 1,8-		3.00E-05	24	7	0	1.29E-03				0.000
53-70-3	DIBENZ(A,H)ANTHRACENE		7.00E-06	24	1	0	1.01E-03		0.01		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		7.00E-06	24	1	0	1.01E-03		0.01		0.000
156-59-2	DICHLOROETHENE, 1,2-,CIS-		0.00E+00	25	0	0					0.000
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)		0.00E+00	10	0	0			0.00		0.000
107-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)		0.00E+00	10	0	0			0.00		0.000

PAYSON REGION
Screening for Carcinogens

CAS No.	Species	Annual Emiss. (t/yr)	Avg. Concen. (ug/m3)	No. Mea	No. Det.	No. Qua	Avg. LQL (ug/m3)	Carcino-genic ERI	Screen ELCR (per mill.)	Non-Carcin. ERI	Screen Hazard Quot.
106-99-0	BUTADIENE, 1,3-	4.56E+00	2.23E-01	35	31	31		12.8	62.43		
50-00-0	FORMALDEHYDE	2.41E+01	8.77E-01	38	38	35	1.79E-01	3.1	11.40	0.03	0.001
71-43-2	BENZENE (INCLUDING BENZENE FROM GASOLINE)	3.30E+01	2.51E+00	35	35	35		2.7	20.83	5.51	0.419
7440-43-9	CADMIUM COMPOUNDS	5.09E-02	2.53E-03	22	20	0	1.69E-02	0.9	4.56	0.03	0.001
7440-38-2	ARSENIC COMPOUNDS (INORGANIC INCLUDING ARSINE)	1.15E-02	9.86E-04	22	21	1	3.69E-03	0.5	4.24	0.01	0.001
18540-29-9	CHROMIUM (VI)	3.86E-03						0.5		0.00	
7440-02-0	NICKEL COMPOUNDS	1.04E-01	8.09E-04	22	21	0	3.13E-03	0.3	0.19	0.00	0.000
50-32-8	BENZO(A)PYRENE	1.74E-02	7.99E-04	34	23	11	8.89E-04	0.2	0.88	0.00	0.000
75-07-0	ACETALDEHYDE	4.16E+00	2.55E+00	38	38	38		0.1	5.60	0.46	0.283
106-46-7	DICHLOROBENZENE, P-	3.48E-01	4.81E-02	35	3	0	9.61E-01	0.0	0.53	0.00	0.000
79-01-6	TRICHLOROETHYLENE	1.24E+00	5.69E-01	35	21	7	2.15E-01	0.0	0.98	0.06	0.027
67-66-3	CHLOROFORM	7.38E-02	1.13E-01	35	30	1	1.95E-01	0.0	2.60	0.00	0.003
75-09-2	METHYLENE CHLORIDE (DICHLOROMETHANE)	2.37E+00	4.31E+00	35	34	34		0.0	2.02	0.00	0.001
75-21-8	ETHYLENE OXIDE	8.31E-03						0.0			
127-18-4	TETRACHLOROETHYLENE (PERCHLOROETHYLENE)	1.39E+00	5.96E-01	35	30	25	2.71E-01	0.0	0.35	0.04	0.017
218-01-9	CHRYSENE	3.11E-02	1.09E-03	34	28	13	3.73E-04	0.0	0.01	0.00	0.000
75-56-9	PROPYLENE OXIDE	5.14E-03						0.0		0.00	
56-23-5	CARBON TETRACHLORIDE	7.08E-04	7.27E-01	35	35	35		0.0	10.91	0.00	0.364
74-87-3	METHYL CHLORIDE (CHLOROMETHANE)	0.00E+00						0.0			
53-70-3	DIBENZ(A,H)ANTHRACENE		9.20E-05	34	8	0	9.01E-04		0.19		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		9.20E-05	34	8	0	9.01E-04		0.19		0.000
205-99-2	BENZO(B)FLUORANTHENE		1.22E-03	34	20	13	4.43E-04		0.13		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.22E-03	34	20	13	4.43E-04		0.13		0.000
56-55-3	BENZO(A)ANTHRACENE		9.57E-04	34	29	9	7.56E-04		0.11		0.000
193-39-5	INDENO(123-CD)PYRENE		4.42E-04	34	20	10	7.14E-04		0.05		0.000
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)		0.00E+00	9	0	0			0.00		0.000
107-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)		0.00E+00	9	0	0			0.00		0.000
79-00-5	TRICHLOROETHANE 1,1,2-		0.00E+00	35	0	0			0.00		0.000
75-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)		0.00E+00	35	0	0			0.00		0.000

THYL KETONE (2-BUTANONE)	2.50E+00	1.11E+00	38	38	29	5.02E-01			0.00	0.001			
P-	7.04E-01								0.00				
ORM	7.38E-02	1.13E-01	35	30	1	1.95E-01	0.0	2.60	0.00	0.003			
OMPOUNDS	1.04E-01	8.09E-04	22	21	0	3.13E-03	0.3	0.19	0.00	0.000			
OBENZENE, O-	2.79E-01	0.00E+00	35	0	0				0.00	0.000			
	1.32E+00								0.00				
NUM	2.19E-02	4.86E-04	22	15	0	3.96E-03			0.00	0.000			
OMPOUNDS	1.61E-02	2.30E-03	22	19	0	1.58E-02			0.00	0.000			
NE CHLORIDE (DICHLOROMETHANE)	2.37E+00	4.31E+00	35	34	34		0.0	2.02	0.00	0.001			
	5.68E-03								0.00				
COMPOUNDS	8.38E-03	1.41E-04	22	14	0	1.80E-03			0.00	0.000			
OMPOUNDS	9.42E-02	4.36E-04	22	17	0	1.84E-02			0.00	0.000			
FORMAMIDE	1.33E-02								0.00				
OBENZENE, P-	3.48E-01	4.81E-02	35	3	0	9.61E-01	0.0	0.53	0.00	0.000			
JM	9.00E-01	5.37E-03	22	22	22				0.00	0.000			
TETRACHLORIDE	7.08E-04	7.27E-01	35	35	35		0.0	10.91	0.00	0.364			
E	3.11E-02	1.09E-03	34	28	13	3.73E-04	0.0	0.01	0.00	0.000			
M (VI)	3.86E-03						0.5		0.00				
(AS FLUORIDE)	3.68E-02								0.00				
NE OXIDE	5.14E-03						0.0		0.00				
PYRENE	1.74E-02	7.99E-04	34	23	11	8.89E-04	0.2	0.88	0.00	0.000			
THENE	1.43E-02	5.31E-03	34	34	34				0.00	0.000			
ERT BUTYL ETHER	2.47E-01								0.00				
M, TOTAL	2.61E-01	1.18E-03	22	19	0	6.30E-03			0.00	0.000			
ETATE	1.74E-01								0.00				
EHYDE	1.51E-02	6.28E-01	38	35	27	1.36E-01			0.00	0.002			
E GLYCOL	2.46E-01								0.00				
THYL ACETATE, 2-	3.47E-02								0.00				
ALCOHOL	1.60E-02								0.00				
OXIDE COMPOUNDS	2.46E-02	1.61E-03	22	14	0	2.38E-02			0.00	0.000			
	2.14E-04								0.00				
METHACRYLATE	2.23E-03								0.00				
DIOL, 1,2-	4.80E-01								0.00				
XANONE	5.27E-02								0.00				
LORIDE (CHLOROETHANE)	2.10E-02								0.00				
	5.16E-04								0.00				
NAPHTHALENE (NAPHTHALENE AS SURROGATE)		2.56E-01	34	34	34					0.002			
NAPHTHALENE, 1-		1.18E-01	34	34	34					0.001			
LUBLE COMPOUNDS)		6.91E-01	22	22	22					0.001			
NAPHTHALENE, 1,7+1,3+1,6-		5.22E-02	34	34	34					0.000			
M		1.29E+00	22	22	22					0.000			

	Screen Hazard Quot.
0.03	0.001
5.51	0.419
0.03	0.001
0.01	0.001
0.00	
0.00	0.000
0.00	0.000
0.46	0.283
0.00	0.000
0.06	0.027
0.00	0.003
0.00	0.001
0.04	0.017
0.00	0.000
0.00	
0.00	0.364
	0.000
	0.000
	0.000
	0.000
	0.000
	0.000
	0.000
	0.000
	0.000
	0.000

DMN267	2,6+2,7-DIMETHYLNAPHTHALENE (NAPHTHALENE AS SURROGATE)		2.41E-02	34	34	34					0.000
D14523	DIMETHYLNAPHTHALENE, 2,3+1,4+1,5-		1.63E-02	34	33	28	2.64E-03				0.000
DMN12	DIMETHYLNAPHTHALENE, 1,2-		1.13E-02	34	33	27	1.61E-03				0.000
208-96-8	ACENAPHTHYLENE		1.19E-02	34	31	25	1.64E-03				0.000
83-32-9	ACENAPHTHENE		9.25E-03	34	29	29					0.000
129-00-0	PYRENE		4.06E-03	34	34	34					0.000
85-01-8	PHENANTHRENE		1.95E-02	34	34	34					0.000
205-99-2	BENZO(B)FLUORANTHENE		1.22E-03	34	20	13	4.43E-04		0.13		0.000
205-99-2	BENZO(B+J+K)FLUORANTHENE		1.22E-03	34	20	13	4.43E-04		0.13		0.000
56-55-3	BENZO(A)ANTHRACENE		9.57E-04	34	29	9	7.56E-04		0.11		0.000
191-24-2	BENZO(GHI)PERYLENE		8.24E-04	34	31	12	9.14E-04				0.000
193-39-5	INDENO(123-CD)PYRENE		4.42E-04	34	20	10	7.14E-04		0.05		0.000
120-12-7	ANTHRACENE		2.92E-03	34	32	32					0.000
DMN18	DIMETHYLNAPHTHALENE, 1,8-		3.15E-04	34	16	3	1.44E-03				0.000
BBCHRN	BENZO(B)CHRYSENE		9.80E-05	34	11	2	4.68E-04				0.000
53-70-3	DIBENZ(A,H)ANTHRACENE		9.20E-05	34	8	0	9.01E-04		0.19		0.000
53-70-3	DIBENZO(AH+AC)ANTHRACENE		9.20E-05	34	8	0	9.01E-04		0.19		0.000
86-73-7	FLUORENE		1.21E-02	34	34	34					0.000
156-59-2	DICHLOROETHENE, 1,2-,CIS-		0.00E+00	35	0	0					0.000
106-93-4	ETHYLENE DIBROMIDE (DIBROMOETHANE)		0.00E+00	9	0	0			0.00		0.000
107-06-2	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)		0.00E+00	9	0	0			0.00		0.000
74-83-9	METHYL BROMIDE (BROMOMETHANE)		0.00E+00	35	0	0					0.000
79-00-5	TRICHLOROETHANE 1,1,2-		0.00E+00	35	0	0			0.00		0.000
75-35-4	VINYLIDENE CHLORIDE (DICHLOROETHYLENE)		0.00E+00	35	0	0			0.00		0.000

APPENDIX C

ATMOSPHERIC SIMULATION MODEL ADJUSTMENT

APPENDIX C - ATMOSPHERIC SIMULATION MODEL ADJUSTMENT

1.0 INTRODUCTION AND GENERAL APPROACH

The atmospheric simulation model was used to estimate hourly and annual-average HAP concentrations in the Phoenix, Tucson and Casa Grande study regions. Adjustments were made to the model predictions to account for background concentrations and to avoid substantial biases between the predictions and available measurements. This appendix describes the approach used for making the adjustments and the specific adjustment factors that were used for each of the three study regions.

1.1 Background Concentrations

The atmospheric simulation model only accounted for the effects on concentrations of HAP emissions within the study regions. Concentrations at the boundaries of the modeling domains were set to zero, as were the initial concentrations at the beginnings of the simulations. However, concentrations at the boundaries are not zero, as shown by the measurements made at the Hillside regional background measurement location.

In order to account for these background concentrations, average concentrations measured at the Hillside site were added to the simulation results. The available data from Hillside included measurements made every 12 days from November 1994 through August 1995. Table 1-1 lists the average concentrations that were measured at Hillside and the background values that were applied to the model results. The background value was set to zero when the standard deviation of the Hillside data exceeded 1.5 times the average value. Although the standard deviation of the formaldehyde average was less than 1.5 times the average concentration, it was found that a large portion of the formaldehyde measured at Hillside was apparently emitted by materials within the sampling shelter. This was found by making simultaneous measurements with all of the sampling equipment outside the shelter. Therefore, the background concentration for formaldehyde was set to zero.

1.2 Adjustment for Bias

Comparisons between the simulation model results, with the background values added, and 24-hour average measurements for the Phoenix study region (the only region with simultaneous measurements and simulations) showed that some HAPs were consistently

TABLE 1-1

Average Concentrations Measured at the Hillside Regional Background Site and Model Background Values

Species	Average Concentration ($\mu g/m^3$)	Standard Deviation ($\mu g/m^3$)	Background Applied to Simulation Model ($\mu g/m3$)
Acetaldehyde	1.50	0.86	1.5
Acrolein	0.021	0.027	0
Benzo(a)pyrene	0.000005	0.000017	0
Benzene	0.29	0.11	0.29
1,3-Butadiene	0.016	0.072	0
Chloroform	0.070	0.059	0
Dichloromethane	2.5	0.2	2.5
Formaldehyde	1.7	0.4	0
n-Hexane	0.46	0.15	0.46
p-Dichlorobenzene	0.0013	0.0009	0
Tetrachloroethylene	0.016	0.042	0
Toluene	0.25	0.11	0.25

over- or under-predicted. These biases could have been caused by one or more of the following factors:

- The model may not simulate the transport and dispersion of emissions correctly. However, as shown in Section 2, the simulation results agreed well with the measurements for some chemical species, which suggests that the treatment of these processes is reasonably accurate.

- Emission estimates may be incorrect. It is possible that the estimates of total emissions within the region may be biased high or low. Additionally, the estimated geographic distribution of emissions may also be incorrect, which could lead to inaccuracies in the simulation results for individual locations, such as the Phoenix residential neighborhood site. Although emissions from individual point sources were specified, emissions from dispersed area sources were distributed geographically using surrogates for the actual locations of these emissions. Uncertainties may have been introduced by the use of these surrogates.

- Atmospheric formation and transformation of HAPs could introduce errors. The simulation model does not account for atmospheric chemical reactions that can form or destroy a chemical species. This leads to inaccuracies for some species that are highly reactive, such as acrolein, 1,3-butadiene and formaldehyde.

In order to avoid substantial biases in the concentrations used for the risk evaluations, the model predictions were adjusted by HAP-specific multiplicative factors such that the predictions and measurement results agreed, on average. Thus, the final, adjusted model concentration was calculated by:

$$C_{a,l} = C_{b,l} + A_l C_{m,l} \qquad (1)$$

where $C_{a,l}$ is the adjusted model result for chemical species l, $C_{b,l}$ is the background concentration, A_l is the model adjustment factor and $C_{m,l}$ is the unadjusted model result.

2.0 DEVELOPMENT OF MODEL ADJUSTMENT FACTORS

All of the simulations were made for 1994, which is the most recent full year for which the meteorological data needed by the model were available. Because measurements did not begin at the same time in all of the regions, the approach used to develop the adjustment factors varied among the regions.

2.1 Adjustment Factors and Comparison with Measurements for Phoenix

Measurements began at the Phoenix residential neighborhood site in April 1994, so simultaneous measurements and model predictions were available for nine month of 1994. The model adjustment factors for Phoenix were calculated by dividing the averages of the concentrations measured during these nine months, after correcting them for the background values, by the averages of the model predictions for the days on which the measurements were made.

Table 2-1 lists the adjustment factors thus derived for the Phoenix region, along with the average measured concentrations, the background concentrations, the average measured non-background concentrations, and the average unadjusted model result. Many of the adjustment factors are close to one, most notably the factors for benzene (0.97) and 1,3-butadiene (0.75). The majority of the factors are between 0.5 and 2, which means that the model predicted the non-background concentrations within a factor of two, on average, for most of the HAPs. The factors for some of the chemical species are substantially different from one, which suggests that there could be substantial uncertainties in emission rates or locations or in the model predictions.

Comparisons between measurements and the adjusted model results for individual days provide additional indications of how the model performed. The adjusted results for individual days agreed with the measured concentrations within a factor of two for acetaldehyde, benzene, 1,3-butadiene and dichloromethane, as shown in Figures 2-1 through 2-4. Agreement on individual days was poorer for the other species, as shown in Figures 2-5 through 2-10.

The deviations for formaldehyde (Figure 2-8) probably reflect the effects of atmospheric chemical reactions, while the deviations for chloroform (Figure 2-6) may be caused by seasonal differences in emission rate that are not accounted for in the emission estimates. The deviations for tetrachloroethylene may be caused by limitations in the treatment of

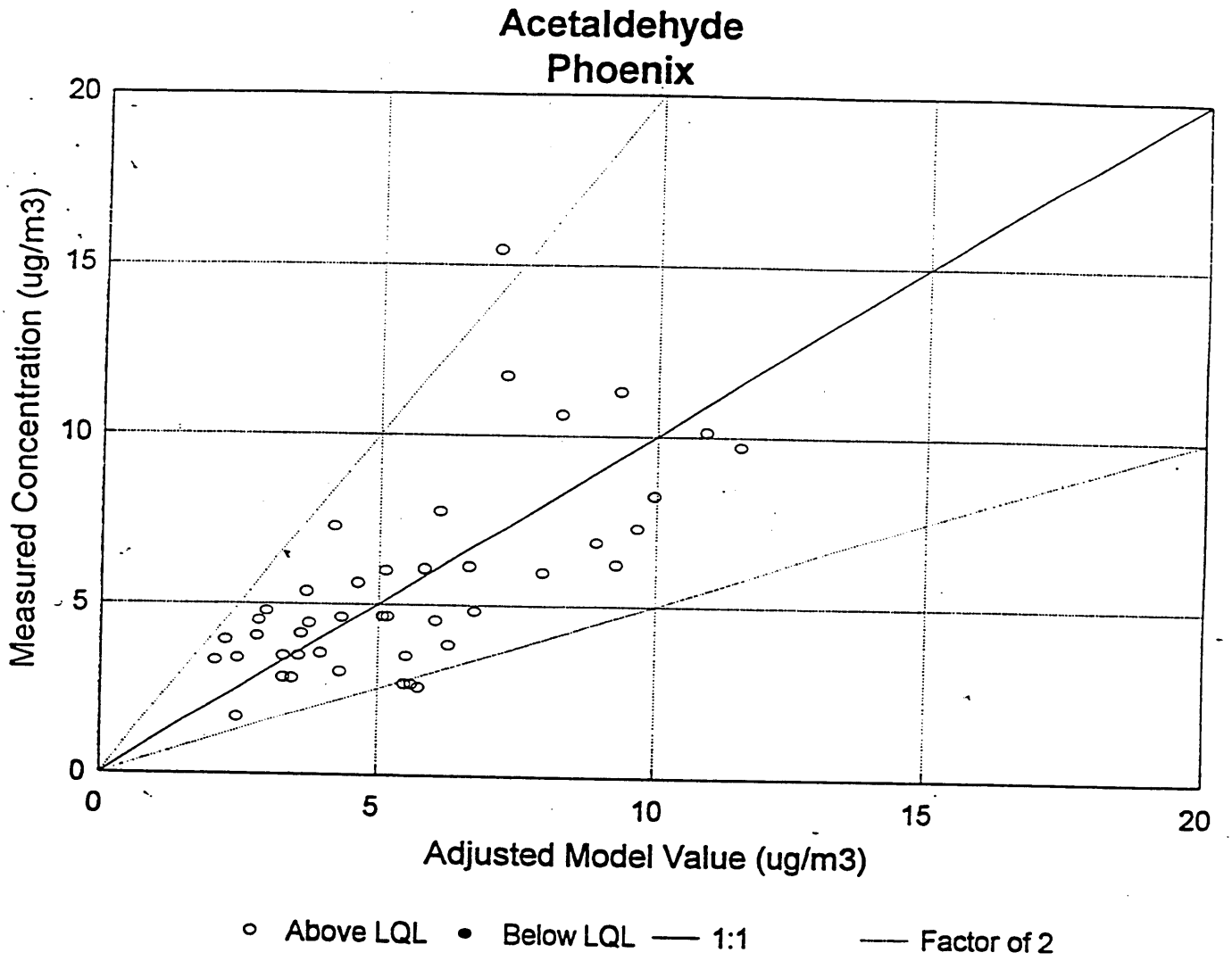


Figure 2-1. Comparison of 24-hour Measured Acetaldehyde Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

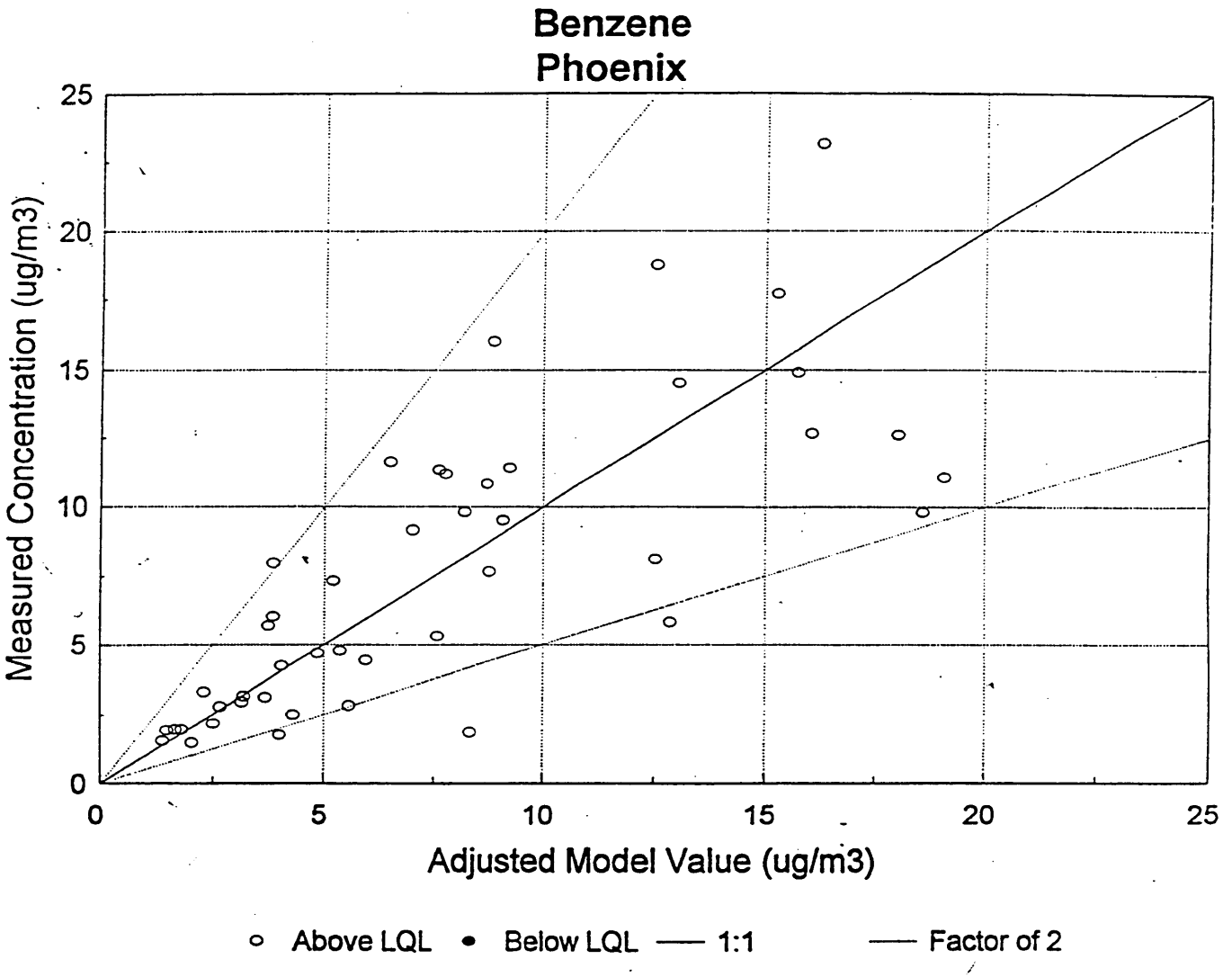


Figure 2-2. Comparison of 24-hour Measured Benzene Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

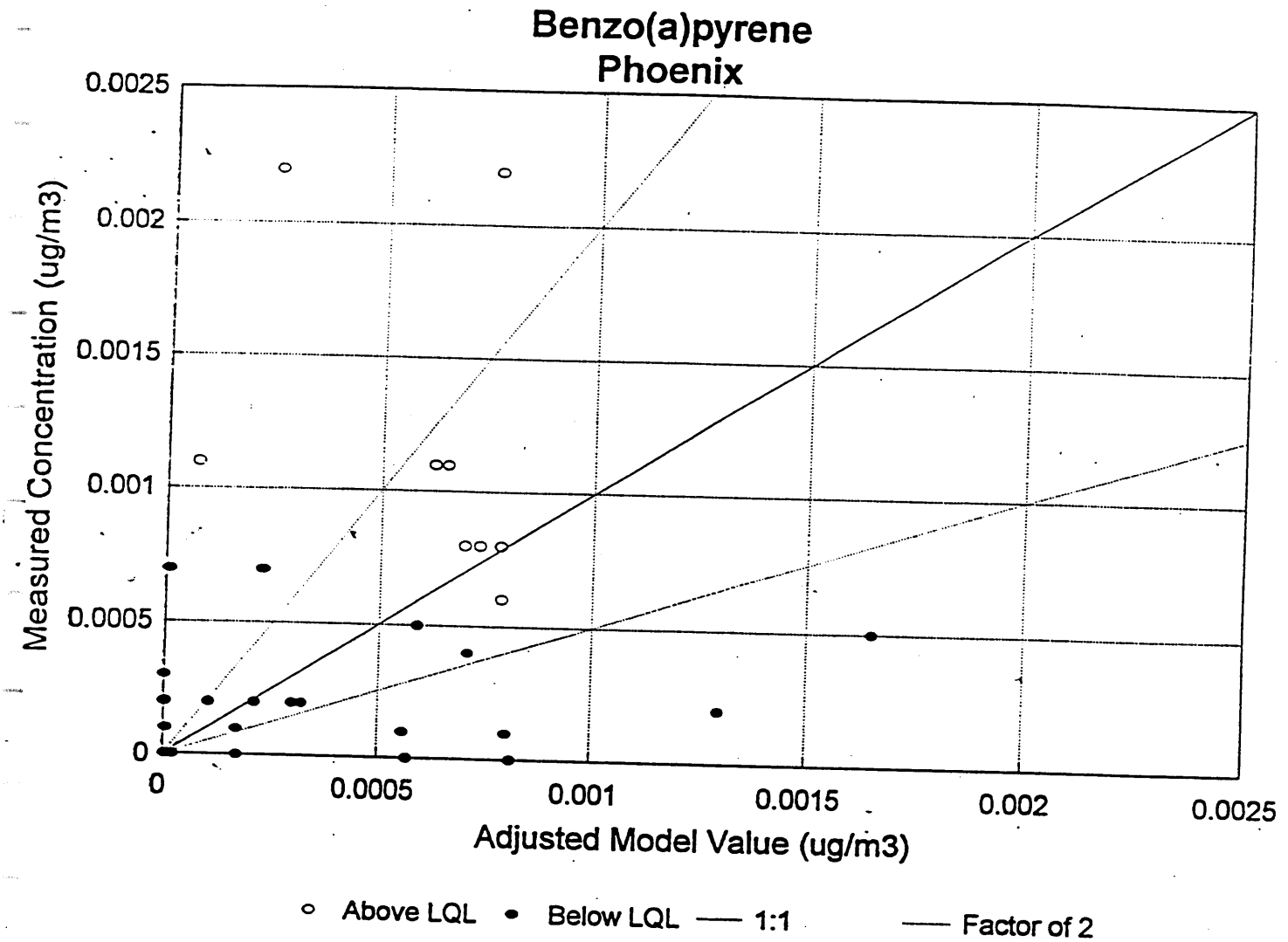


Figure 2-3. Comparison of 24-hour Measured Benzo(a)pyrene Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

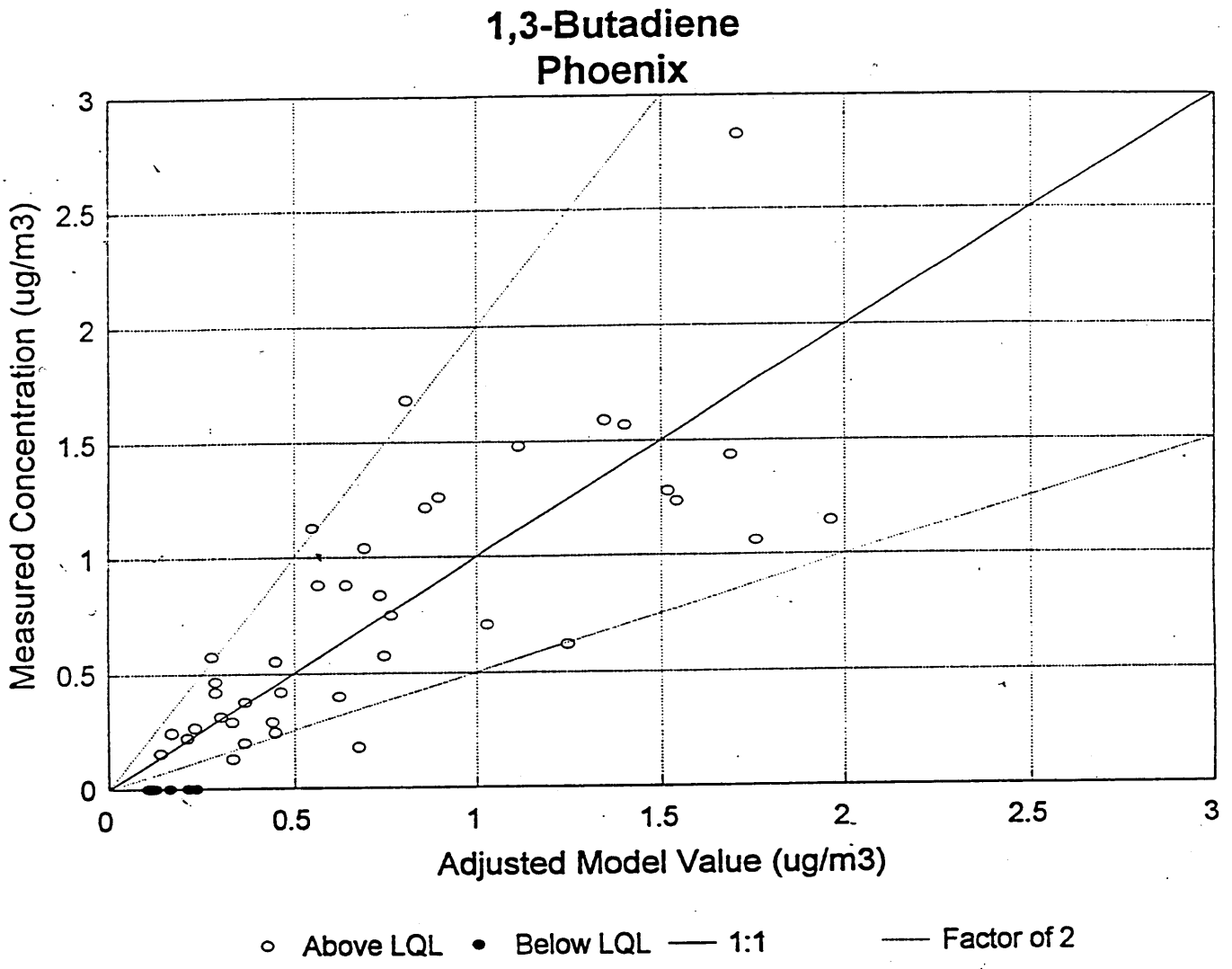


Figure 2-4. Comparison of 24-hour Measured 1,3-Butadiene Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

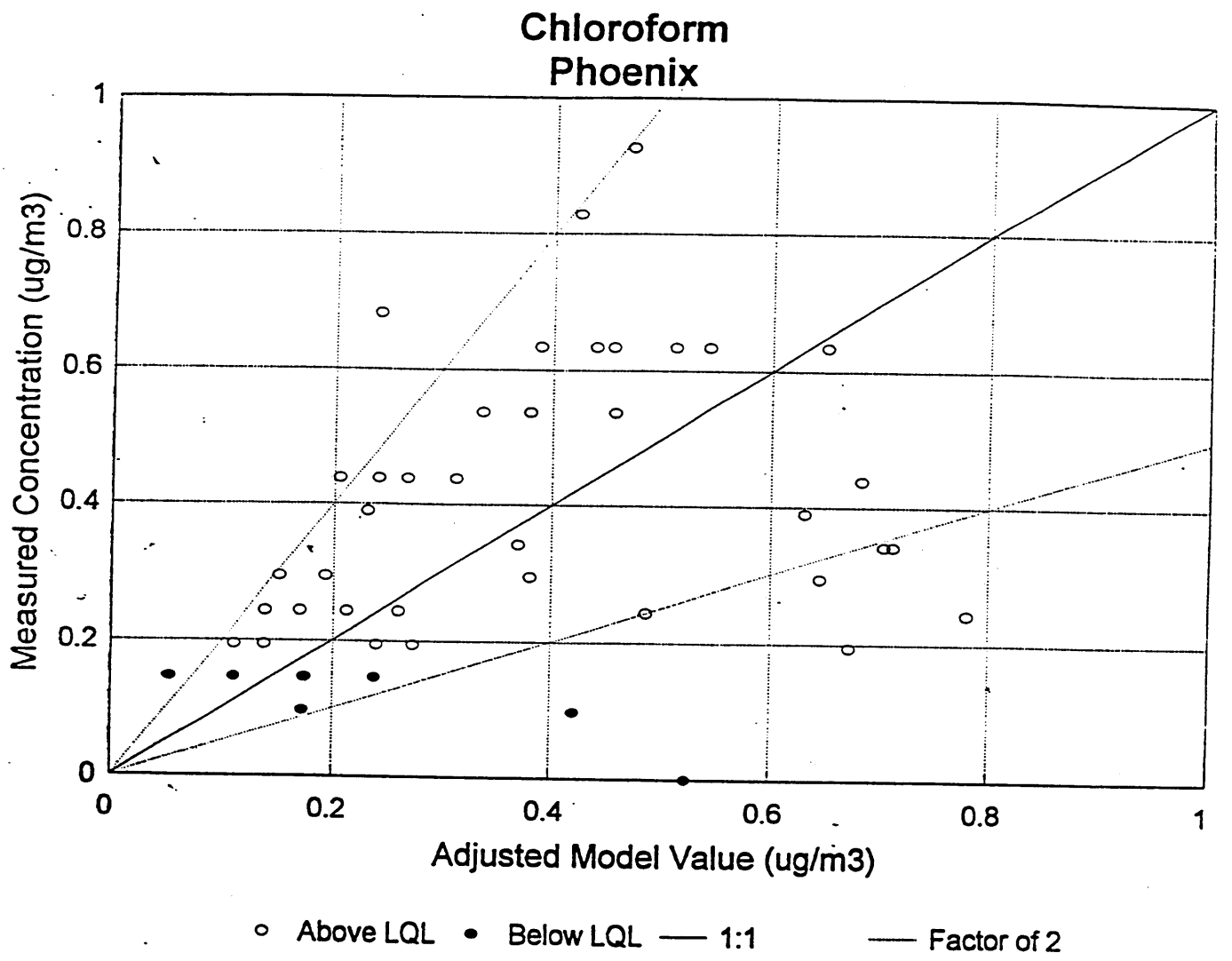


Figure 2-5. Comparison of 24-hour Measured Chloroform Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

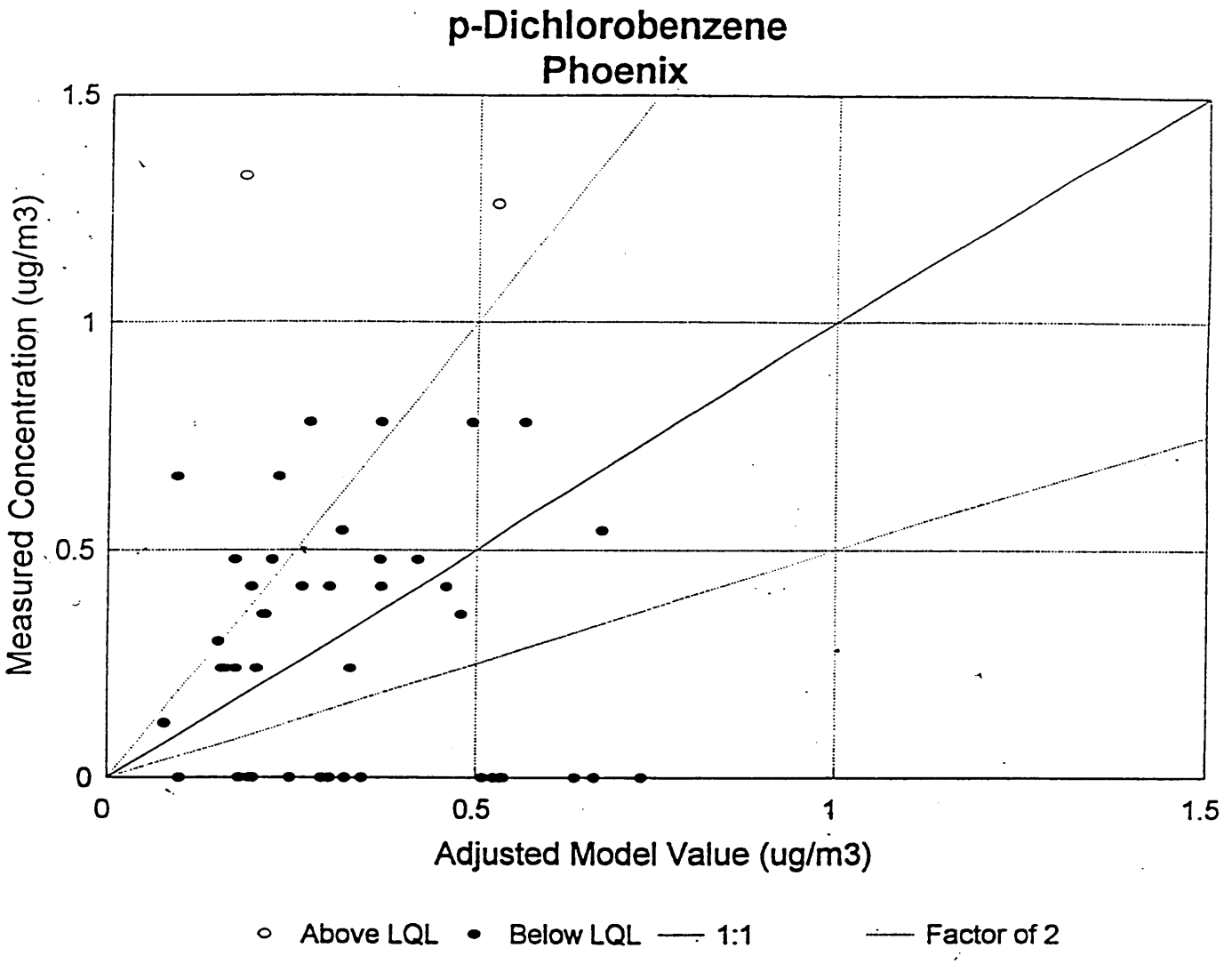


Figure 2-6. Comparison of 24-hour Measured p-Dichlorobenzene Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

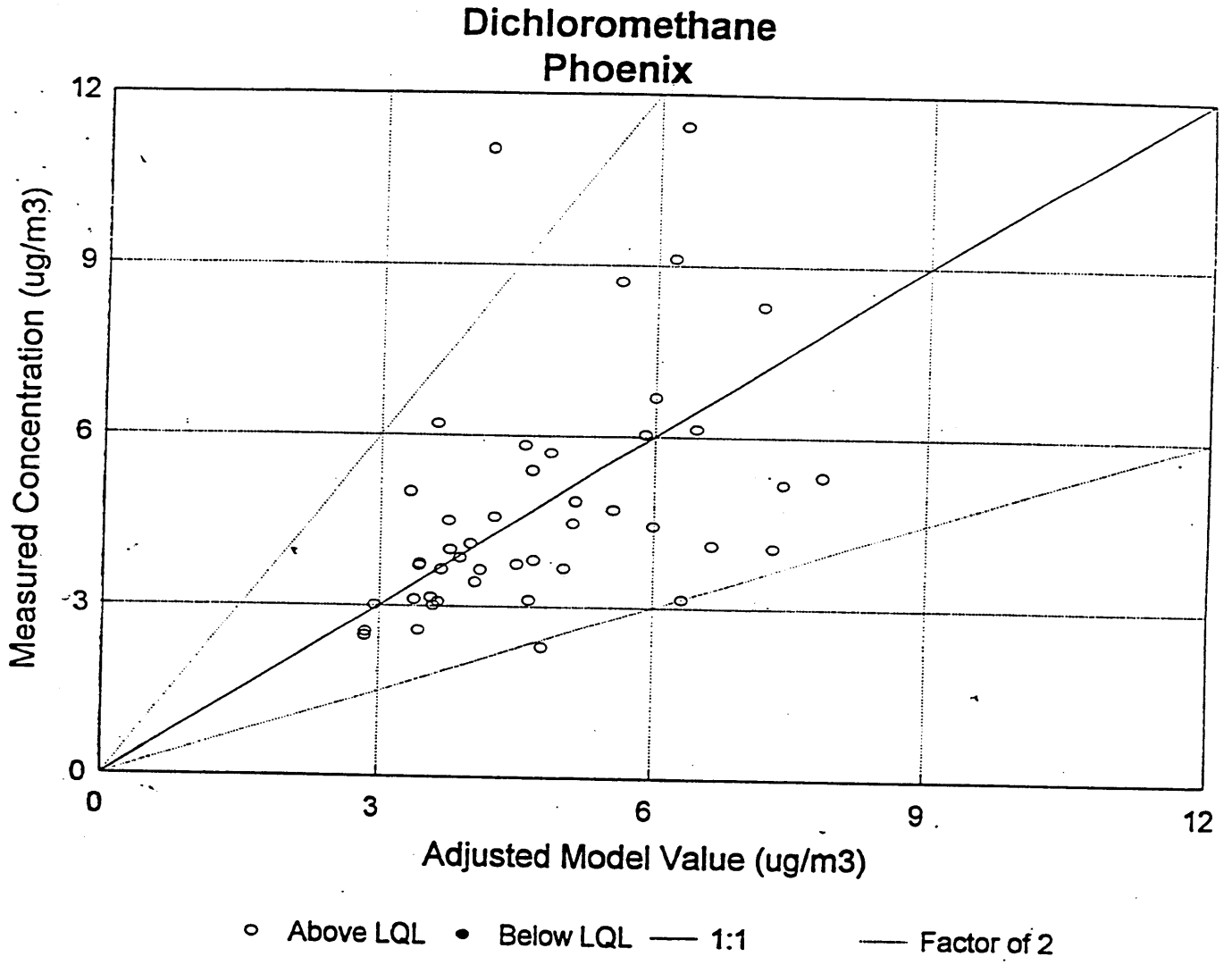


Figure 2-7. Comparison of 24-hour Measured Dichloromethane Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

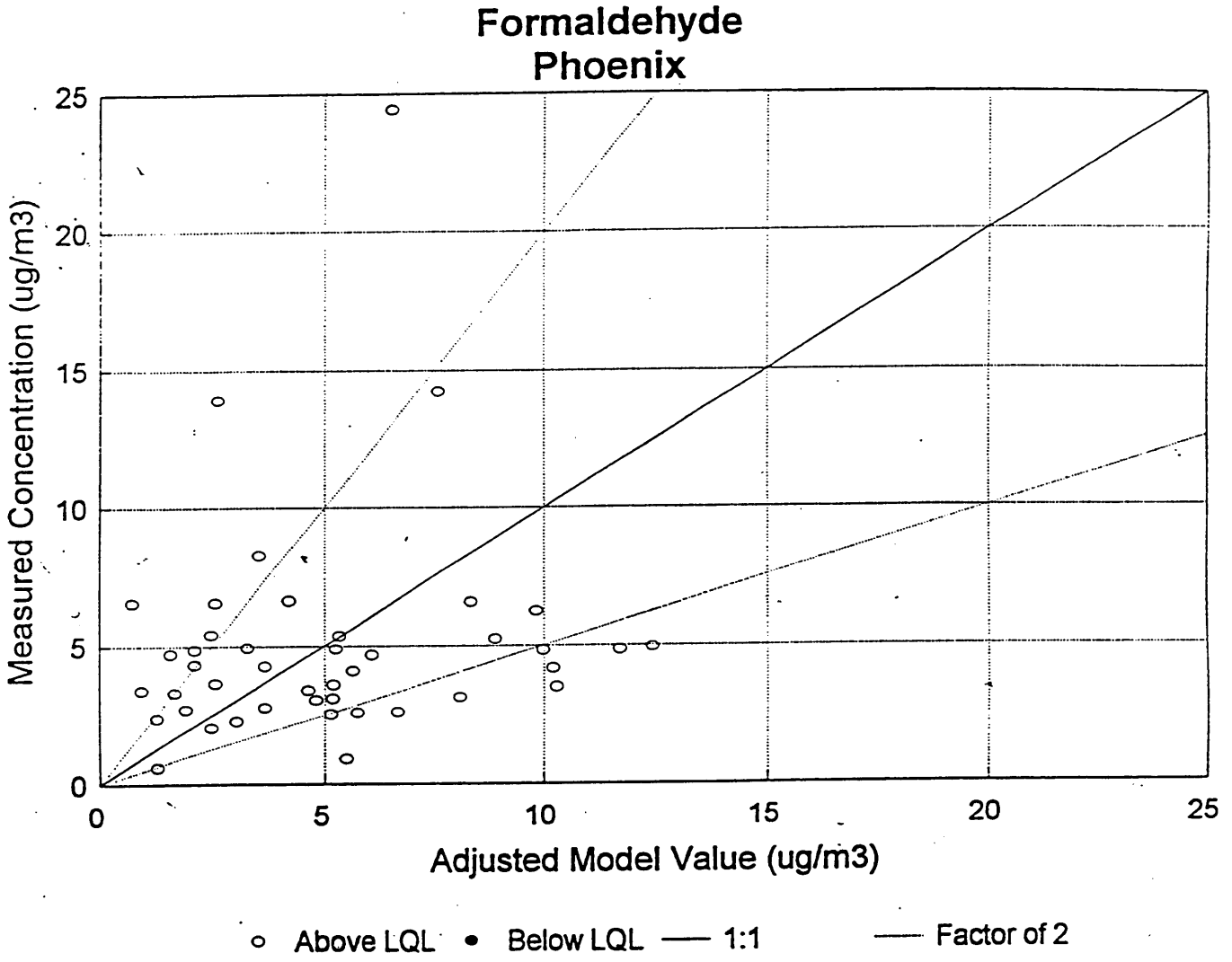


Figure 2-8. Comparison of 24-hour Measured Formaldehyde Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

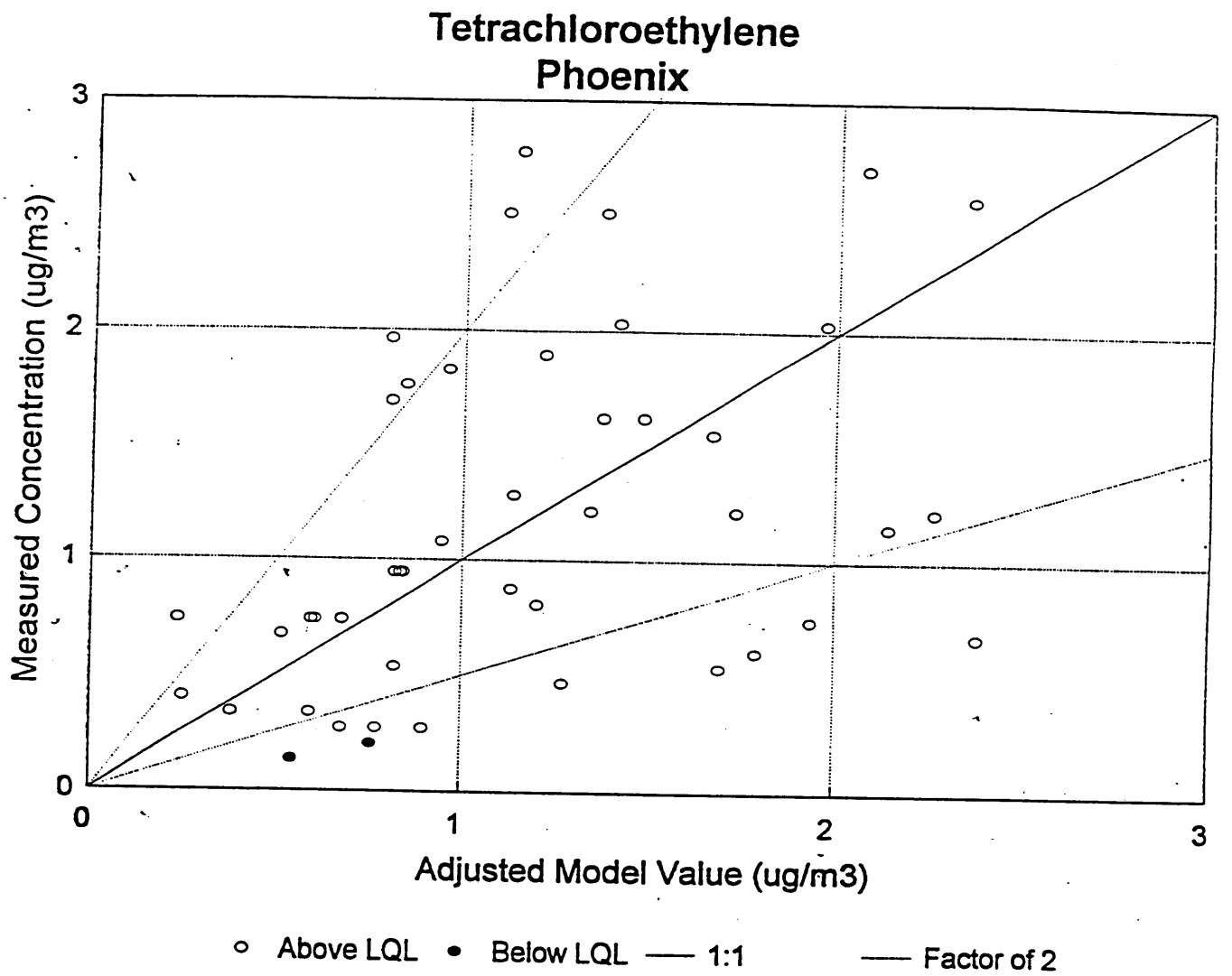


Figure 2-9. Comparison of 24-hour Measured Tetrachloroethylene Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

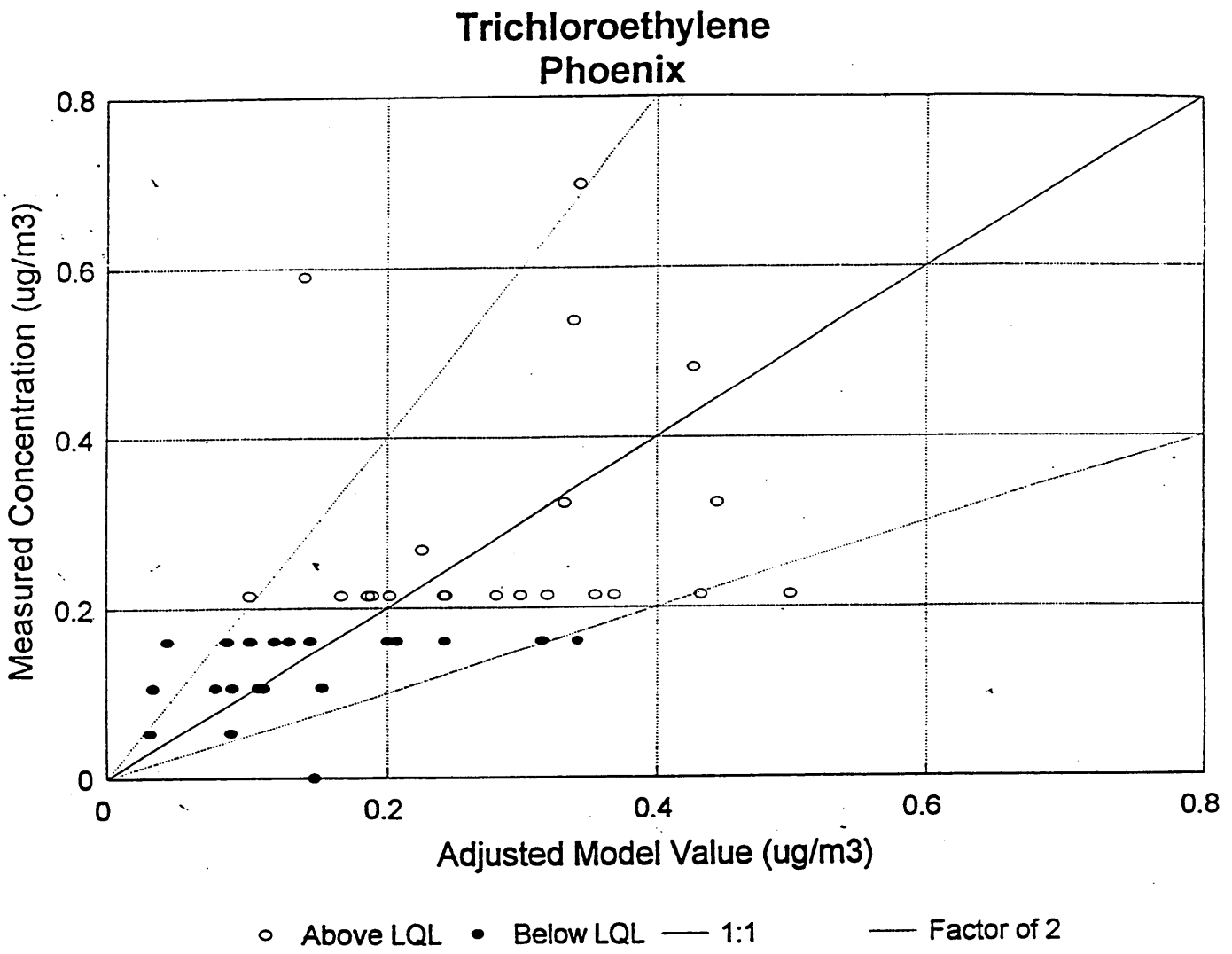


Figure 2-10. Comparison of 24-hour Measured Trichloroethylene Concentrations with Adjusted Simulation Model Results at the Phoenix Super Site.

emission source location. Most tetrachloroethylene emissions are estimated to come from dry cleaners. These small facilities are treated in the emissions data and in the simulation model as being distributed among modeling grid cells in proportion to population and as being distributed uniformly within individual grid cells. Therefore, effects on concentrations of individual dry cleaners are not accounted for in the model. The measured concentrations of the other species (benzo(a)pyrene, p-dichlorobenzene and trichloroethylene) were frequently below lower quantifiable limits, which reduces the significance of the comparisons.

The ability of the adjusted model results to simulate general seasonal variations in concentrations is shown in Figures 2-11 through 2-20, which show 24-hour average adjusted model results for April through December 1994, along with the measured concentrations. The model predicts generally higher concentrations of most species during the fall and winter period, which is caused by the decreased dispersion during that part of the year. This pattern is reflected in many of the measurements, as well. The model results also show the effects of seasonal emission differences for benzo(a)pyrene (Figure 2-13), which is emitted primarily by wood burning for heating during the winter. Both the model results and the measurements are near zero during the summer and substantially higher during the winter.

On the other hand, the highest chloroform concentrations occurred during the summer (Figure 2-15). This behavior is consistent with the major emission source being swimming pool chlorination, which would be greatest during the summer. However, the model results do not show this behavior, because the seasonal pattern in the emission estimates does not include this variation.

2.2 Adjustment Factors for Tucson

Measurements did not begin in Tucson until April 1995, so it was not possible to compare measured concentrations directly with model results. Instead, carbon monoxide (CO) concentrations measured by the Pima County Department of Environmental Quality (PDEQ) were used as an indicator of general atmospheric dispersion of emissions to scale 1995 data to 1994. These scaled concentrations were then used to calculate model adjustment factors.

Hourly-average carbon monoxide data were available for all of 1994 and for the first six months of 1995. CO is measured at several locations in the Tucson area, but not at the location that was used for the HAP measurements. Instead, data from the PDEQ 22nd and Craycroft site, which is east of the HAP monitoring site near a residential neighborhood removed from heavily traveled roads, were used.

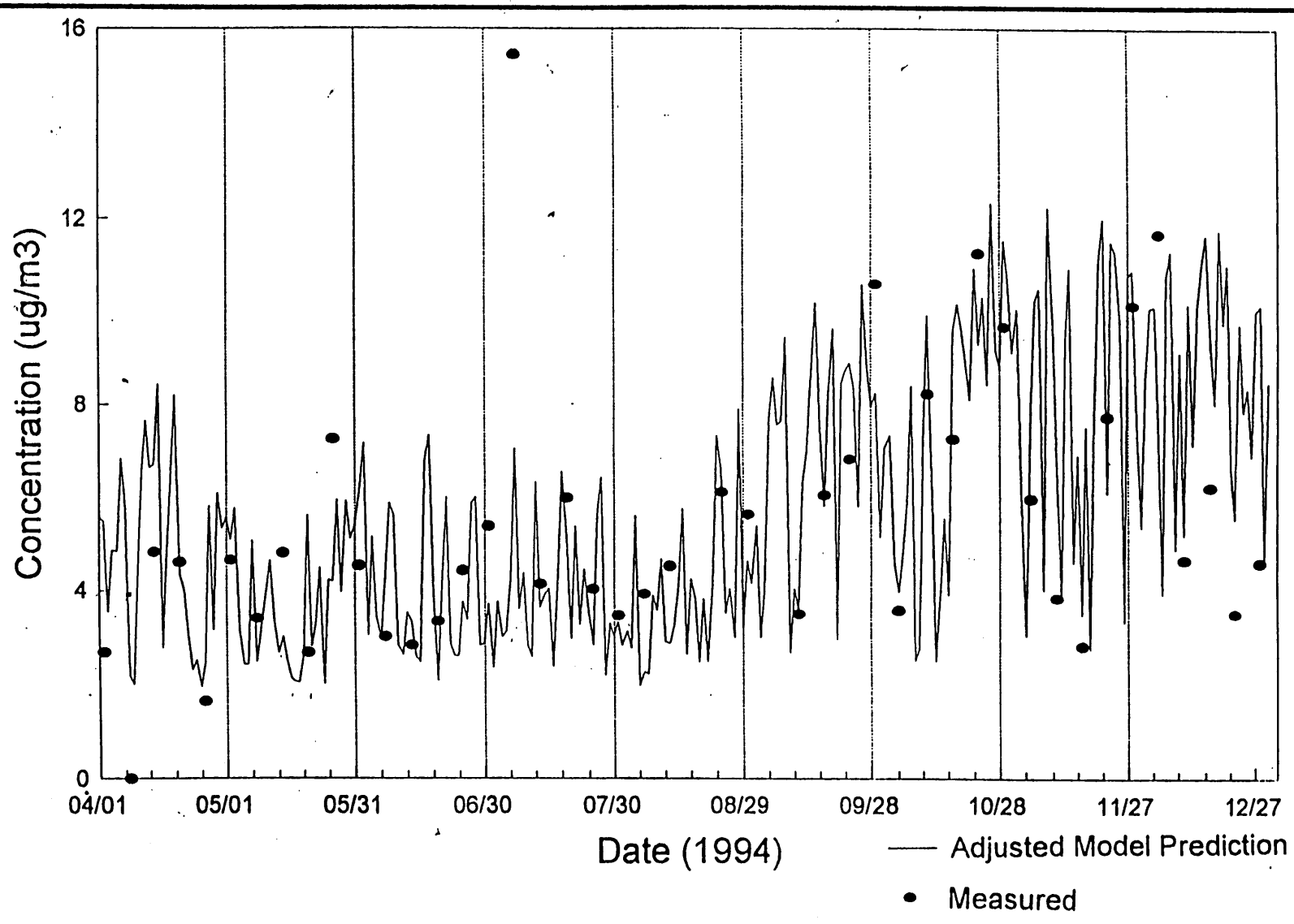
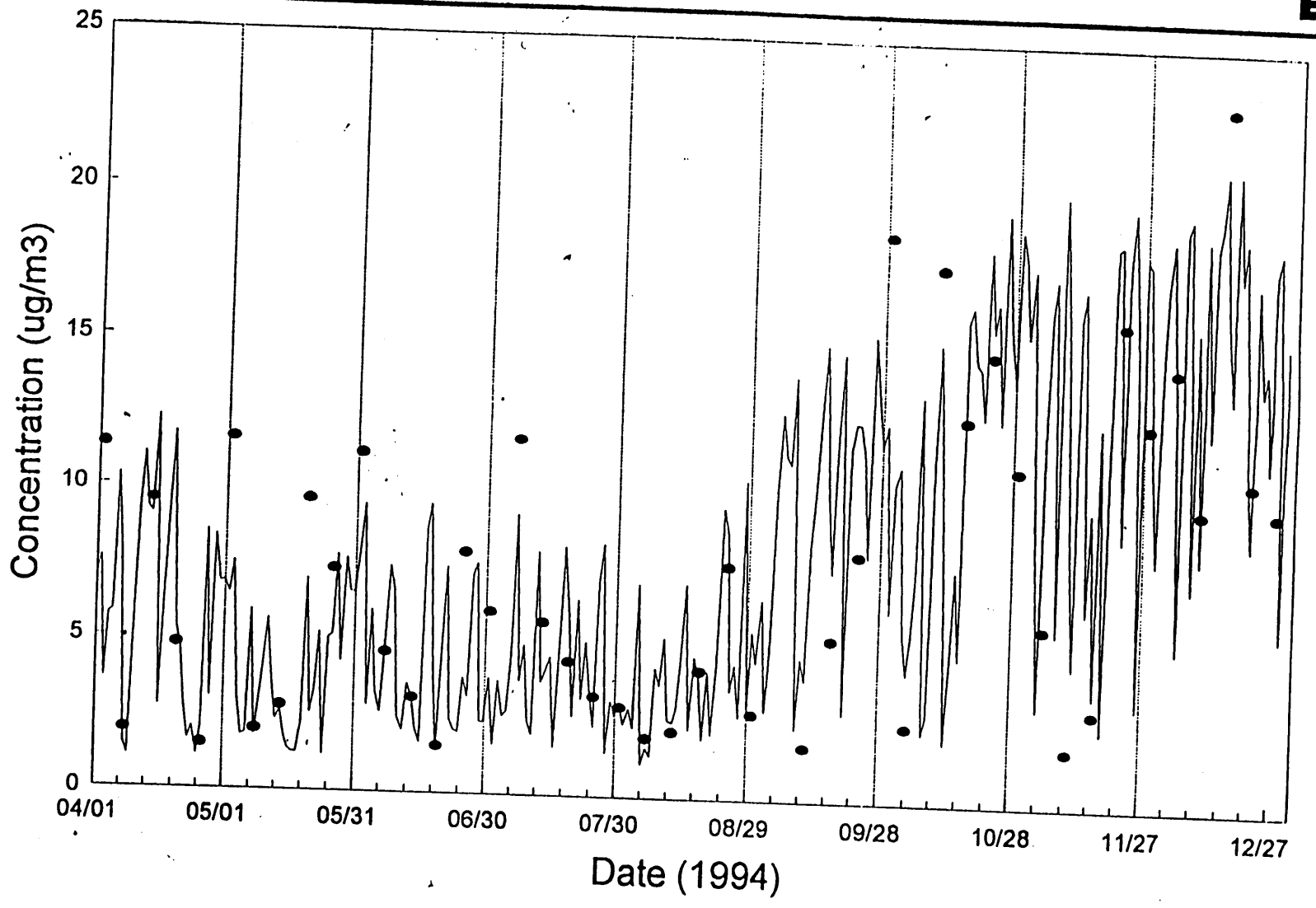


Figure 2-11. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Acetaldehyde at the Phoenix Super Site.



— Adjusted Model Prediction • Measured

Figure 2-12. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Benzene at the Phoenix Super Site.

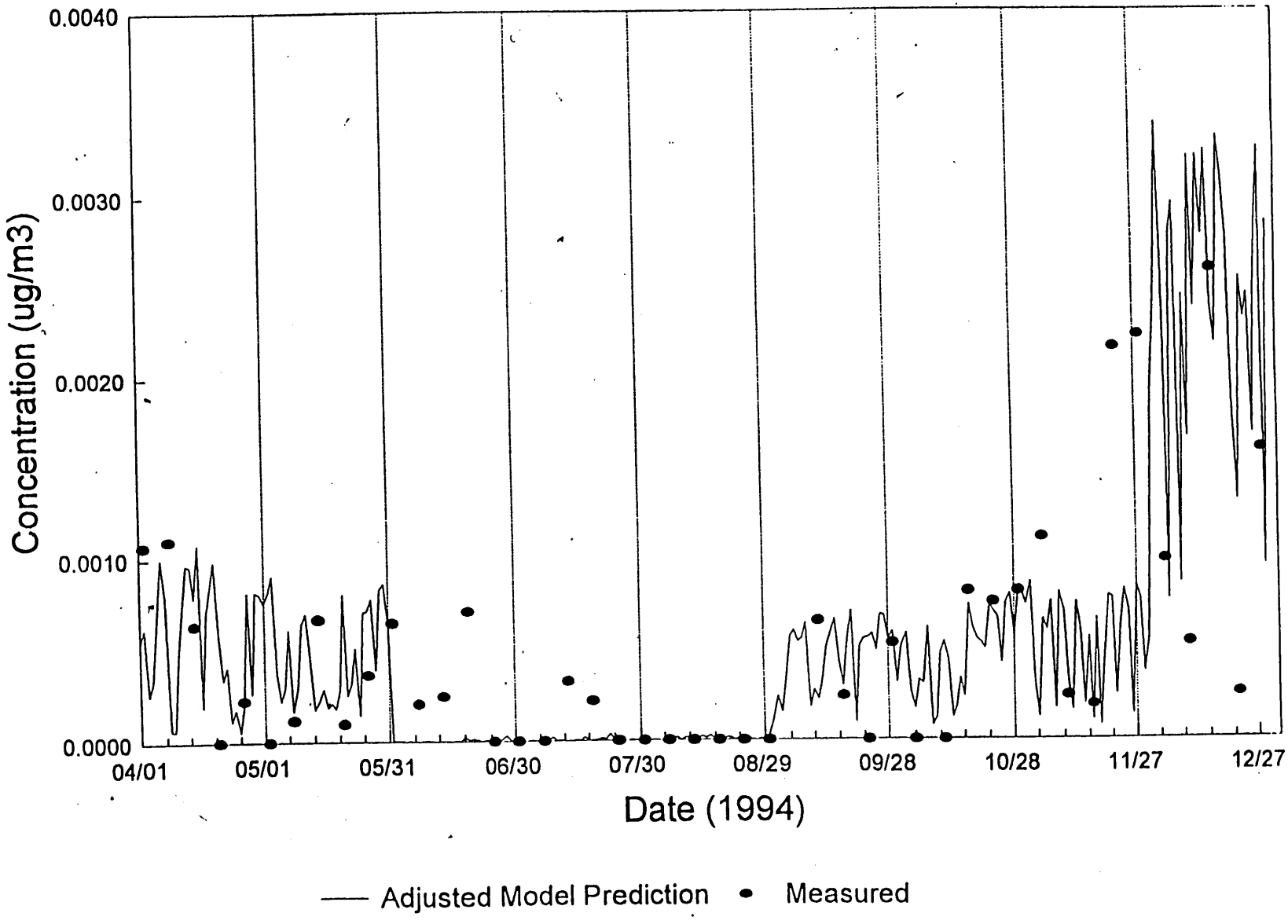


Figure 2-13. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Benzo(a)pyrene at the Phoenix Super Site.

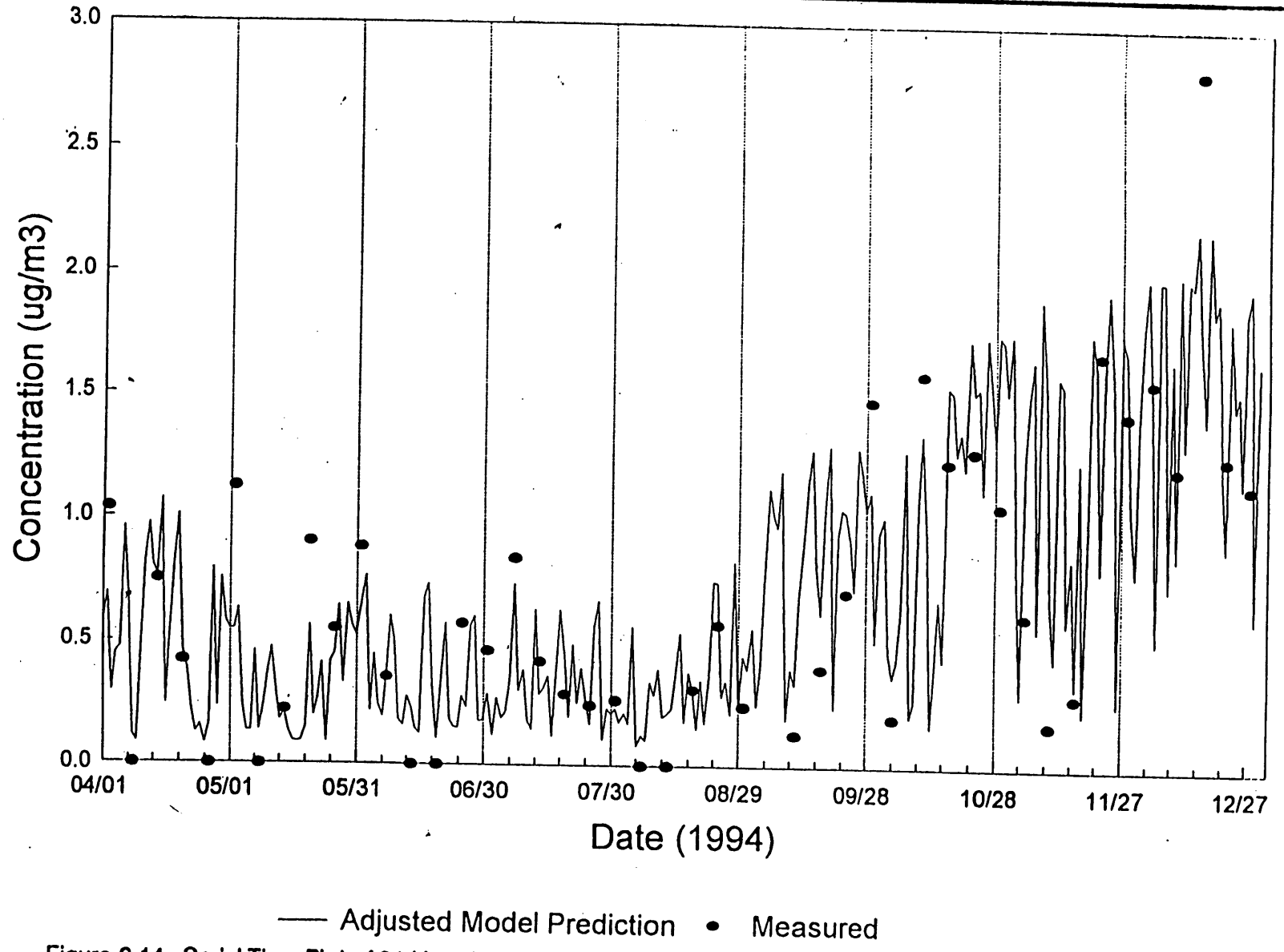


Figure 2-14. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for 1,3-Butadiene at the Phoenix Super Site.

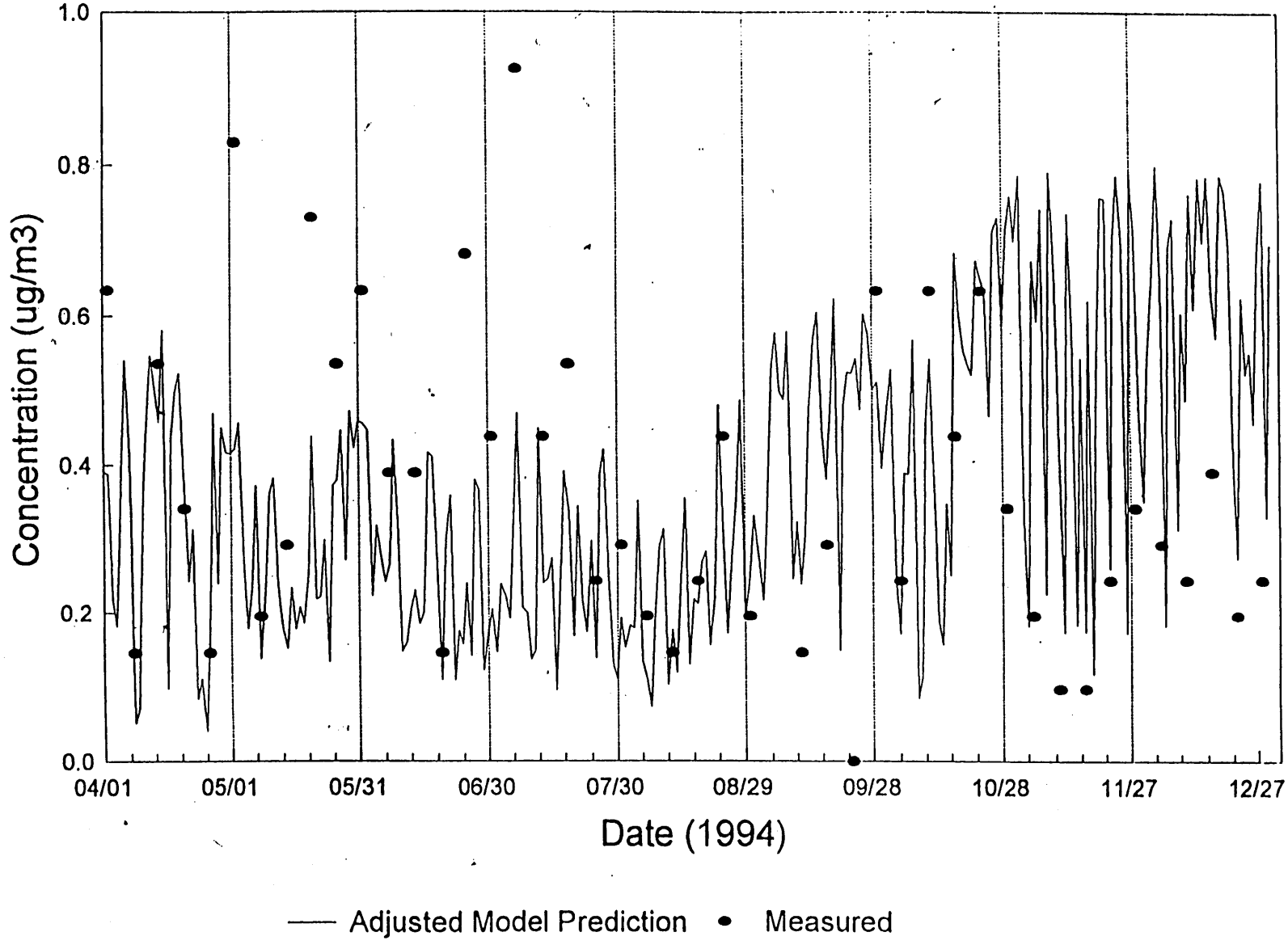


Figure 2-15. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Chloroform at the Phoenix Super Site.

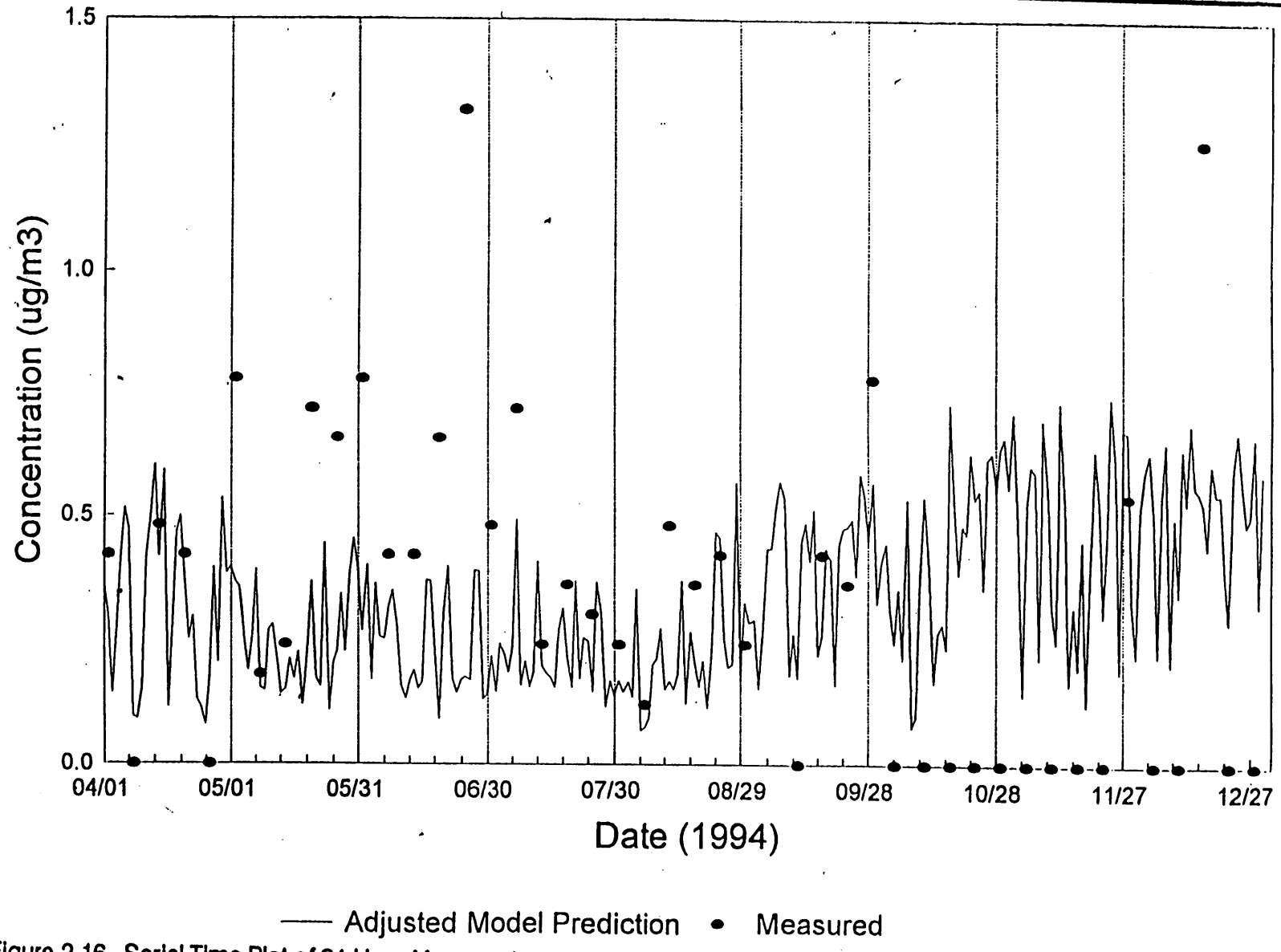
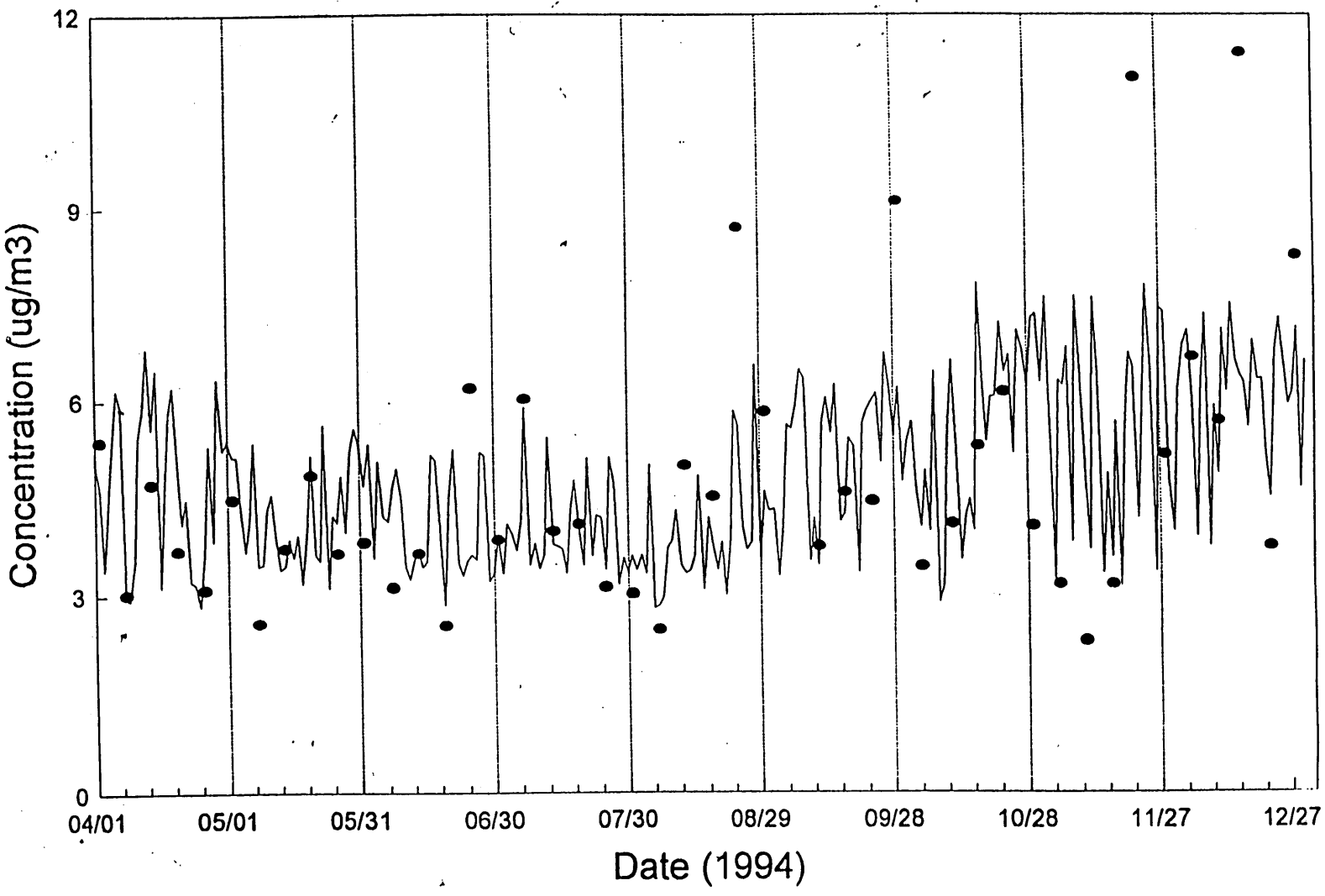
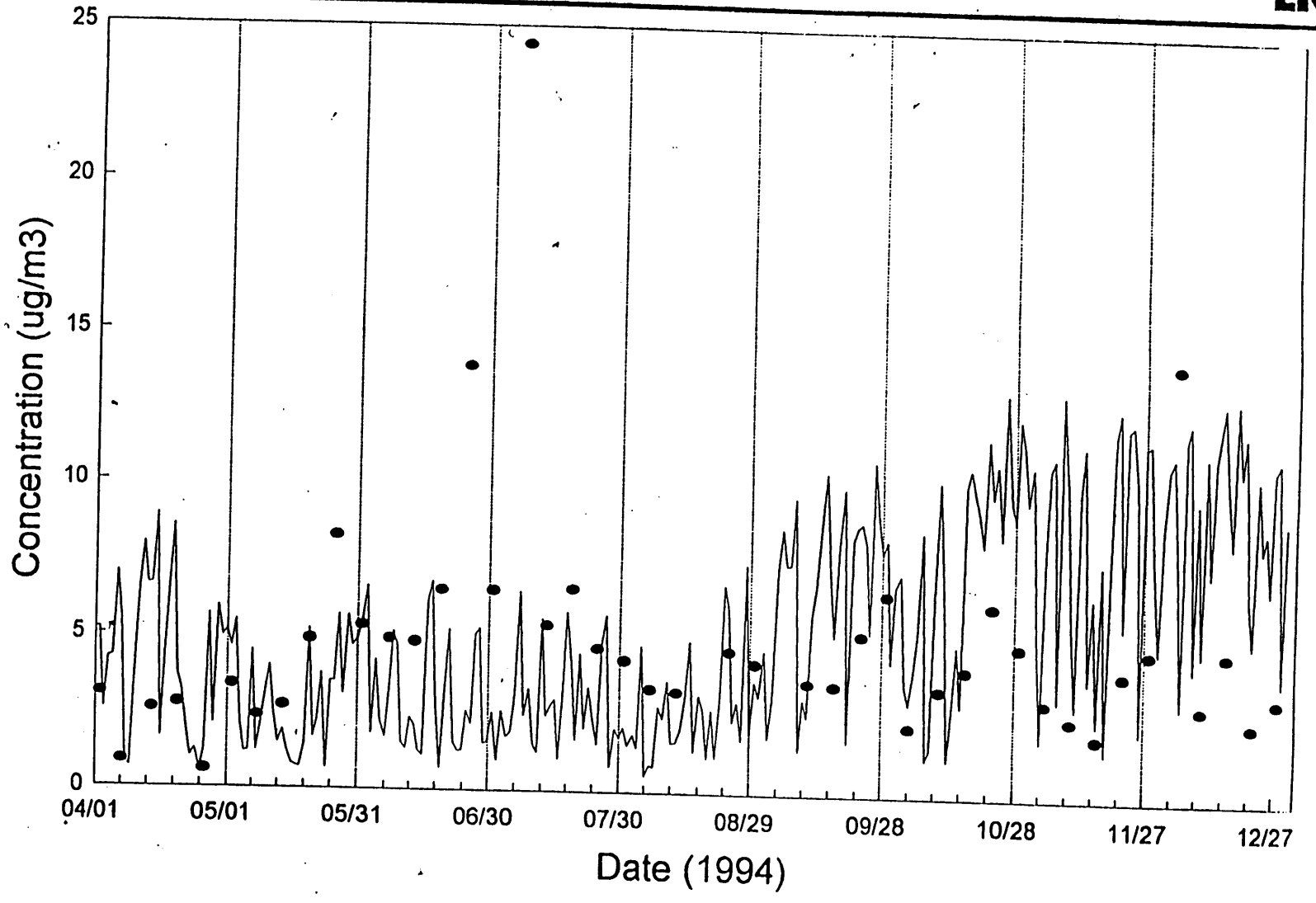


Figure 2-16. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for p-Dichlorobenzene at the Phoenix Super Site.



—— Adjusted Model Prediction • Measured

Figure 2-17. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Dichloromethane at the Phoenix Super Site.



— Adjusted Model Prediction • Measured

Figure 2-18. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Formaldehyde at the Phoenix Super Site.

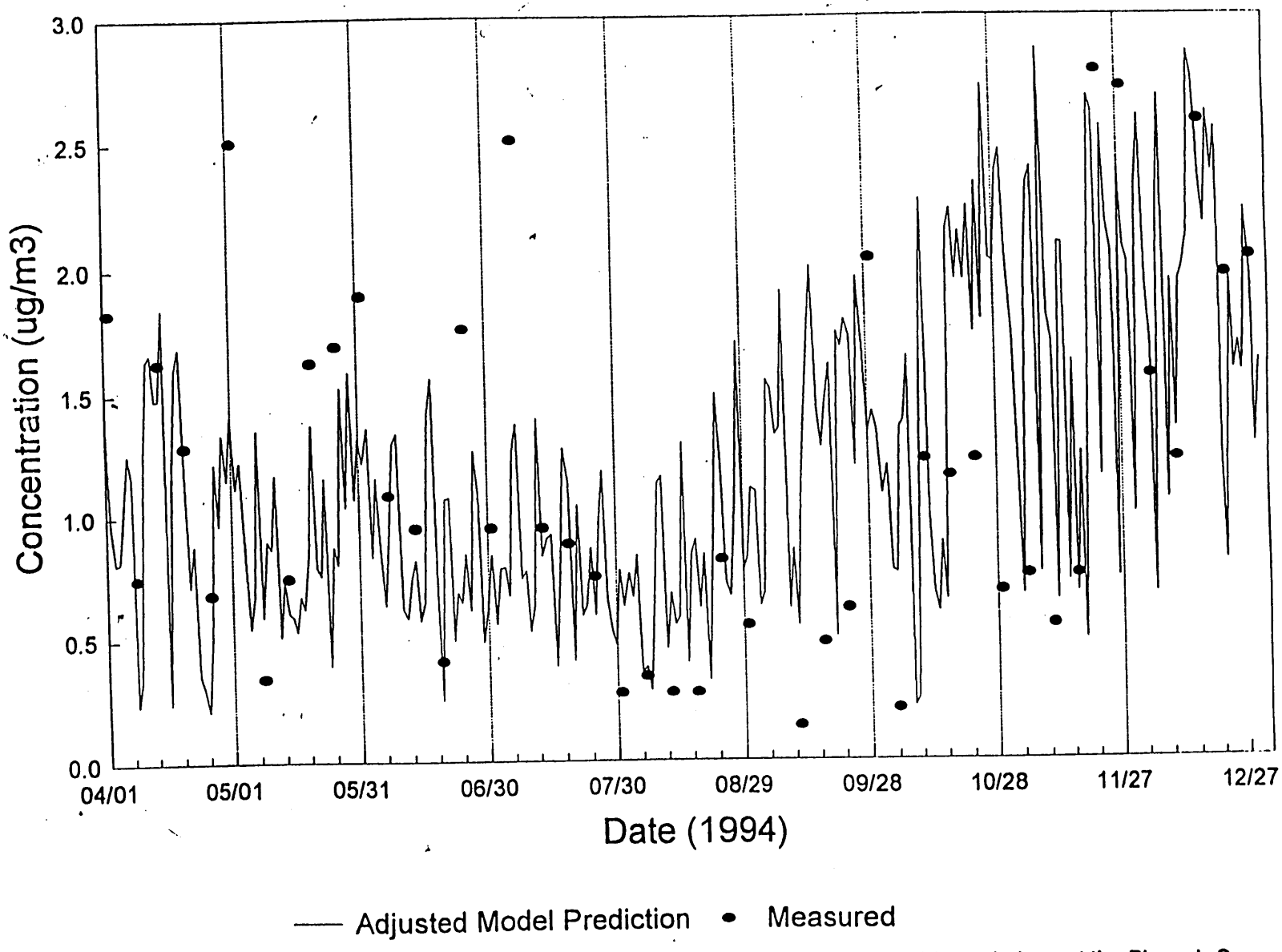
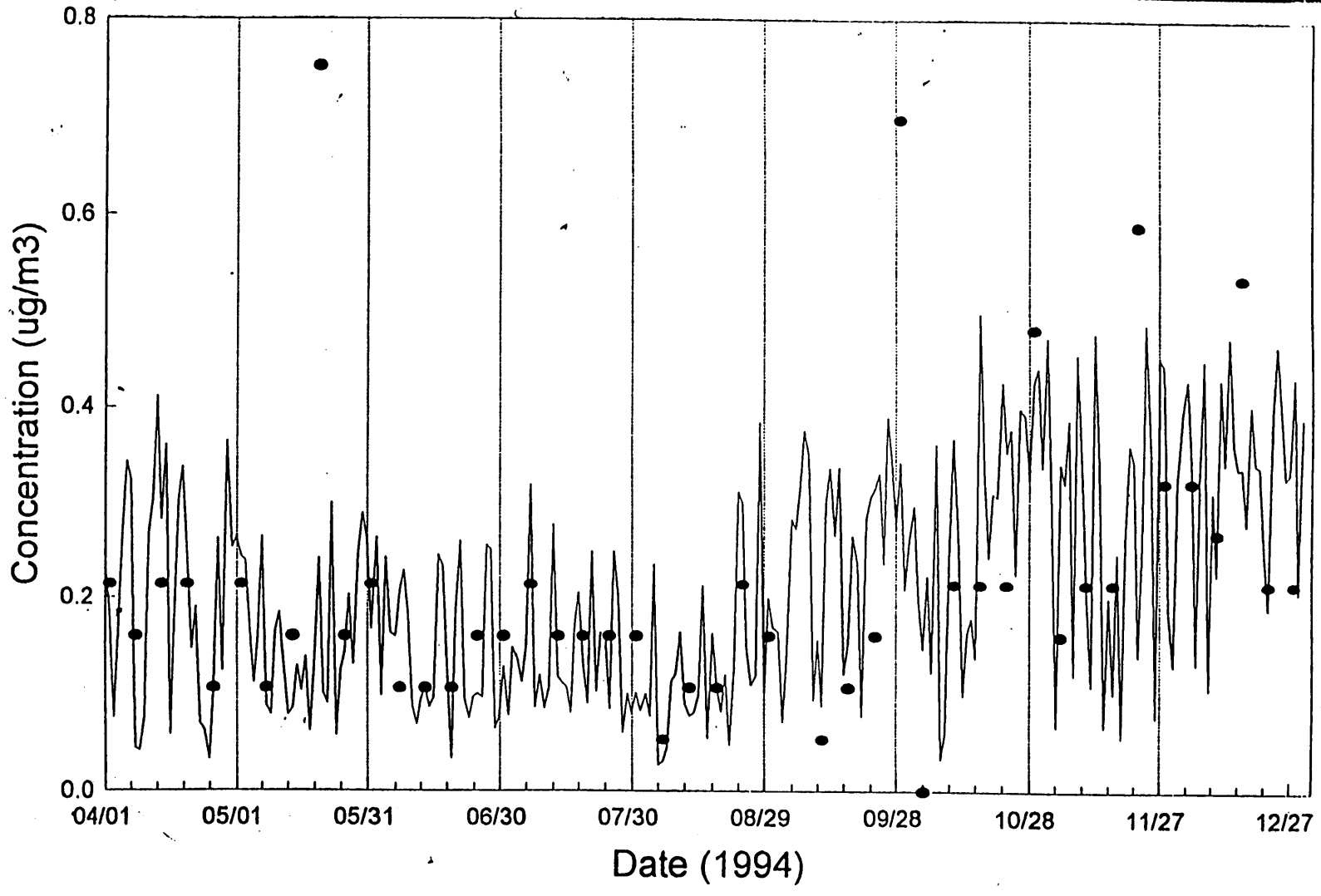


Figure 2-19. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Tetrachloroethylene at the Phoenix Super Site.



—— Adjusted Model Prediction • Measured

Figure 2-20. Serial Time Plot of 24-Hour Measured and Adjusted Model Results for Trichloroethylene at the Phoenix Super Site.

Averages of the HAP concentrations measured from April through June 1995 were extrapolated to the same time period during 1994 and to the entire year of 1994. The extrapolations were made by multiplying the average measured HAP concentrations by the ratios of the average CO concentration measured during the appropriate period during 1994 (i.e., average for the entire year and average for April-June 1994) to the average CO concentration measured when the HAP samples were collected during 1995:

$$\overline{C_{e,i}} = \overline{C_{m,i}} \frac{\overline{[CO]_e}}{\overline{[CO]_m}}$$

where

$\overline{C_{e,i}}$ = extrapolated average concentration of HAP i

$\overline{C_{m,i}}$ = measured average concentration of HAP i

$\overline{[CO]_e}$ = measured average CO concentration during extrapolation timeframe

$\overline{[CO]_m}$ = measured average CO concentration during HAP measurements

The resulting scaling factors were 1.5 and 1.15 for the annual and three-month extrapolations.

Table 2-2 lists the average HAP concentrations measured during the second quarter of 1995 (April-June) and during April through August 1995 along with the extrapolated average concentrations for all of 1994 and for the second quarter of 1994, and Table 2-3 lists the extrapolated concentrations, adjusted for background, the average simulation model results for the same time periods, and the resulting model adjustment factors calculated from the corresponding values (labeled "model scale factors" in the table).

A third set of model scale factors was also calculated, based on the average concentrations measured from April through August 1995 and the model results for April through August 1994. Although this last set of factors does not account for year-to-year variations (i.e., no extrapolation from 1995 to 1994), it includes more of the measurements made during the summer to reflect the possible influence of increased photochemical activity.

TABLE 2-2

Average Concentrations Measured in Tucson during 1995 and Extrapolated to 1994

Species	Measured Average 4/95-6/95 ($\mu g/m^3$)	Measured Average 4/95-8/95 ($\mu g/m^3$)	Background Concentration ($\mu g/m^3$)	Extrapolated Average 4/94-6/94 ($\mu g/m^3$)	Extrapolated Average 1/94-12/94 ($\mu g/m^3$)
1,3-Butadiene	0.19	0.21	0	0.22	0.29
Acetaldehyde	3.1	2.4	1.5	3.6	4.7
Acrolein	0.0093	0.0140	0	0.011	0.014
Benzene	2.8	3.0	0.3	3.3	4.2
Benzo(a)pyrene	0.00013	0.00012	0	0.00015	0.00020
Chlorobenzene	0.018	0.026	0	0.021	0.027
Chloroform	0.18	0.21	0	0.22	0.28
Dichloromethane	4.7	3.9	2.5	5.6	7.1
Formaldehyde	0.86	0.84	0	1.0	1.3
n-Hexane	3.8	3.0	0.5	4.5	5.7
p-Dichlorobenzene	0.051	0.029	0	0.059	0.077
Tetrachloroethylene	0.55	0.45	0	0.64	0.82
Toluene	6.6	6.1	0.3	7.7	9.9
Trichloroethylene	0.17	0.18	0	0.20	0.26

TABLE 2-3

Tucson Simulation Model Adjustment Factors

Species	1994 Annual Avgerages ($\mu g/m^3$)		April-June, 1994 Averages ($\mu g/m^3$)		April-August Averages ($\mu g/m^3$)		Model Scale Factors			Model Adjust. Factor
	Extrap. Measured, Non-Back-ground	Model	Extrap. Measured, Non-Back-ground	Model	Measured, Non-Back-ground, 1995	Model, 1994	From Extrap. to Annual 1994	From Extrap. to 4/94-6/94	From 4/95-8/95	
1,3-Butadiene	0.29	0.45	0.19	0.28	0.21	0.29	0.64	0.69	0.75	1
Acetaldehyde	3.2	0.72	1.6	0.49	0.89	0.52	4.4	3.2	1.7	3
Acrolein	0.014	0.084	0.0093	0.058	0.014	0.062	0.17	0.16	0.22	0.2
Benzene	3.9	4.0	2.5	2.6	2.7	2.7	0.99	0.96	0.98	1
Benzo(a)pyrene	0.00020	0.00051	0.00013	0.00029	0.00012	0.00017	0.39	0.45	0.70	1
Chlorobenzene	0.027	0.47	0.018	0.39	0.026	0.38	0.056	0.046	0.068	0.05
Chloroform	0.28	0.24	0.18	0.20	0.21	0.19	1.1	0.92	1.1	1
Dichloromethane	4.6	0.97	2.2	0.79	1.4	0.77	4.8	2.9	1.8	4
Formaldehyde	1.3	2.1	0.86	1.5	0.84	1.5	0.61	0.58	0.55	0.5
n-Hexane	5.2	4.9	3.3	3.8	2.5	4.0	1.1	0.87	0.63	1
p-Dichlorobenzene	0.077	0.040	0.051	0.034	0.03	0.034	1.9	1.5	0.85	1
Tetrachloroethylene	0.8	1.5	0.55	1.2	0.45	1.1	0.55	0.46	0.40	0.5
Toluene	9.6	15	6.3	11	5.9	11	0.64	0.59	0.52	0.5
Trichloroethylene	0.26	0.49	0.17	0.40	0.18	0.39	0.54	0.44	0.47	0.5

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Thus, the table shows three model scale factors for each HAP: (1) a factor based on an extrapolation of second quarter 1995 measurements to all of 1994; (2) a factor based on an extrapolation of second quarter 1995 measurements to the second quarter of 1994; and (3) a factor based on a direct comparison between April-August 1995 measurements and April-August 1994 model results. These three factors were examined for each species, and the "typical" adjustment factor shown in the far right column of the table was selected for use as the model adjustment factors for the Tucson region.

2.3 Adjustment Factors for Casa Grande

As in Tucson, measurements did not begin in Casa Grande until April 1994, so adjustment factors could not be developed from direct comparisons between measurements and model results. Unlike Tucson, carbon monoxide is not measured in Casa Grande, so 1995 measurements could not be extrapolated to 1994. Therefore, adjustment factors were calculated from the April through August 1995 measurements combined with the April through August 1994 model results.

Table 2-4 lists the average of the April through August 1995 measurements, the background concentrations, the average measured values with the background concentrations subtracted, the average unadjusted model results for April through August 1994, the ratio of the average model results to the background-adjusted average measurements, and the model adjustment factors that were chosen for application to the Casa Grande results. Because of the uncertainties introduced by comparing the 1994 model results with the 1995 measurements, the adjustment factors are only precise to one significant figure

TABLE 2-4

Casa Grande Region Model Adjustment Factors

Species	Average Measured Concen. 4/95-8/95 (µg/m³)	Back-ground Concen. (µg/m³)	Average Non-Back-ground Concen. 4/95-8/95 (µg/m³)	Model Average Concen. 4/94-8/94 (µg/m³)	Model Scale Factor	Model Adjustment Factor
1,3-Butadiene	0.14	0	0.14	0.067	2.1	2
Acetaldehyde	2.3	1.5	0.80	0.14	5.9	5
Acrolein	0.0080	0	0.0080	0.016	0.5	1
Arsenic	0.00024	0	0.00024	0.00042	0.6	0.5
Benzene	1.5	0.3	1.2	0.70	1.7	1.5
Benzo(a)pyrene	0.00025	0	0.00025	0.000035	7.0	1
Cadmium	0.00069	0	0.00069	0.0011	0.6	0.5
Chloroform	0.15	0	0.15	0.0060	25.6	25
Dichloromethane	3.0	2.5	0.57	0.18	3.2	3
Formaldehyde	0.62	0	0.62	0.35	1.8	2
Lead	0.0040	0	0.0040	0.030	0.1	0.1
Manganese	0.018	0	0.018	0.041	0.4	0.5
Nickel	0.0017	0	0.0017	0.0040	0.4	0.5
p-Dichlorobenzene	0.0	0	0.0	0.049	0.0	1
Toluene	5.9	0.3	5.6	5.2	1.1	1
Trichloroethylene	0.62	0	0.62	0.12	5.3	5

APPENDIX D

ESTIMATION OF ANNUAL AVERAGE HAP CONCENTRATIONS IN PAYSON

TABLE 1

Average Concentrations of HAPs in Payson

Species	Average Concentration ($\mu g/m^3$)		
Pyrene	0.004688	0.005079	0.004487
Metals			
Arsenic	0.000858	0.000700	0.000936
Barium	0.014790	0.007800	0.018285
Cadmium	0.002019	0.001000	0.002528
Lead	0.006304	0.006400	0.006256
Manganese	0.014007	0.014900	0.013561
Nickel	0.000673	0.000500	0.000759

TABLE 1

Average Concentrations of HAPs in Payson

Species	Average Concentration ($\mu g/m^3$)		
	Annual	08/24-12/22/94 Measured	01/09-08/19/95 Measured
Polycyclic Organic Matter			
1,2-Dimethylnaphthalene	0.014406	0.017616	0.012748
1,7+1,3+1,6-Dimethylnaphthalene	0.056483	0.058691	0.055343
1,8-Dimethylnaphthalene	0.000369	0.000393	0.000357
1-Methylnaphthalene	0.130542	0.135743	0.127856
2,3+1,4+1,5-Dimethylnaphthalene	0.016949	0.016616	0.017122
2,6+2,7-Dimethylnaphthalene	0.025817	0.026073	0.025685
2-Methylnaphthalene	0.280659	0.290585	0.275532
Acenaphthene	0.012467	0.015964	0.010661
Acenaphthylene	0.015053	0.017396	0.013843
Anthracene	0.003148	0.003221	0.003110
Benz(a)anthracene	0.001254	0.001489	0.001132
Benzo(a)pyrene	0.001071	0.001289	0.000958
Benzo(b)chrysene	0.000134	0.000163	0.000119
Benzo(b+j+k)fluoranthene	0.001605	0.001915	0.001445
Benzo(ghi)perylene	0.001002	0.001132	0.000935
Biphenyl	0.015499	0.015417	0.015541
Chrysene	0.001383	0.001604	0.001269
Dibenzo(ah+ac)anthracene	0.000125	0.000152	0.000111
Fluoranthene	0.005812	0.006016	0.005707
Fluorene	0.013059	0.013434	0.012865
Indeno[123-cd]pyrene	0.000583	0.000694	0.000525
Naphthalene	0.475805	0.500292	0.463155
Phenanthrene	0.020303	0.020233	0.020340

APPENDIX D

ESTIMATION OF ANNUAL AVERAGE HAP CONCENTRATIONS IN PAYSON

The atmospheric simulation model was not used to estimate annual average concentrations in the Payson region, as it was in the other three study regions, because the complex terrain and resulting meteorology in Payson make simulations very inaccurate. Instead, annual average concentrations were estimated from the HAP measurements at the neighborhood sampling site. The measurements began at the Weber Well site in January 1994, so eight months of data were available. Although these data represent three-quarters of the year, they did not include fall or all of the winter, when wood burning emissions are expected to be greatest. Therefore, it was necessary to extrapolate the available data to include the rest of the wood-burning period in order to characterize annual-average HAP concentrations from this source category.

General Approach

A multi-step process, that used estimates of atmospheric potassium concentrations from wood burning, was used to estimate HAP concentrations from wood burning during the rest of the year. ADEQ operates a particulate matter sampler in Payson as part of the State Implementation Plan (SIP) monitoring for the region. Samples collected every sixth day with this sampler from July 13, 1994 through April 21, 1995 were analyzed for chemical elements, including potassium, for use in the extrapolation. Additionally, measurements of particulate metals made with this sampler (arsenic, barium, cadmium, lead, manganese and nickel) were combined with the data from the research program sampler to calculate annual average concentrations of those HAPs.

The basic steps in the extrapolation were the following:

1. Atmospheric concentrations of HAPs from wood burning were estimated for September 1994 through January 1995.

2. Some HAPs are emitted by other sources in addition to wood burning. The average concentrations of these HAPs from non-wood burning sources between February and August 1995 were calculated from the HAP measurements. These non-wood burning concentrations were assumed to represent annual averages.

3. The wood burning HAP concentrations for September 1994 through January 1995, from the first step, were added to the average non-wood burning concentrations from the second step to estimate total HAP concentrations for September 1994 through January 1995.

4. The September 1994 through January 1995 total HAP concentrations from the step were combined with the average HAP concentrations measured from February through August 1995 to estimate annual average HAP concentrations.

These steps are described in greater detail below.

Estimation of HAP Concentrations from Wood Burning

The steps used to estimate HAP concentrations from wood burning were as follows:

1. The atmospheric concentrations of potassium from wood burning were estimated.

2. The relationships between HAP and potassium concentrations from wood burning were estimated.

3. The relationships from the second step were applied to the wood burning potassium concentrations from the first step to estimate HAP concentrations from wood burning.

Wood burning emits potassium, which is taken up by trees when they grow. If potassium were emitted only by wood burning, the concentration of potassium in the atmosphere could serve directly as a tracer for wood burning emissions. However, there is also potassium in suspended soil dust, which contributes to the potassium concentration in the atmosphere. Fortunately, suspended soil dust also contains iron, which is not emitted in large amounts by wood burning and whose concentration is a constant multiple of the potassium concentration. Therefore, the iron concentration measured in the atmosphere was used to estimate the amount of potassium that was from suspended soil dust. This "soil potassium" was then subtracted from the total potassium to estimate potassium from wood burning.

Additionally, potassium emitted by wood burning is primarily in particles with diameters smaller than 2.5 μm aerodynamic diameter (called $PM_{2.5}$), while most of the suspended soil dust is in larger particles. The ADEQ SIP sampler collected samples of particles above and below 2.5 μm in diameter separately. Using the concentrations from the $PM_{2.5}$ samples reduced the "interference" from soil potassium.

The ratio of potassium to iron in $PM_{2.5}$ from suspended soil dust was estimated from particulate matter measurements made during the warmer months, when wood burning is not expected to occur. Thus, the average $PM_{2.5}$ potassium-to-iron ratio measured with the SIP sampler from July through September 1994 was used as the ratio of potassium to iron in suspended soil dust.

Retene is a polycyclic aromatic hydrocarbon that is emitted exclusively by wood burning and which was measured during the research program. Figure 1 shows the retene concentration as a function of calculated wood burning potassium for the seven days that the research program and SIP samplers operated simultaneously. The line in the figure is the linear least-squares fit to the data. The square of the correlation coefficient is 0.76, which indicates that the calculated wood burning potassium is a good tracer for wood burning emissions.

The relationship between HAP emissions from wood burning and wood burning potassium emissions was not determined directly. Instead, the ratios of individual HAPs to retene in wood burning emissions were determined, and retene was calculated from the regression shown in Figure 1. The ratios of HAPs to retene in wood burning emissions were determined as follows, using the research program HAP measurements:

1. The concentrations of HAPs in individual samples due to non-wood burning sources were estimated. This estimation used benzene as a tracer for emissions from non-wood burning sources (primarily motor vehicles). The ratios of individual HAPs to benzene from non-wood burning sources were estimated to equal the average values of these ratios in samples collected from May through June 1995, when wood burning would not be expected to occur. Benzene concentrations measured from January through April 1995 were multiplied by these ratios to estimate HAP concentrations from non-wood burning sources during the period when wood burning would be expected to occur.

2. The HAP concentrations from non-wood burning sources from the first step were subtracted from the total HAP concentrations measured in individual samples from January through April 1995 to estimate HAP concentrations from wood burning. Differences less than zero were set to zero.

3. The average HAP concentrations from wood burning from the second step were divided by the average retene concentration from the same samples to give the desired ratios.

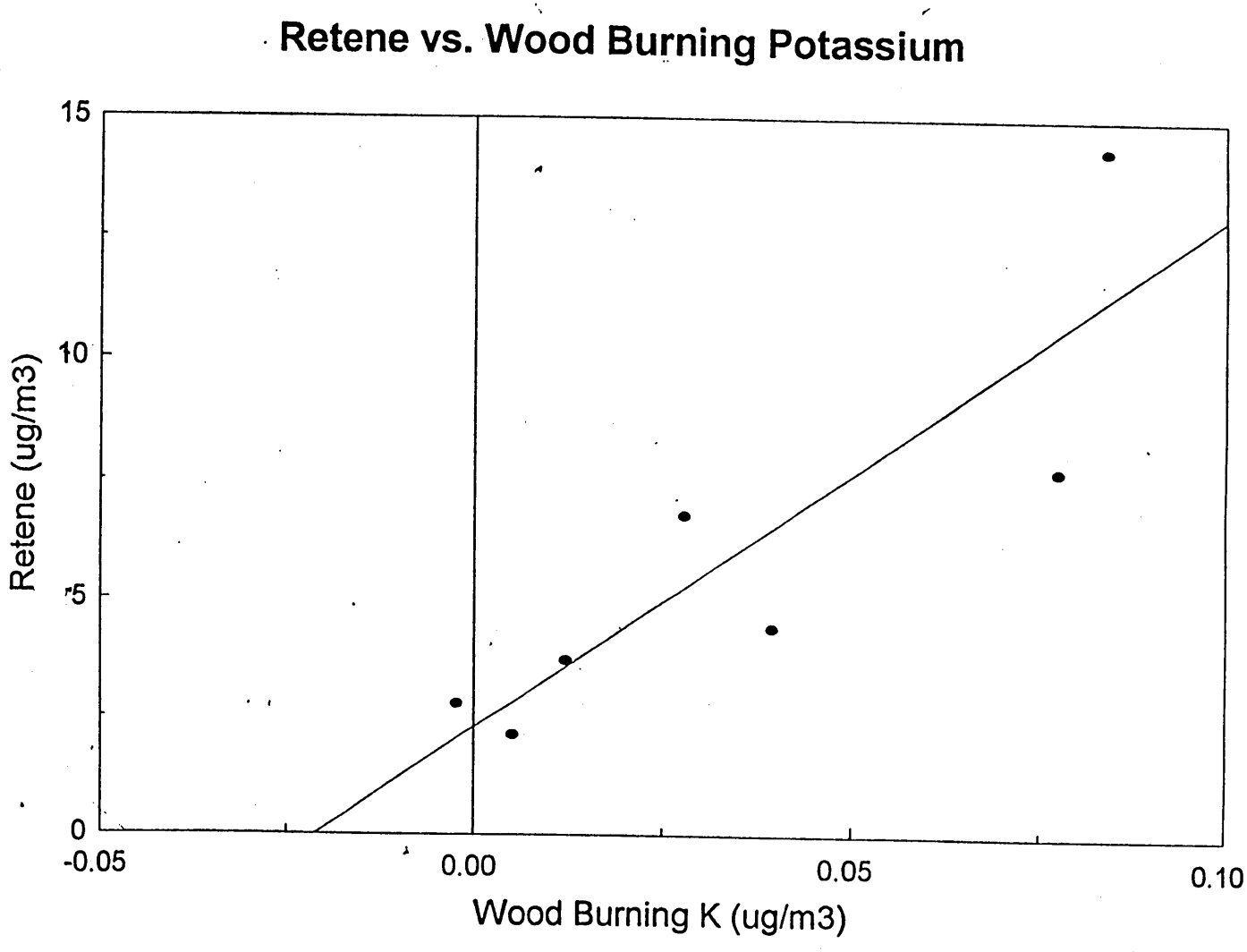


Figure 1 Retene vs. Wood Burning Potassium, Payson SIP Sampler

Annual Average Concentrations

An example of HAPs estimates by the above process for individual days is shown in Figure 2, which is a plot of benzo(a)pyrene concentration as a function of time between mid-July 1994 and mid-August 1995. The figure shows concentrations measured during the research program as well as concentrations estimated on other days as described above. The concentrations follow the expected seasonal pattern, with an increase beginning in early October, a peak in early January, and a decline in April.

Table 1 lists the estimated annual average concentrations along with both the average measured and extrapolated concentrations, for all of the HAPs of interest.

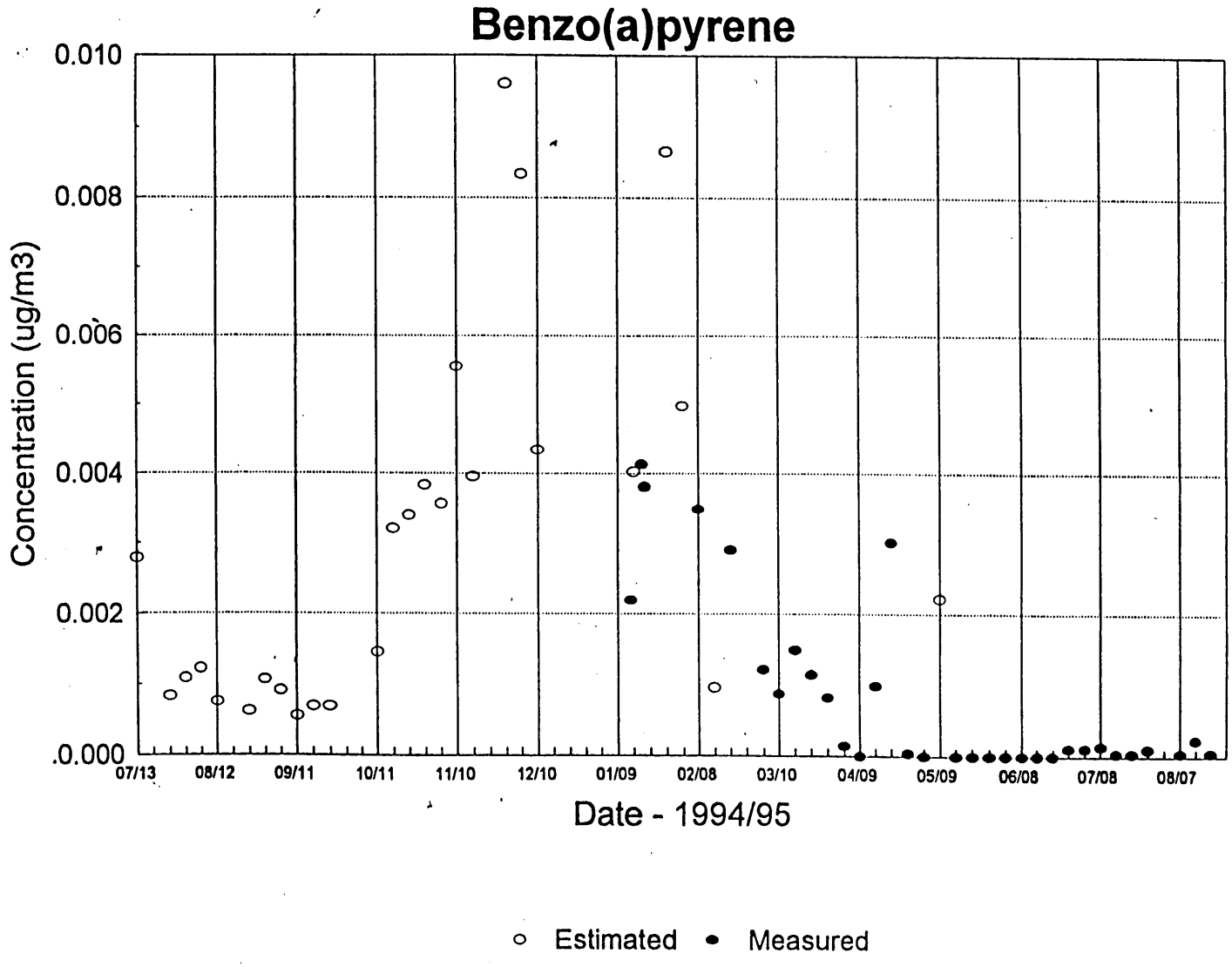


Figure 2 Benzo(a)pyrene Concentrations in Payson

APPENDIX E

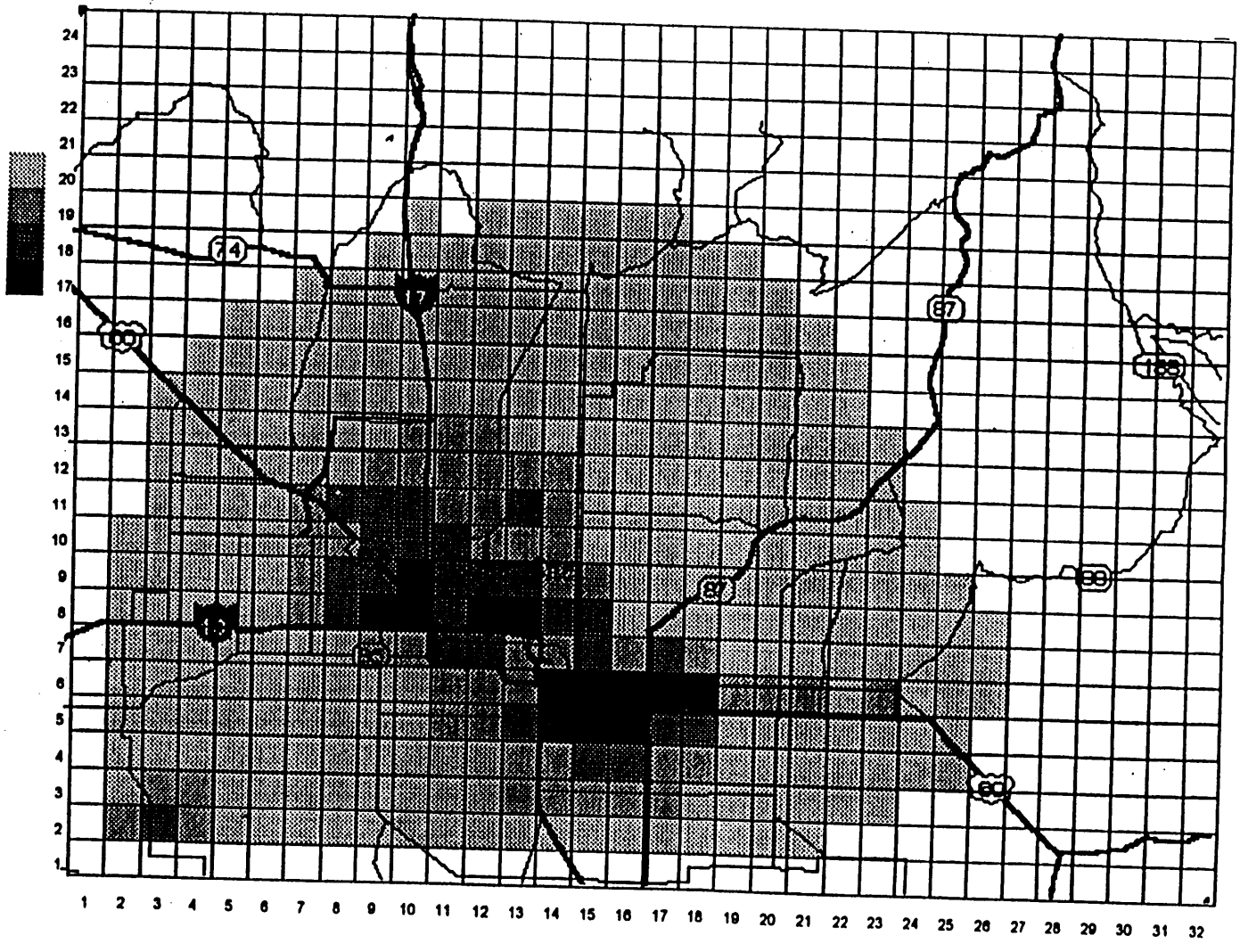
GEOGRAPHIC DISTRIBUTIONS OF ESTIMATED COI CONCENTRATIONS

PHOENIX STUDY AREA

PHOENIX REGION

Acetaldehyde
Concentrations (ug/m3)

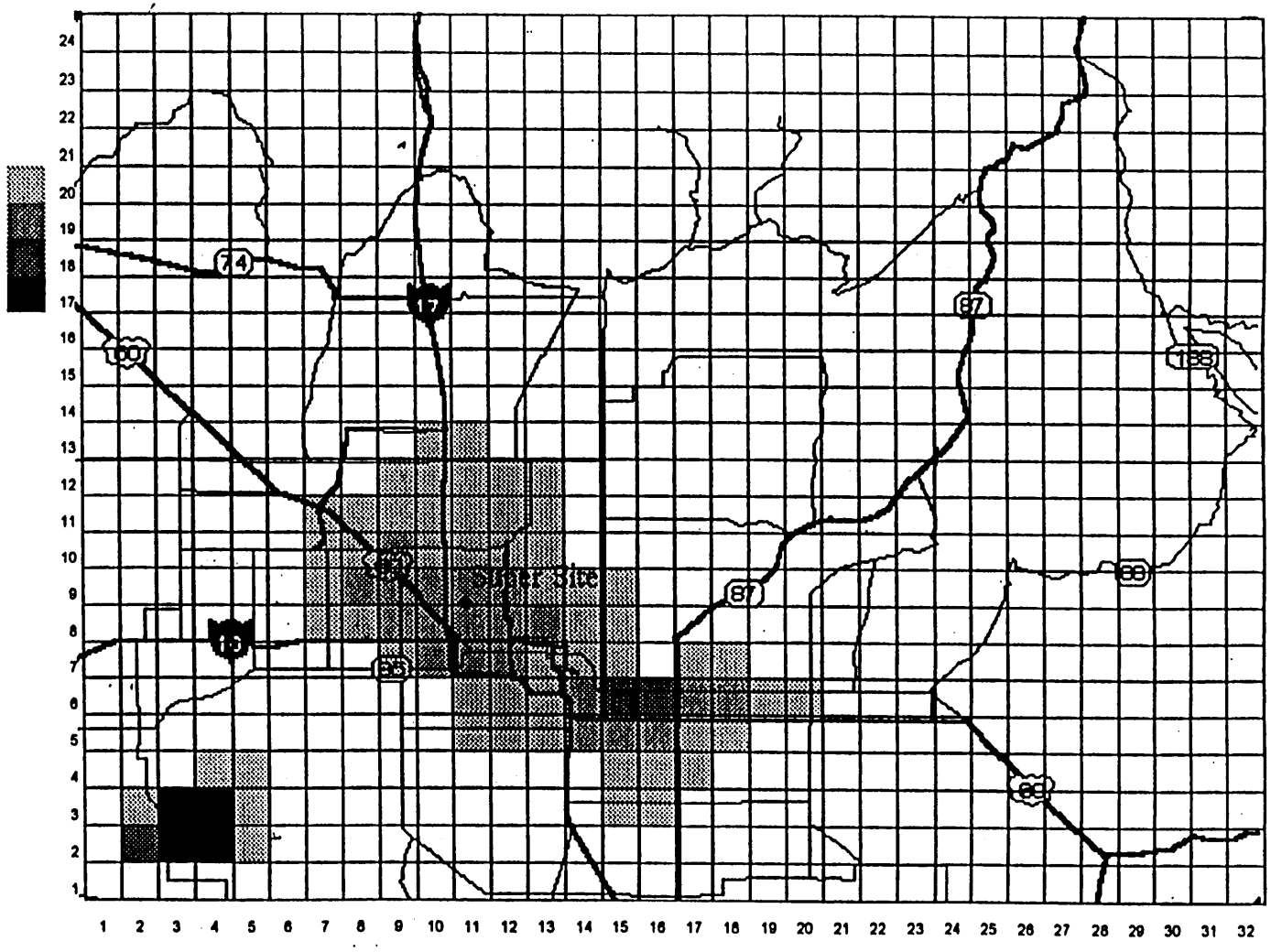
<1.6
1.6-3.2
3.2-4.8
4.8-6.4
>6.4
Highest Cell = 9.5



PHOENIX REGION

Acrolein
Concentrations (ug/m3)

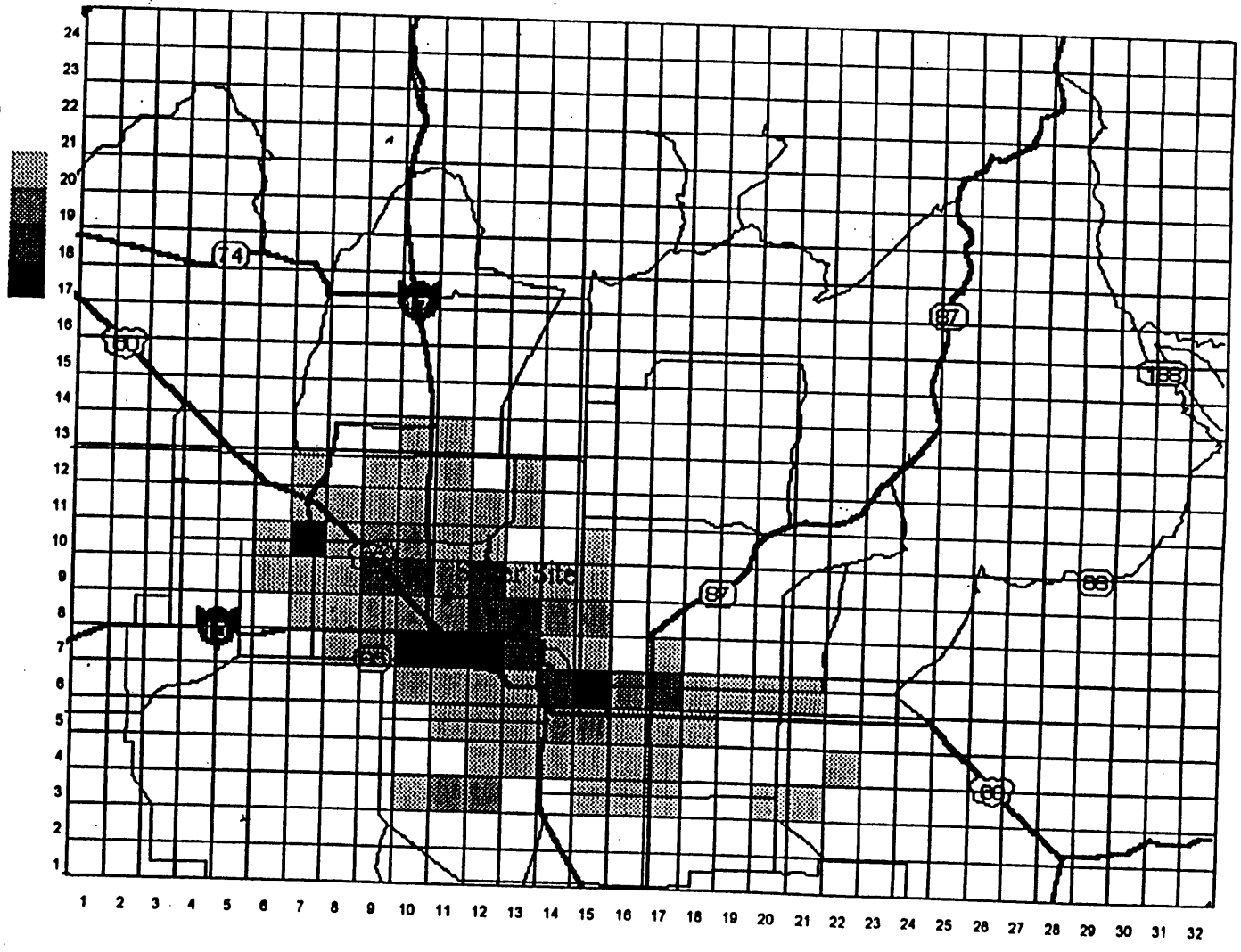
<0.016
0.016-0.032
0.032-0.048
0.048-0.064
>0.064
Highest Cell = 0.105



PHOENIX REGION

Acrylonitrile
Concentrations (ug/m3)

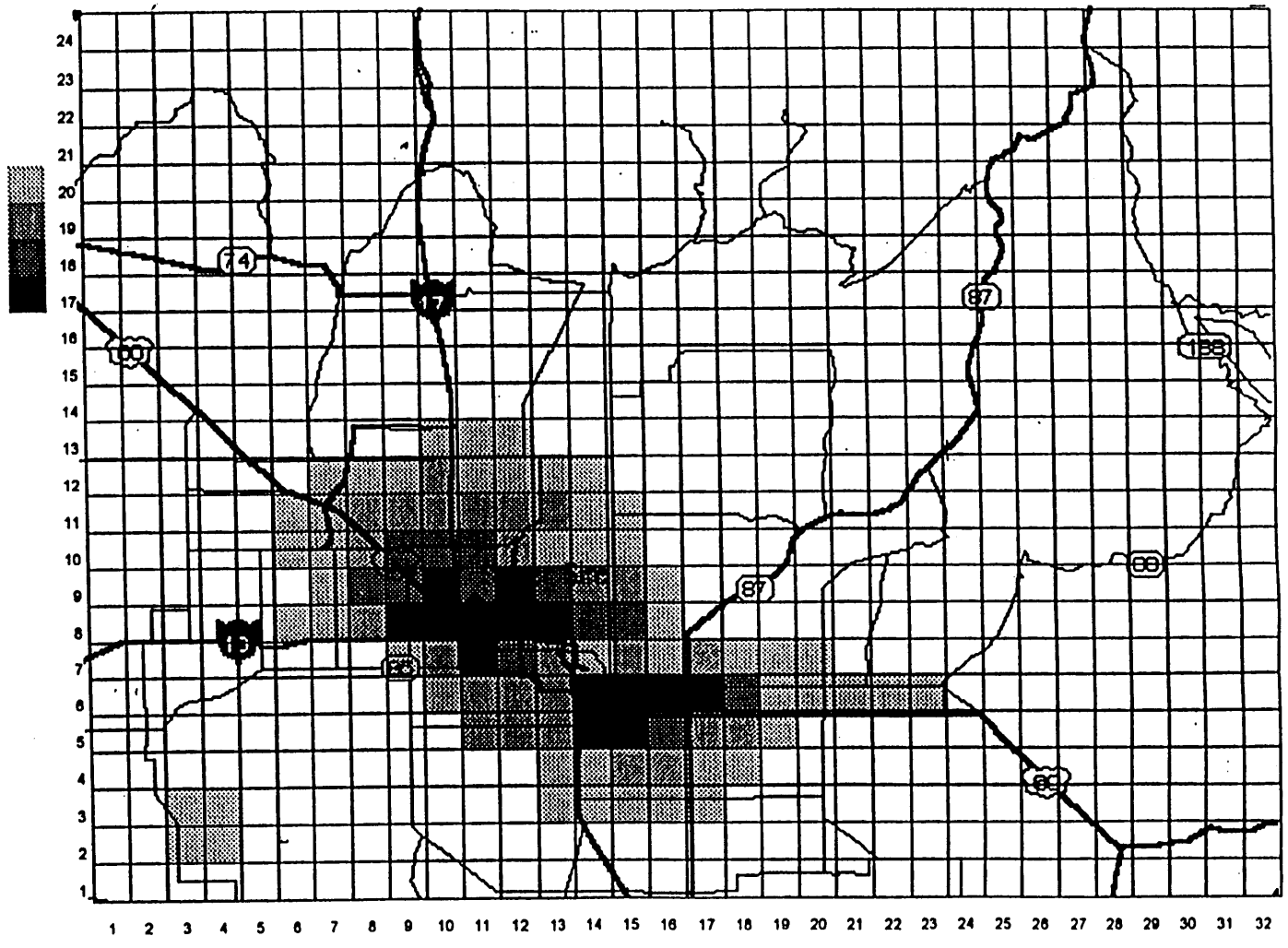
<0.005
0.005-0.010
0.010-0.016
0.016-0.021
>0.021
Highest Cell = 0.036



PHOENIX REGION

1,3-Butadiene
Concentrations (ug/m3)

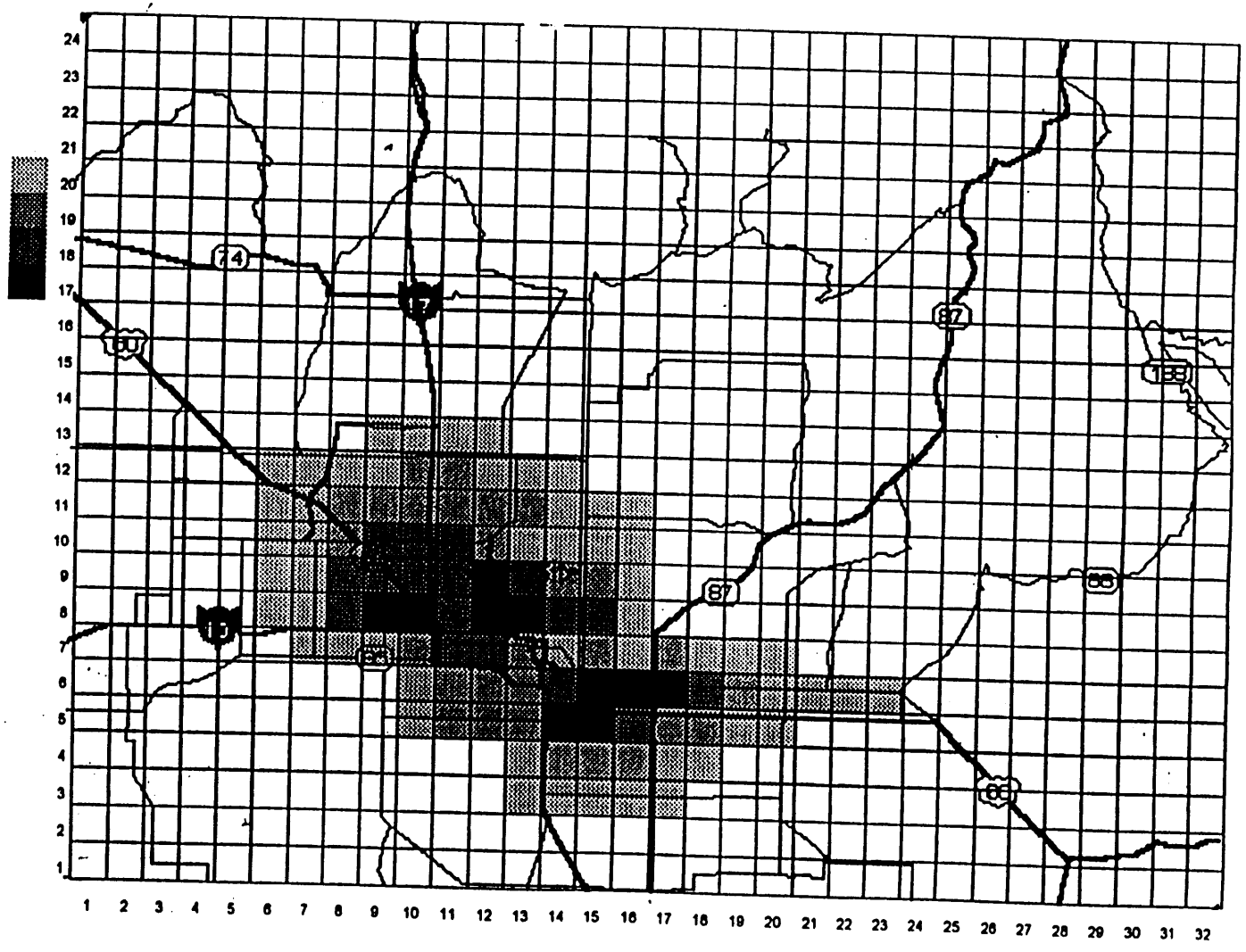
<0.20
0.20-0.41
0.41-0.61
0.61-0.81
>0.81
Highest Cell = 1.28



PHOENIX REGION

Benzene
Concentrations (ug/m3)

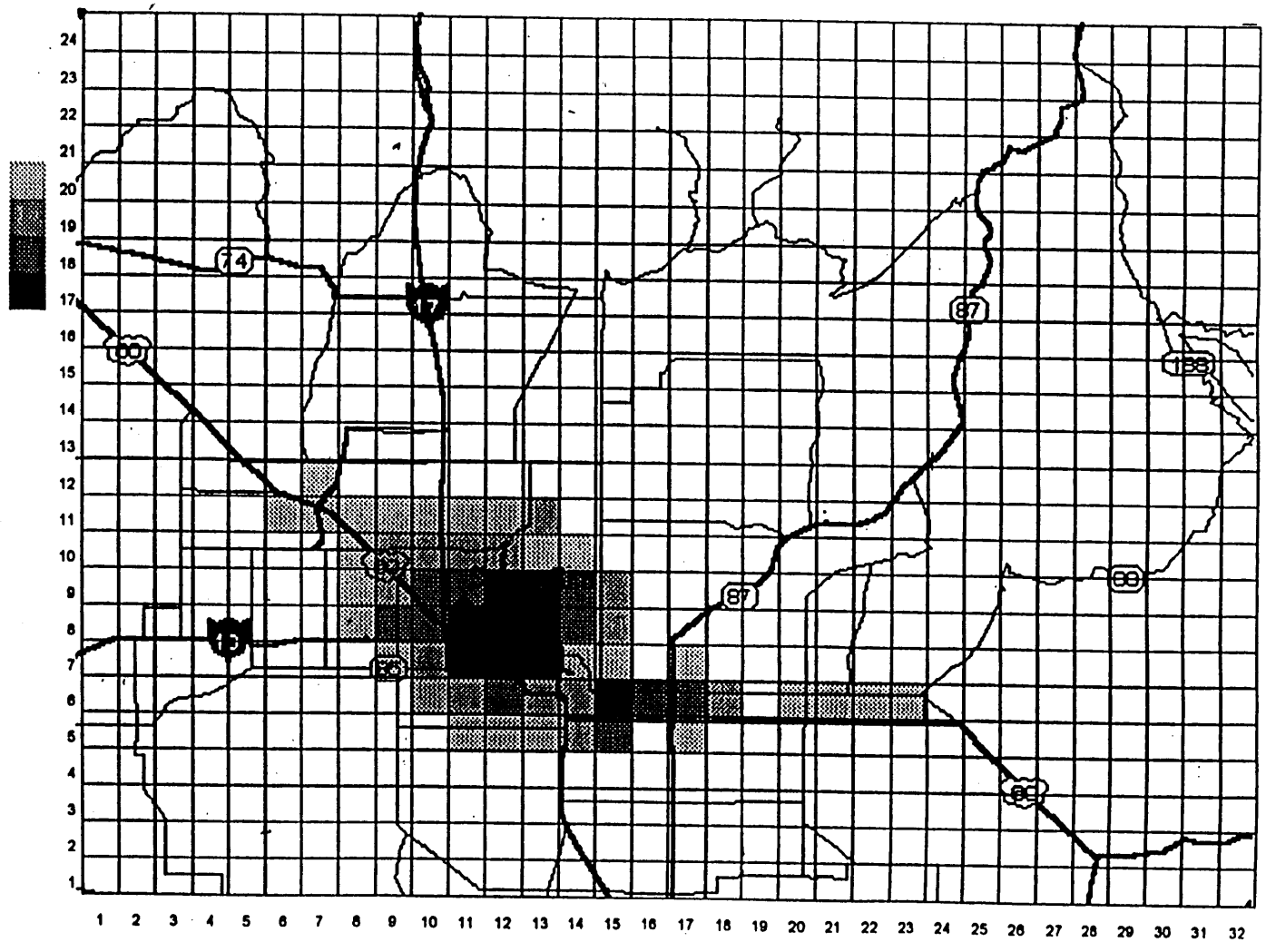
<2.3
2.3-4.6
4.6-7.0
7.0-9.3
>9.3
Highest Cell = 14.2



PHOENIX REGION

Benzo(a)pyrene
Concentrations (ug/m3)

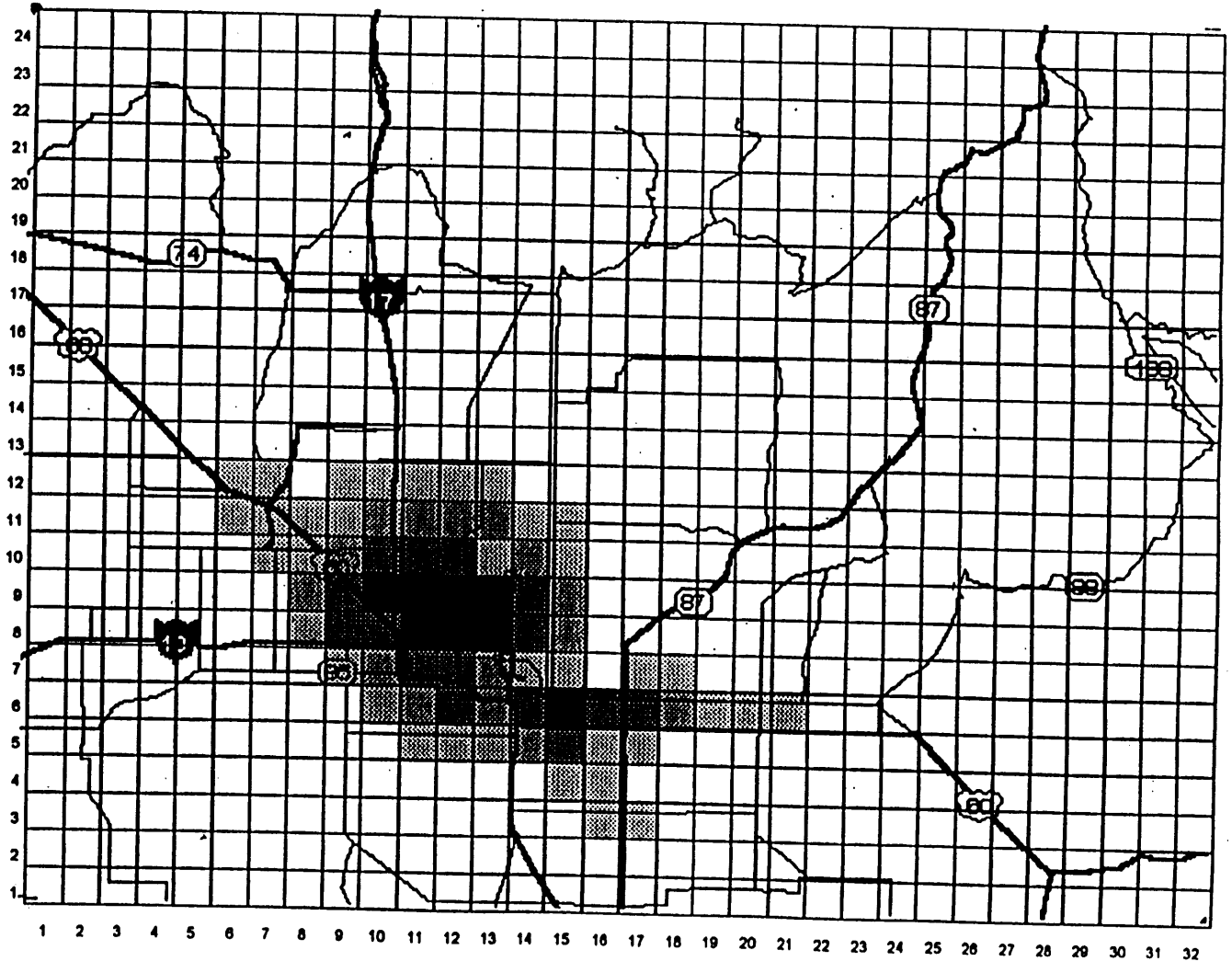
<0.00018
0.00018-0.00035
0.00035-0.00053
0.00053-0.00070
>0.00070
Highest Cell = 0.00089



PHOENIX REGION

Chloroform
Concentrations (ug/m3)

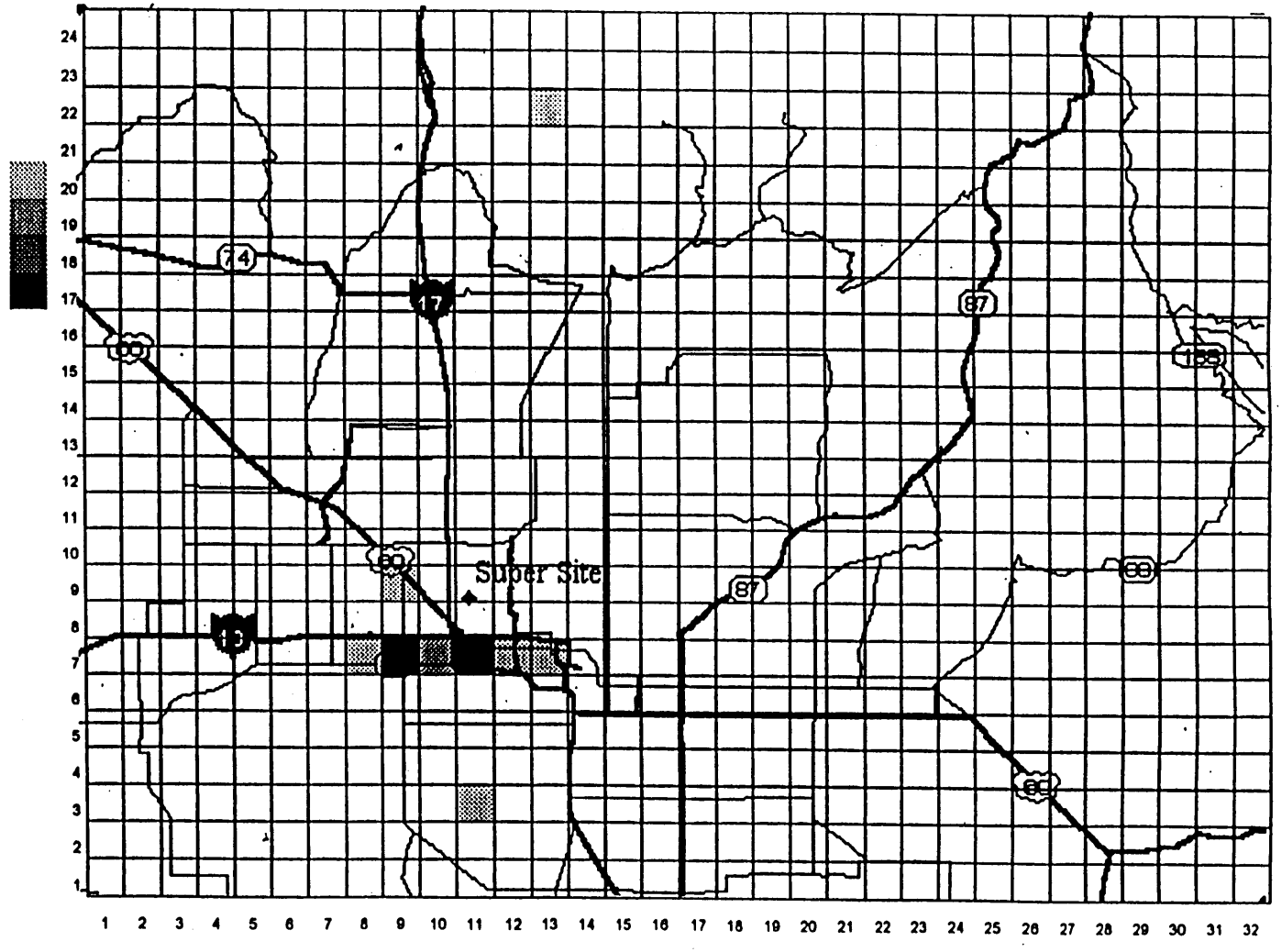
<0.09
0.09-0.19
0.19-0.28
0.28-0.37
>0.37
Highest Cell = 0.48



PHOENIX REGION

Cr (VI)
Concentrations (ug/m3)

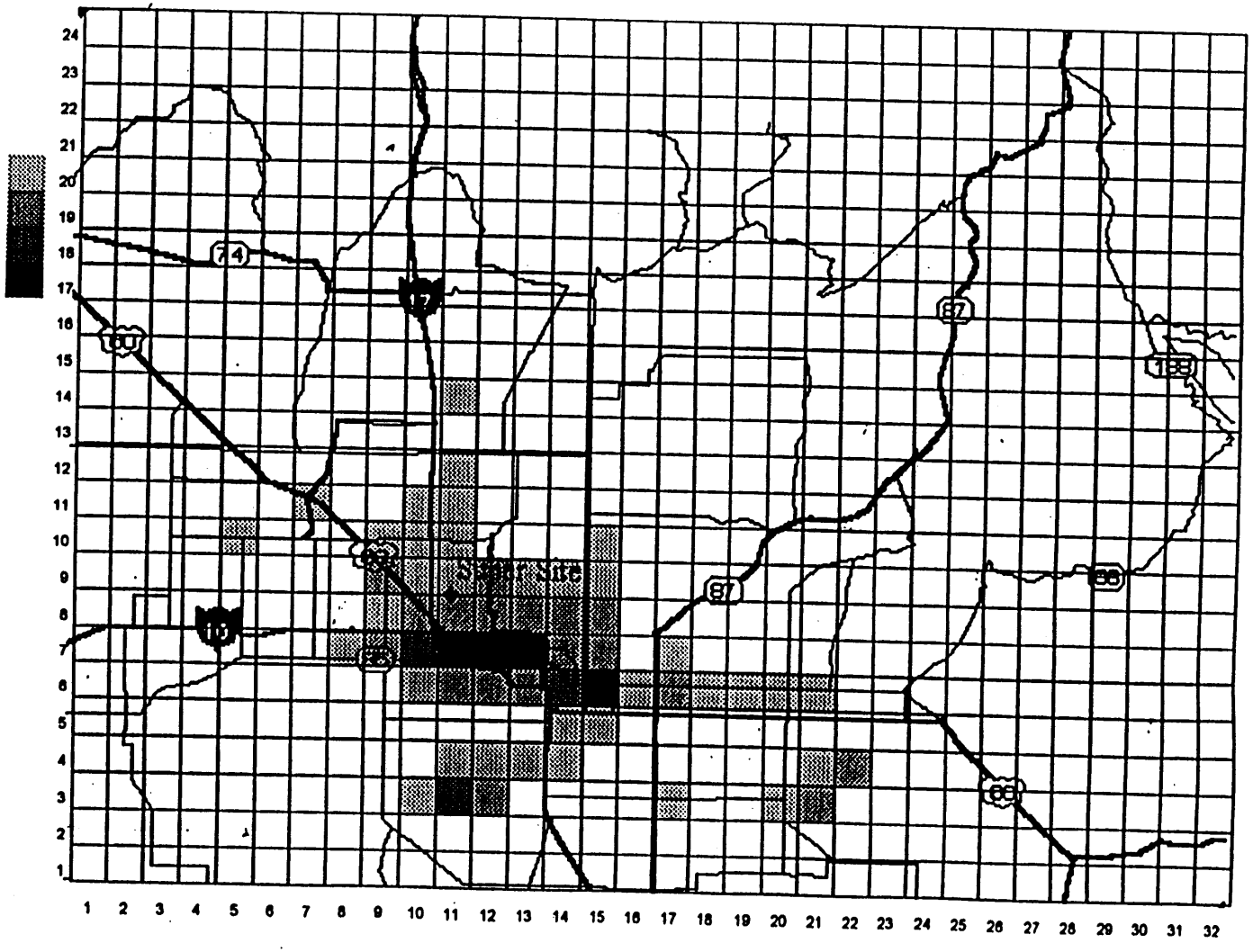
<0.0006
0.0006-0.0012
0.0012-0.0018
0.0018-0.0024
>0.0024
Highest Cell = 0.0038



PHOENIX REGION

p-Dichlorobenzene
Concentrations (ug/m3)

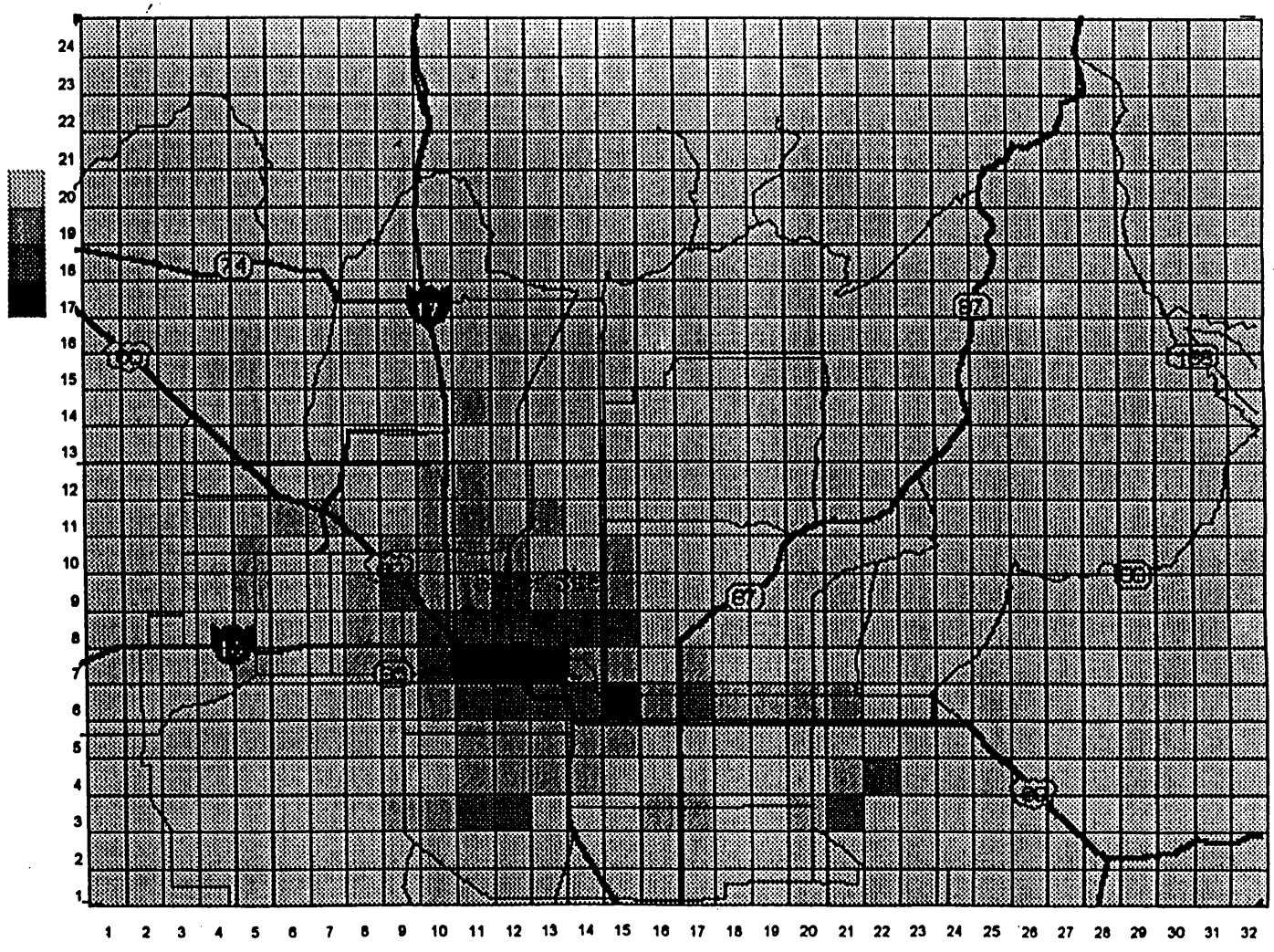
<0.18
0.18-0.35
0.35-0.53
0.53-0.71
>0.71
Highest Cell = 0.91



PHOENIX REGION

Dichloromethane
Concentrations (ug/m3)

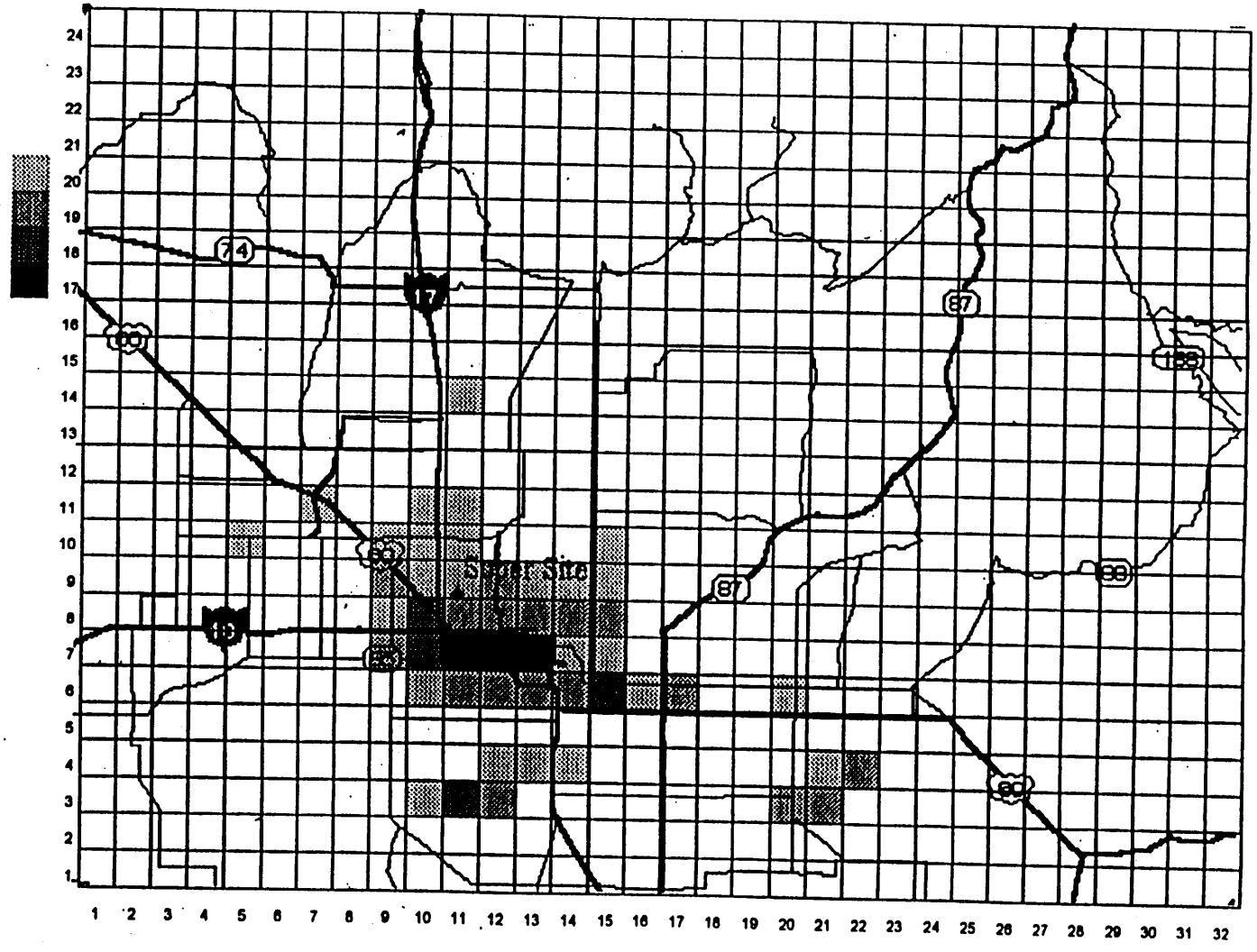
<1.8
1.8-3.5
3.5-5.3
5.3-7.0
>7.0
Highest Cell = 9.0



PHOENIX REGION

Ethylene Dibromide
Concentrations (ug/m3)

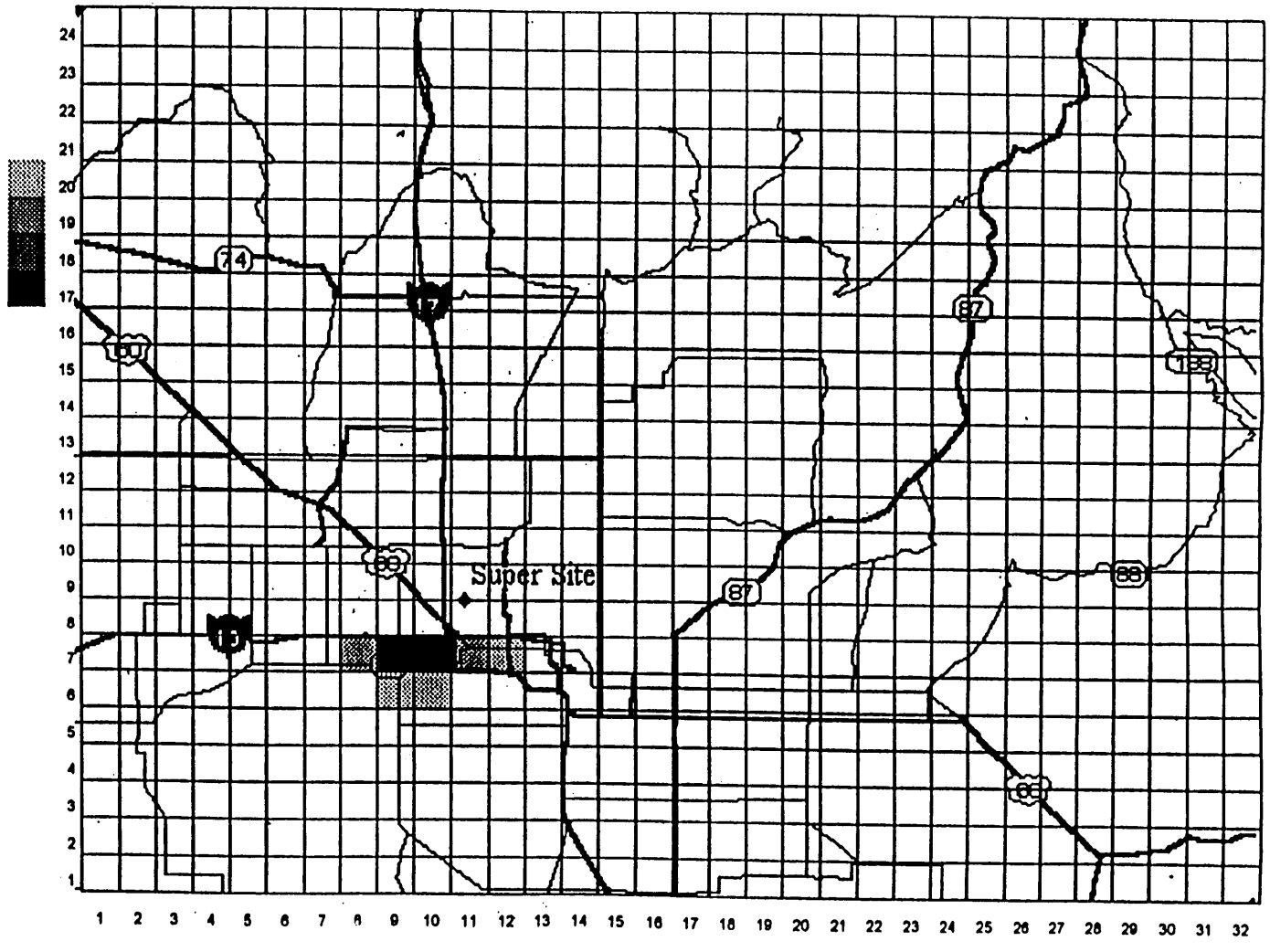
<0.0033
0.0033-0.0066
0.0066-0.0099
0.0099-0.0132
>0.0132
Highest Cell = 0.0167



PHOENIX REGION

Ethylene Oxide
Concentrations (ug/m3)

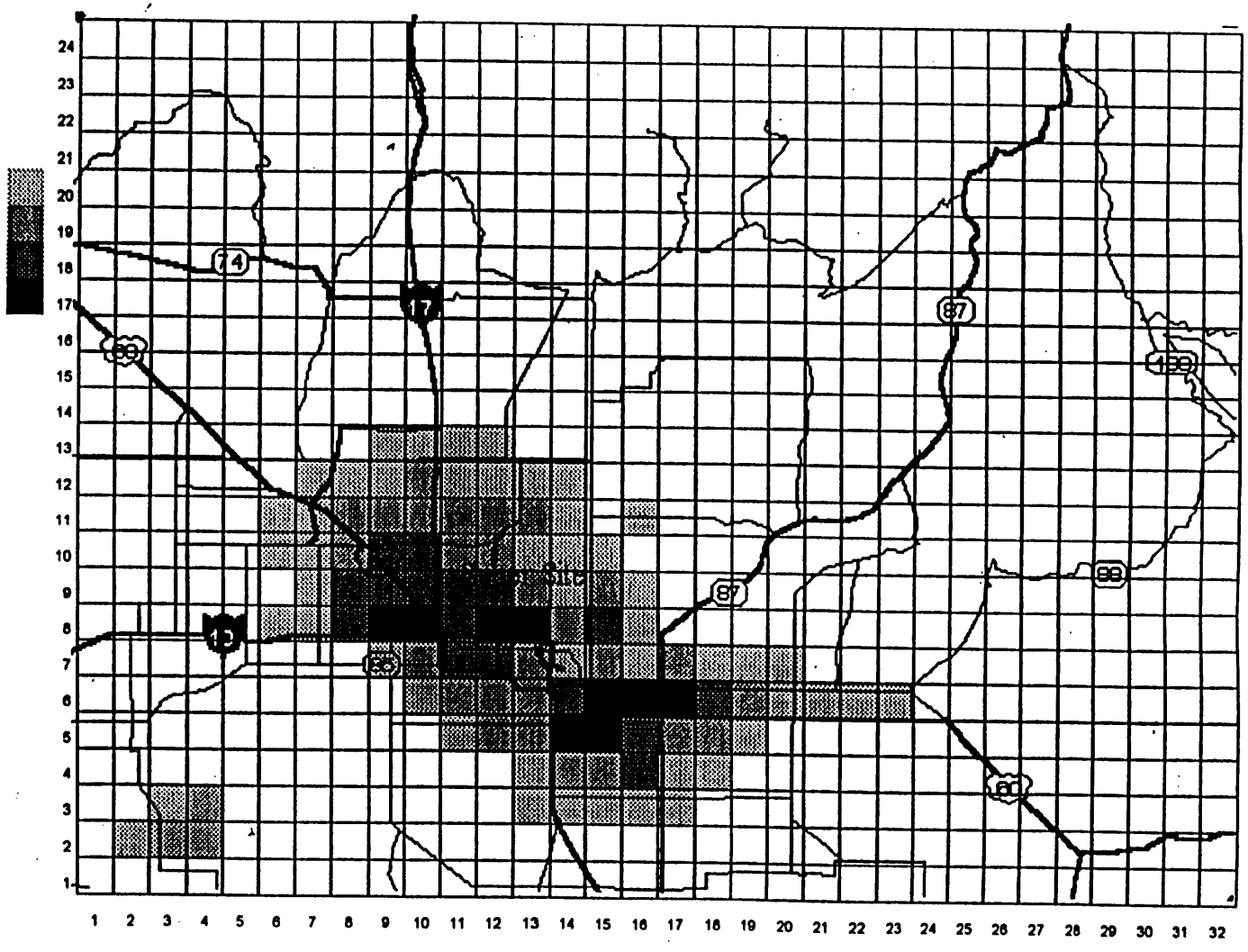
<0.06
0.06-0.12
0.12-0.19
0.19-0.25
>0.25
Highest Cell = 0.89



PHOENIX REGION

Formaldehyde
Concentrations (ug/m3)

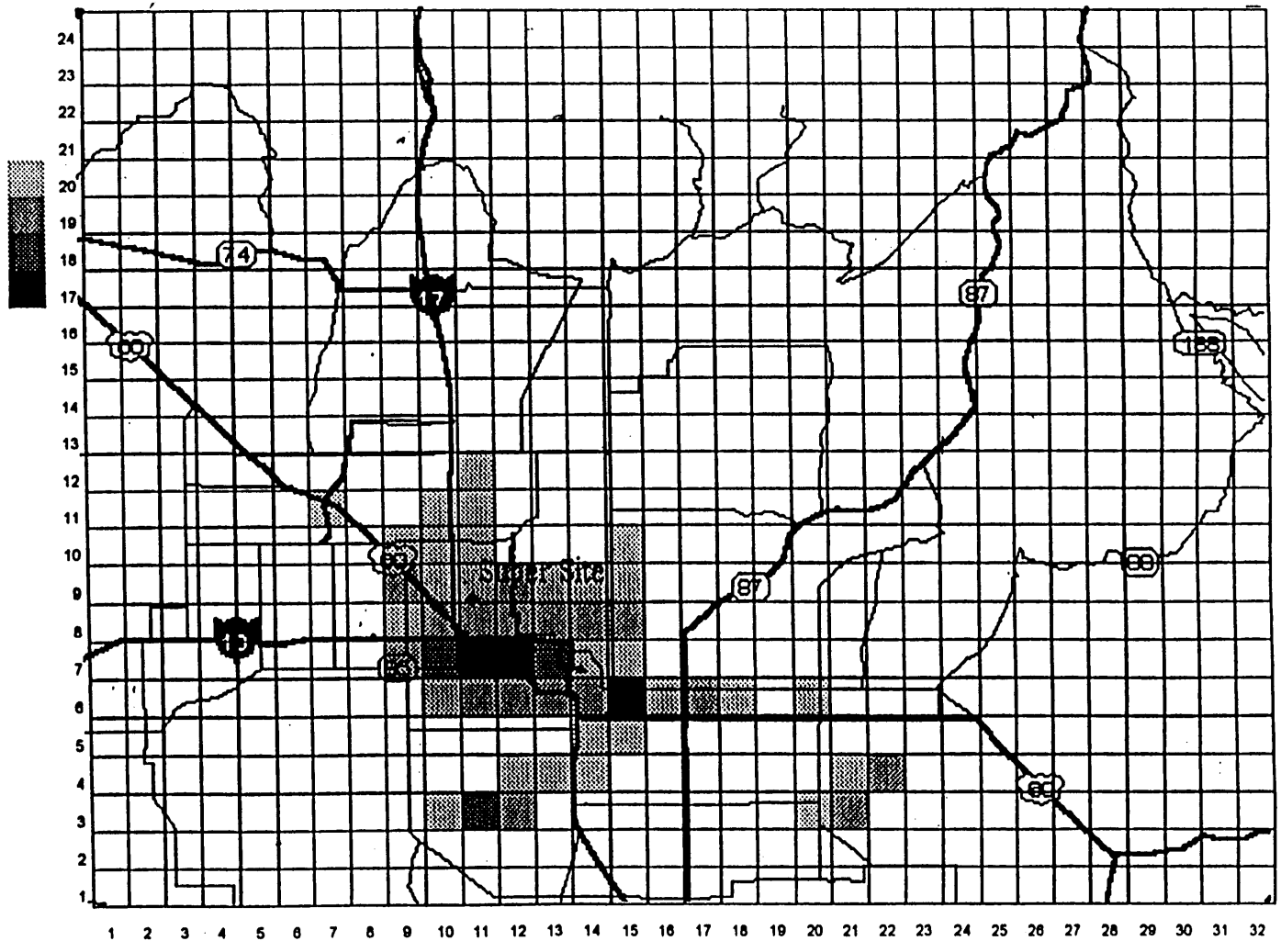
<1.6
1.6-3.2
3.2-4.7
4.7-6.3
>6.3
Highest Cell = 9.8



PHOENIX REGION

Mercury
Concentrations (ug/m3)

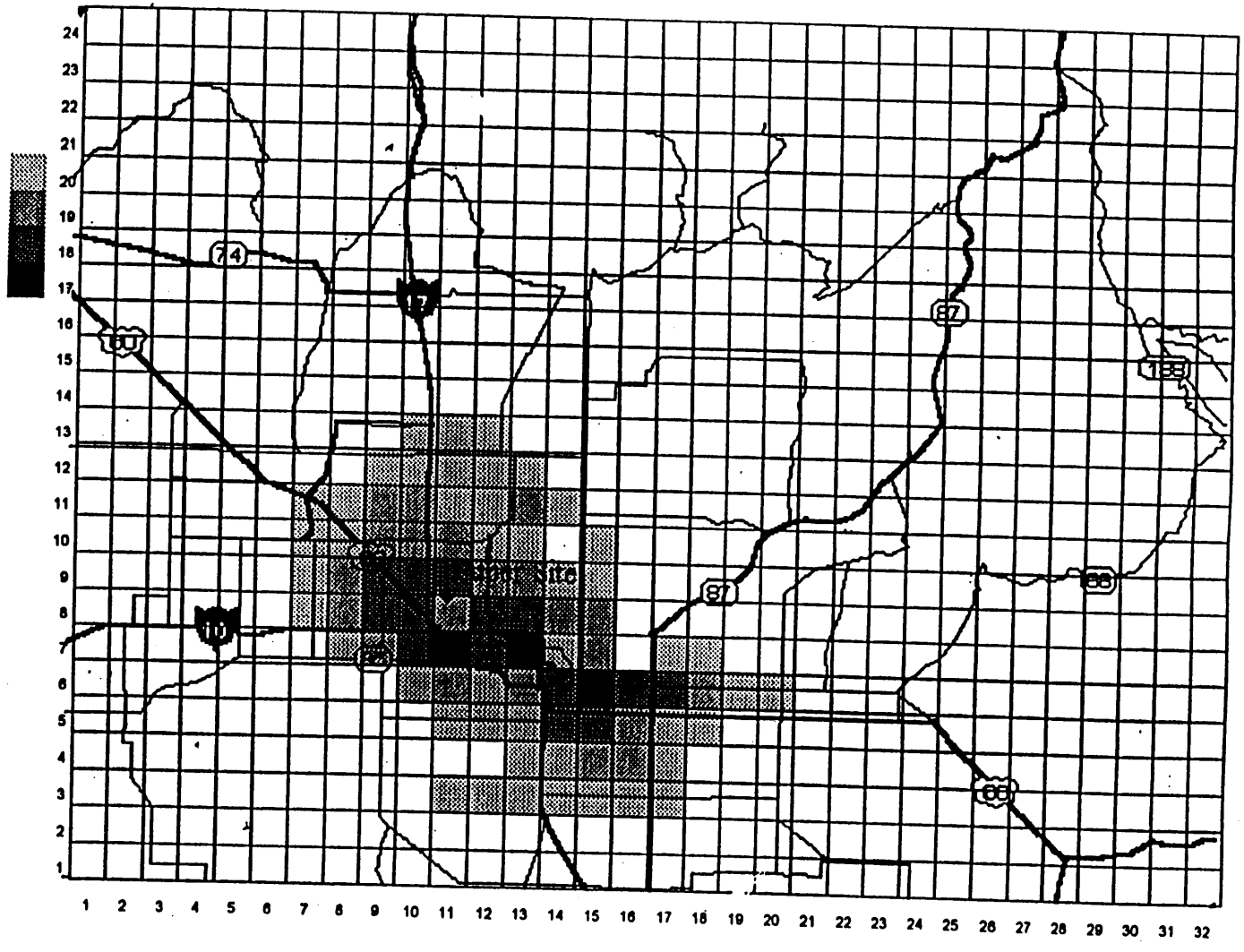
<0.006
0.006-0.012
0.012-0.018
0.018-0.025
>0.025
Highest Cell = 0.039



PHOENIX REGION

Tetrachloroethylene
Concentrations (ug/m3)

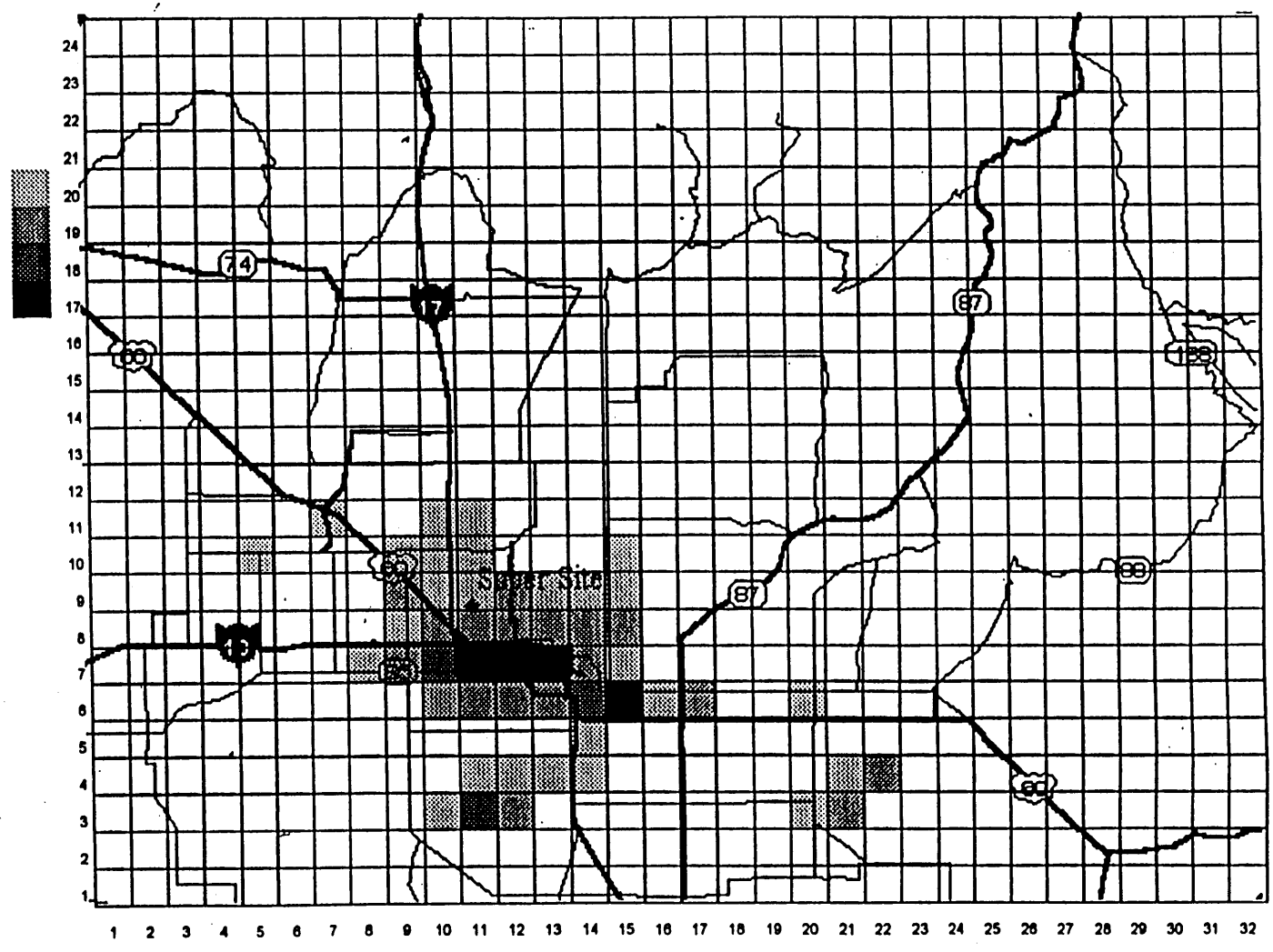
<0.43
0.43-0.87
0.87-1.30
1.30-1.74
>1.74
Highest Cell = 3.20



PHOENIX REGION

Trichloroethylene
Concentrations (ug/m3)

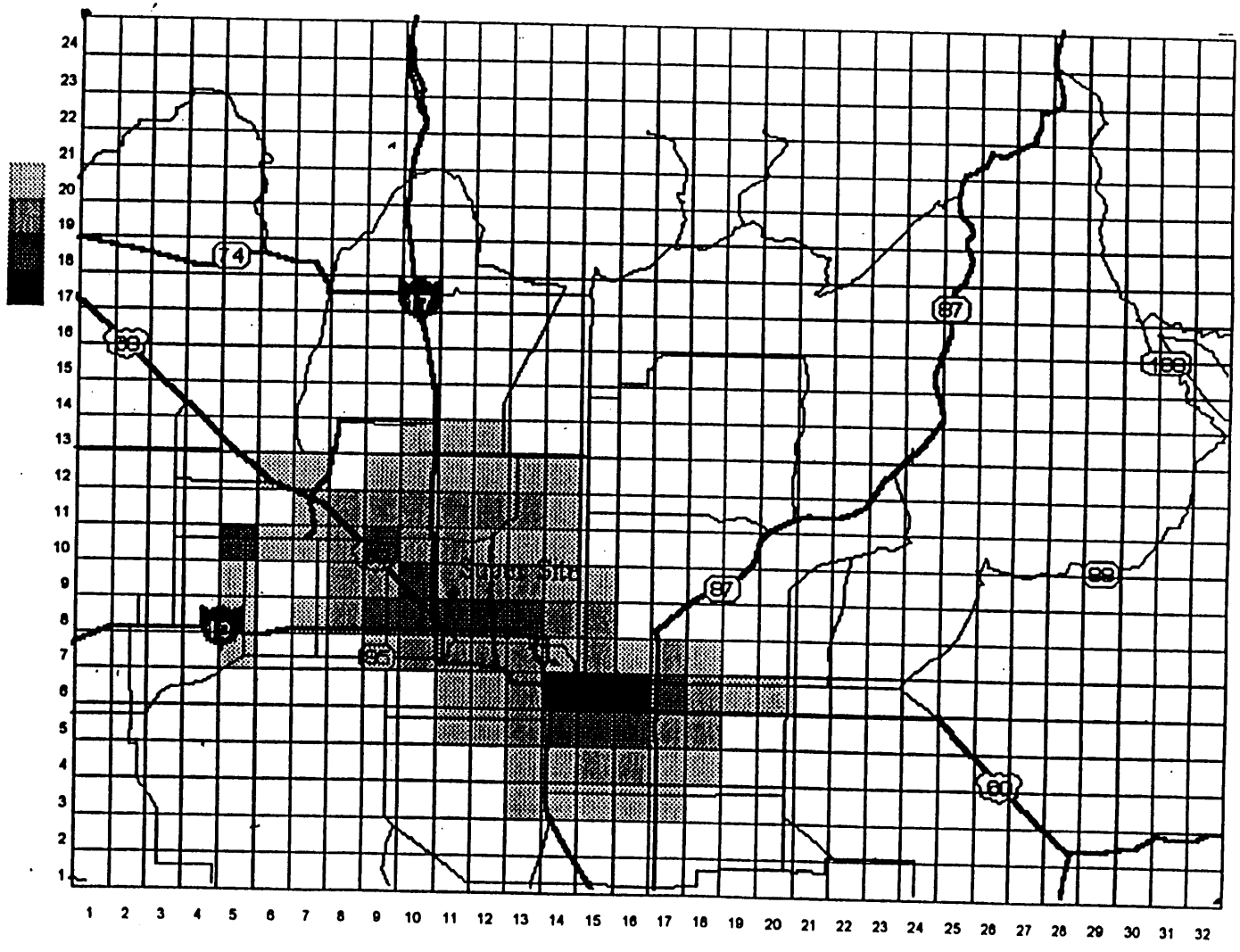
<0.13
0.13-0.25
0.25-0.38
0.38-0.50
>0.50
Highest Cell = 0.65



PHOENIX REGION

Trimethylbenzene
Concentrations (ug/m3)

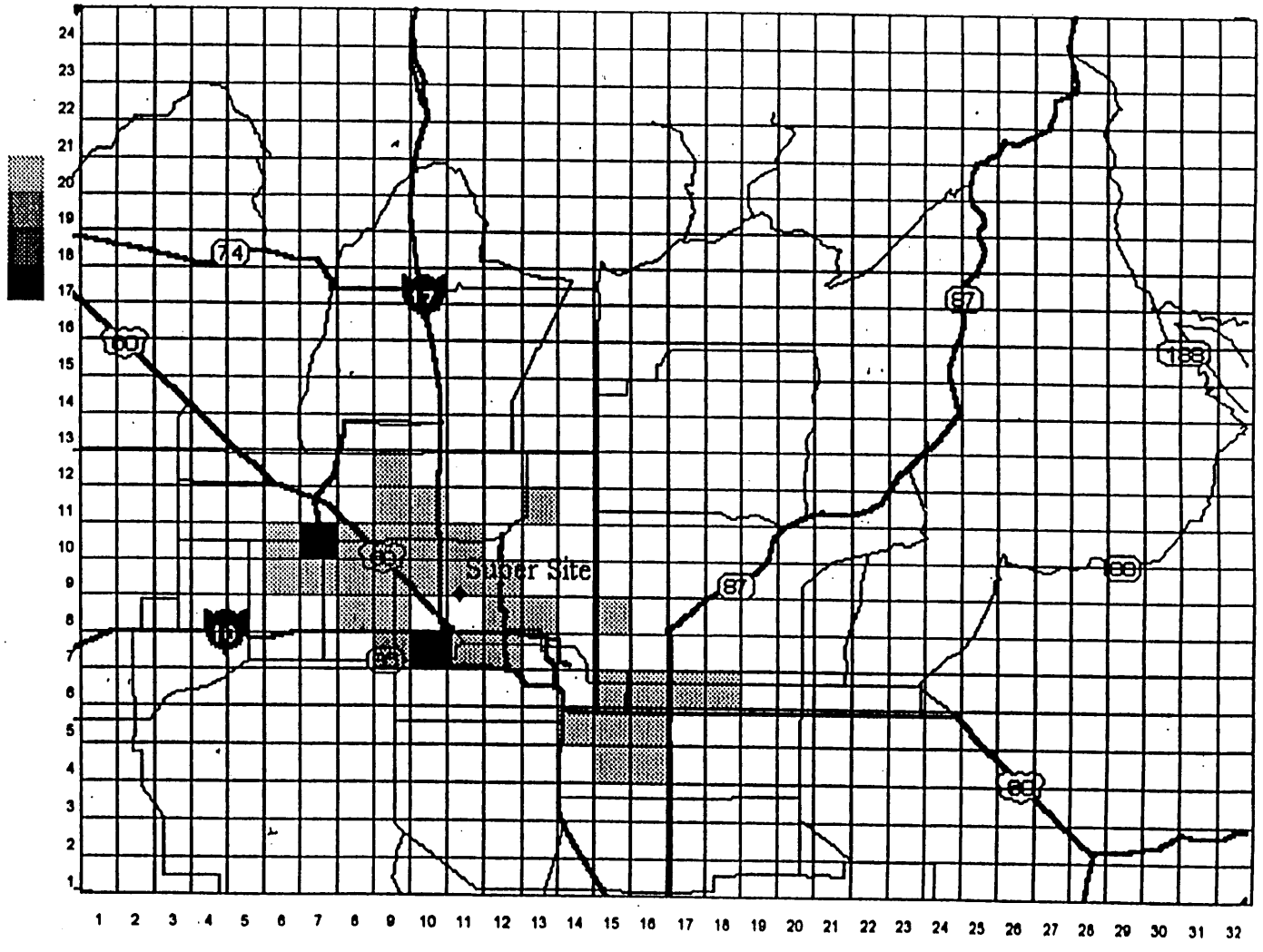
<0.22
0.22-0.43
0.43-0.65
0.65-0.86
>0.86
Highest Cell = 1.11



PHOENIX REGION

Vinyl Chloride
Concentrations (ug/m3)

<0.007
0.007-0.014
0.014-0.020
0.020-0.027
>0.027
Highest Cell = 0.039



TUCSON STUDY AREA

TUCSON REGION

Acrolein

Concentrations (ug/m3)

<0.0042

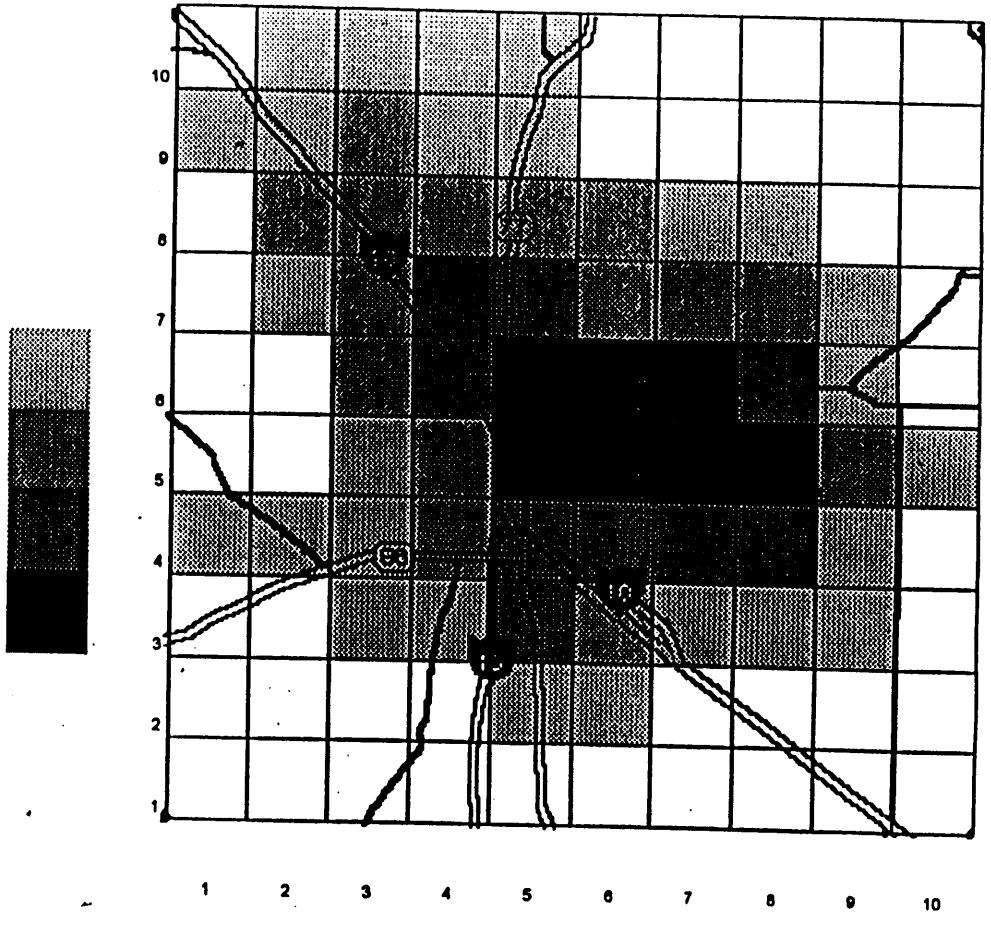
0.0042-0.0083

0.0083-0.0125

0.0125-0.0166

>0.0166

Highest Cell = 0.0217



TUCSON REGION

Acetaldehyde

Concentrations (ug/m3)

<0.8

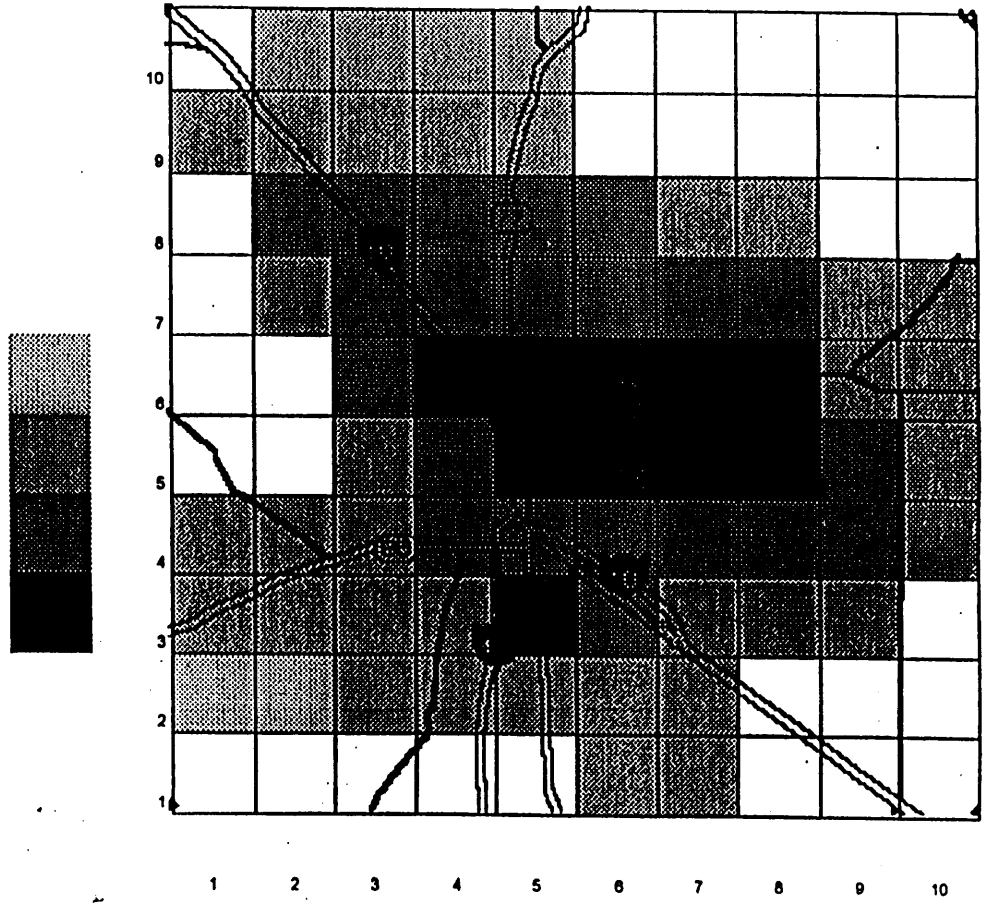
0.8-1.7

1.7-2.5

2.5-3.3

>3.3

Highest Cell = 4.3



TUCSON REGION

1,3-Butadiene

Concentrations (ug/m3)

<0.11

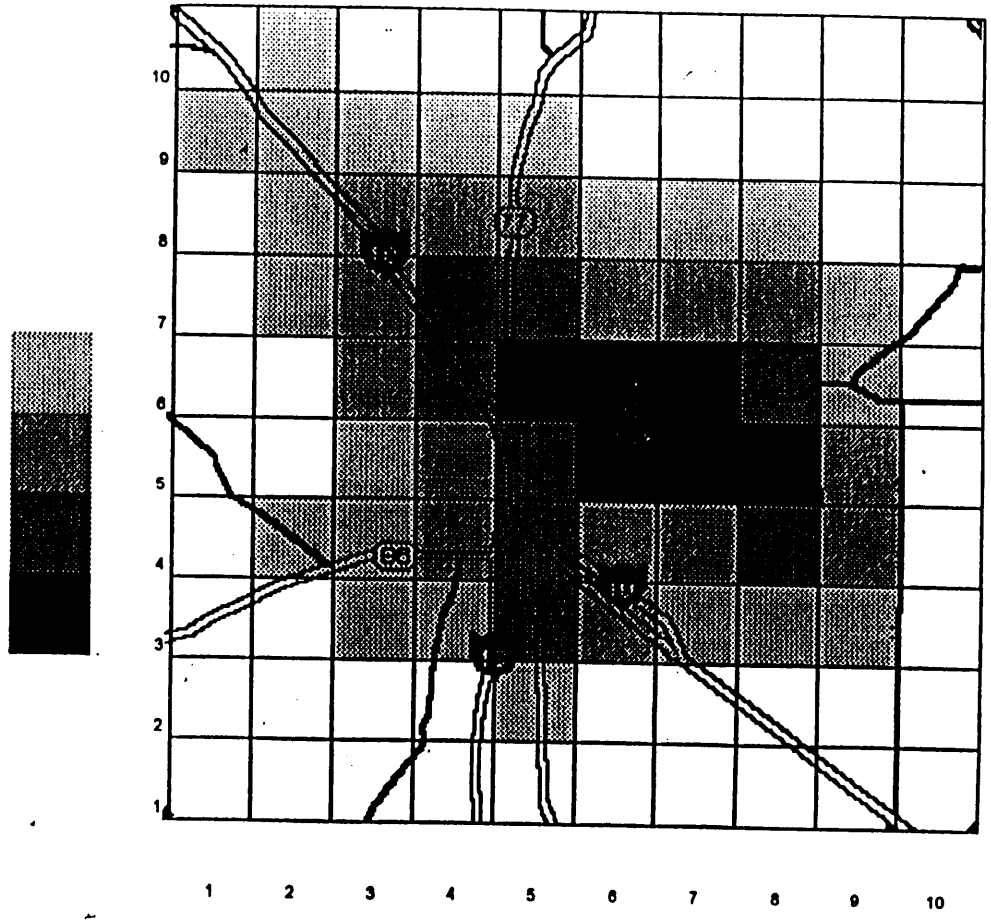
0.11-0.23

0.23-0.34

0.34-0.45

>0.45

Highest Cell = 0.59



TUCSON REGION

Benzo(a)pyrene

Concentrations (ug/m3)

<0.00015

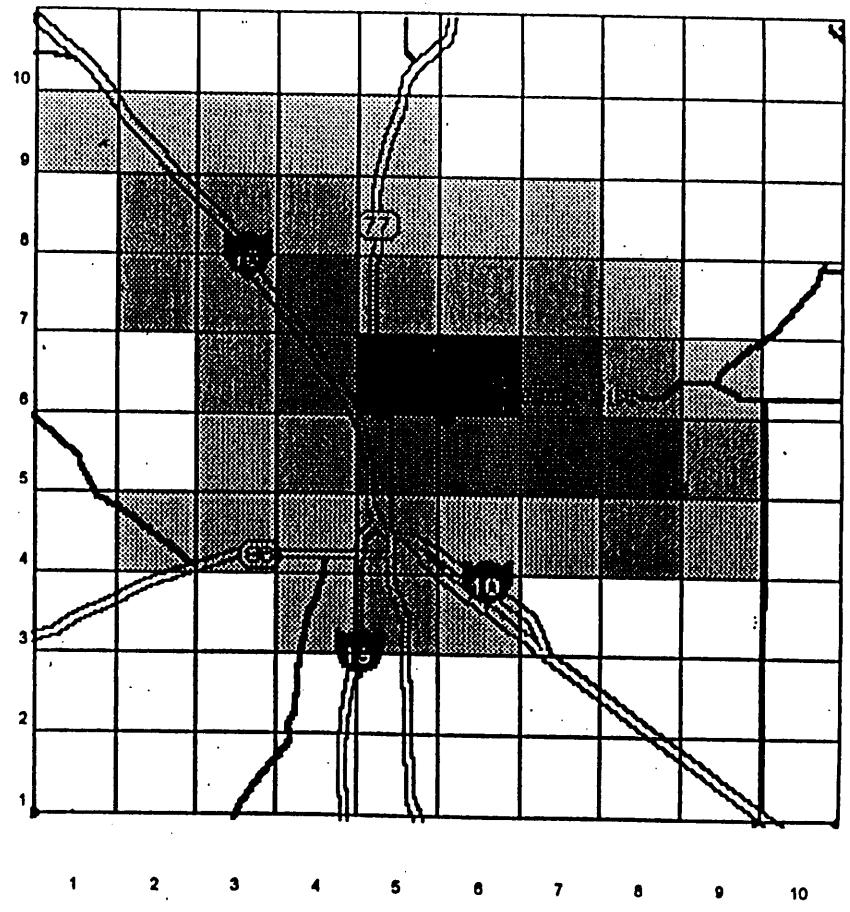
0.00015-0.00029

0.00029-0.00044

0.00044-0.00058

>0.00058

Highest Cell = 0.00076



TUCSON REGION

Chlorobenzene

Concentrations (ug/m3)

<0.005

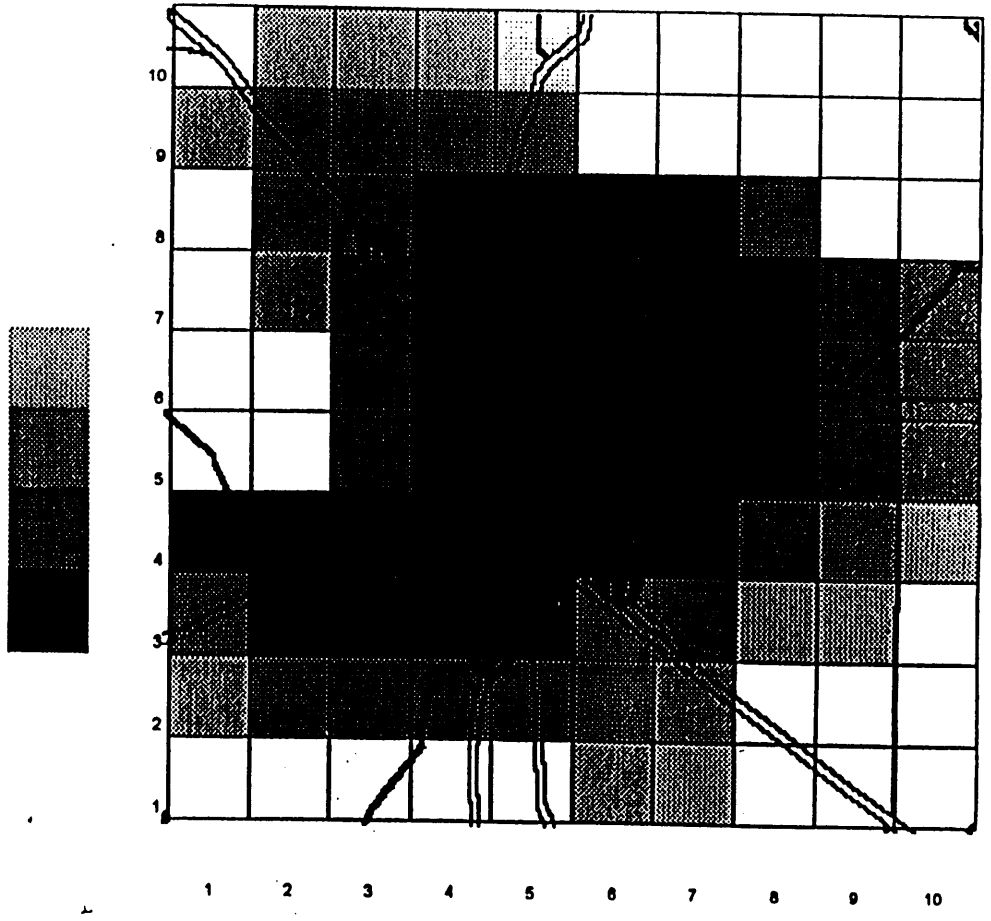
0.005-0.011

0.011-0.016

0.016-0.021

>0.021

Highest Cell = 0.027



TUCSON REGION

Benzene

Concentrations (ug/m3)

<1.1

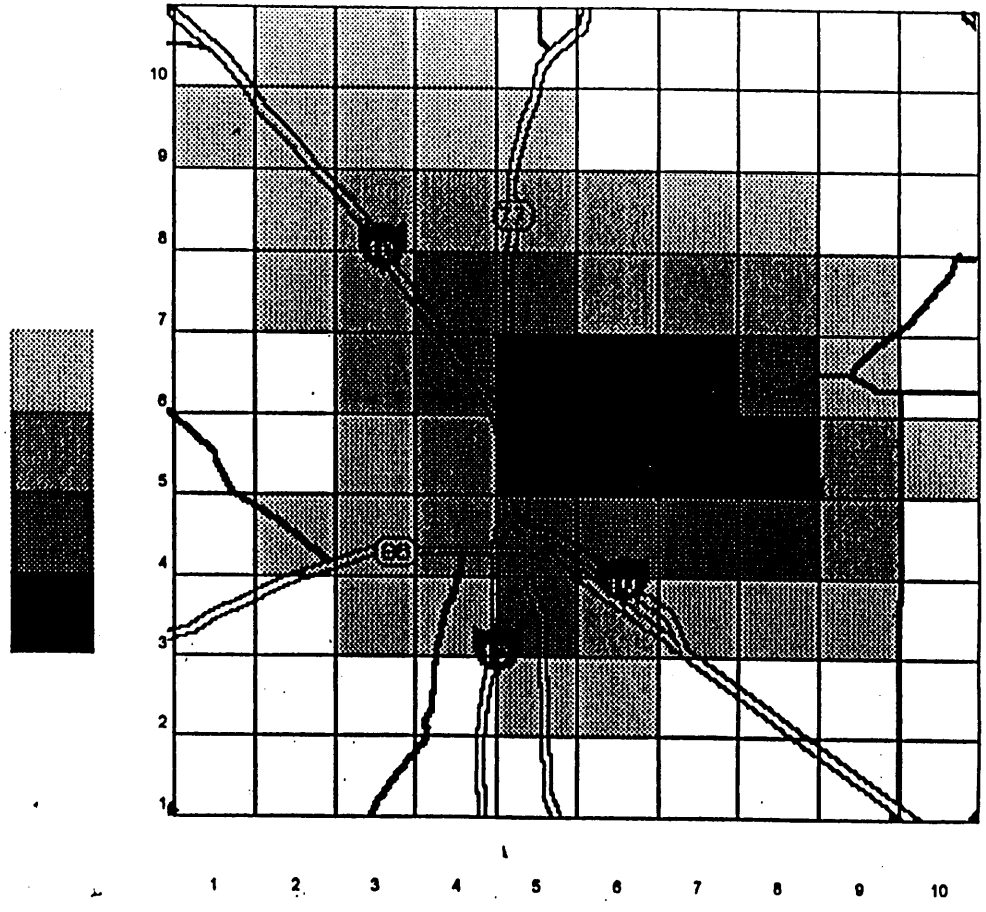
1.1-2.1

2.1-3.2

3.2-4.2

>4.2

Highest Cell = 5.5



TUCSON REGION

Cr (VI)

Concentrations (ug/m3)

<0.000004

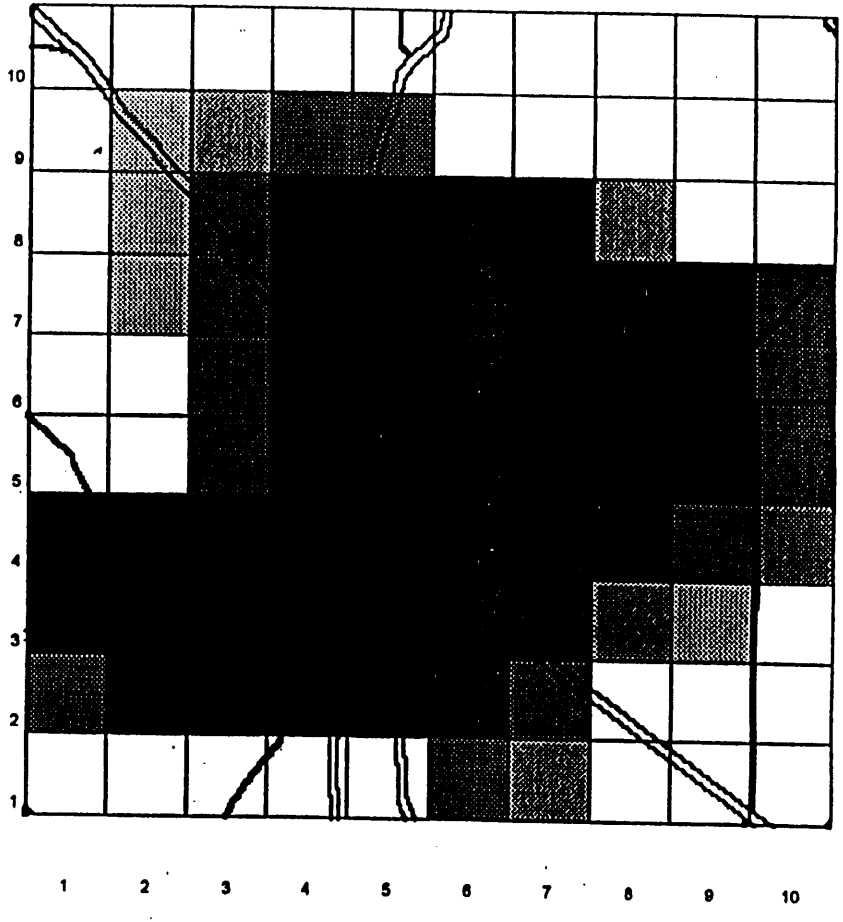
0.000004-0.000009

0.000009-0.000013

0.000013-0.000018

>0.000018

Highest Cell = 0.000022



TUCSON REGION

Chloroform

Concentrations (ug/m3)

<0.07

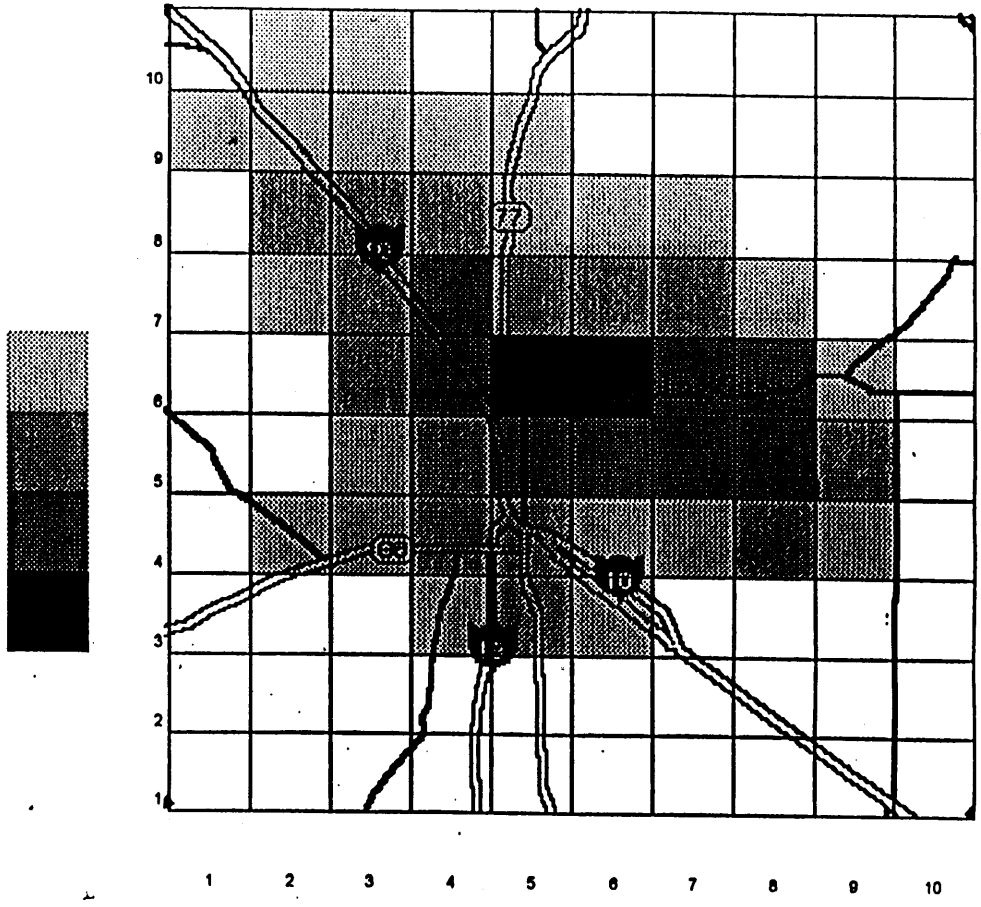
0.07-0.14

0.14-0.21

0.21-0.28

>0.28

Highest Cell = 0.37



TUCSON RE 'ON

Dichloromethane

Concentrations (ug/m3)

<1.3

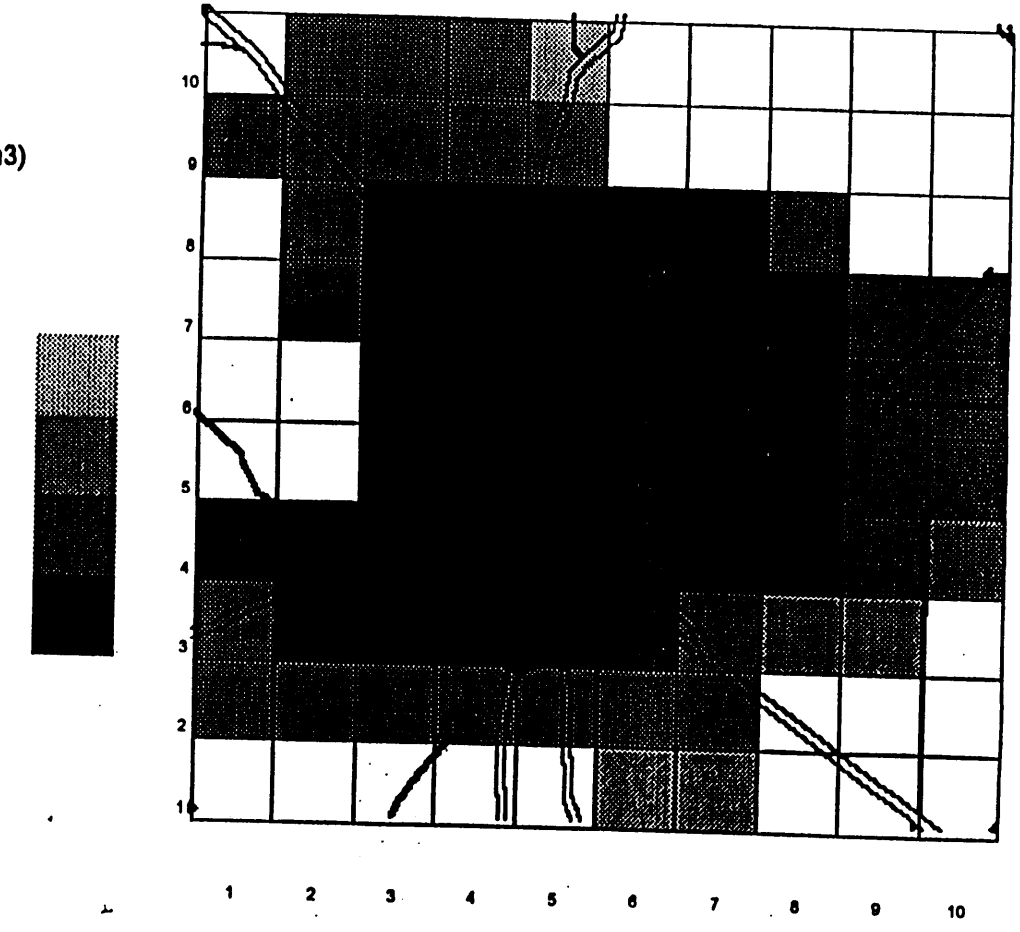
1.3-2.7

2.7-4.0

4.0-5.4

>5.4

Highest Cell = 6.9



TUCSON REGION

p-Dichlorobenzene

Concentrations (ug/m3)

<0.009

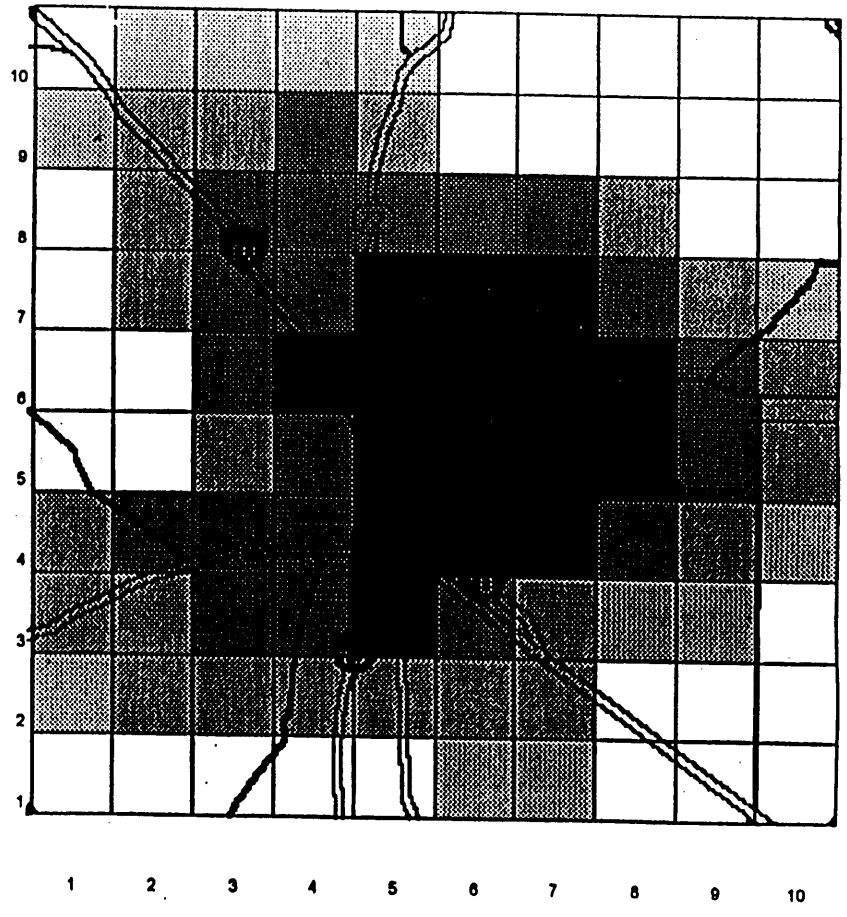
0.009-0.017

0.017-0.026

0.026-0.035

>0.035

Highest Cell = 0.046



TUCSON REGION

Formaldehyde

Concentrations (ug/m3)

<0.27

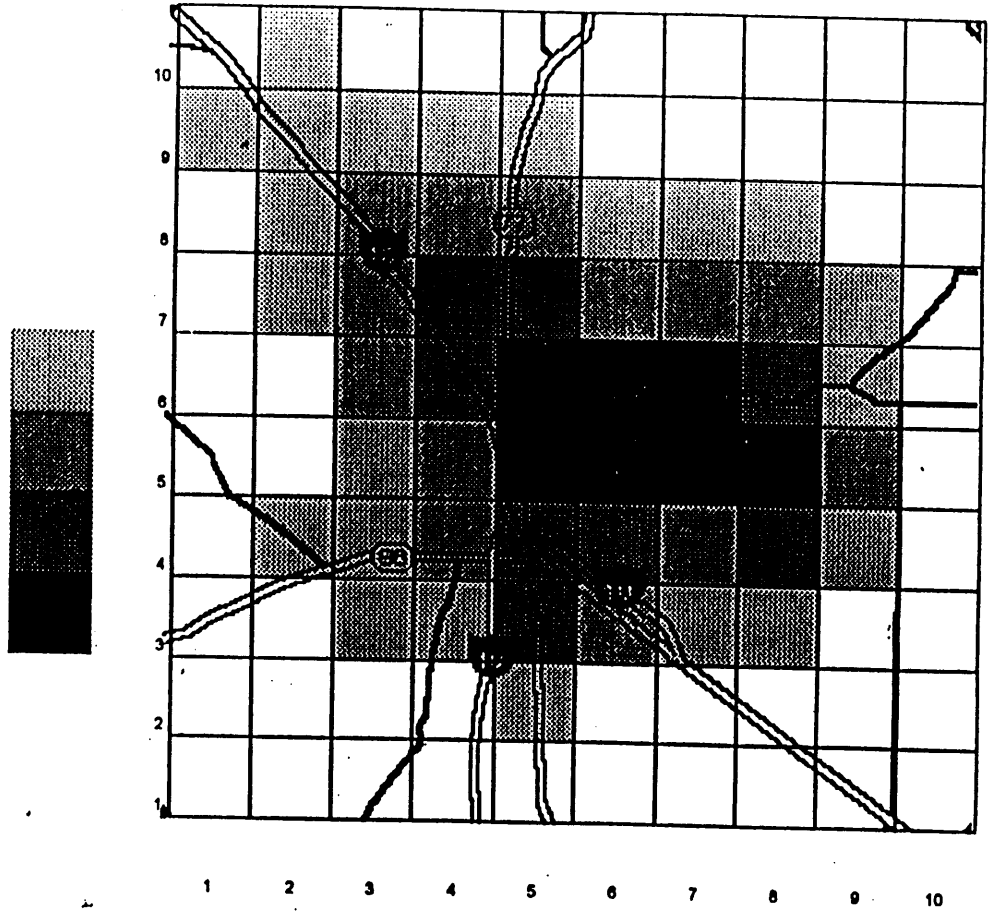
0.27-0.53

0.53-0.80

0.80-1.07

>1.07

Highest Cell = 1.39



TUCSON REGION

Ethylene Oxide

Concentrations (ug/m3)

<0.004

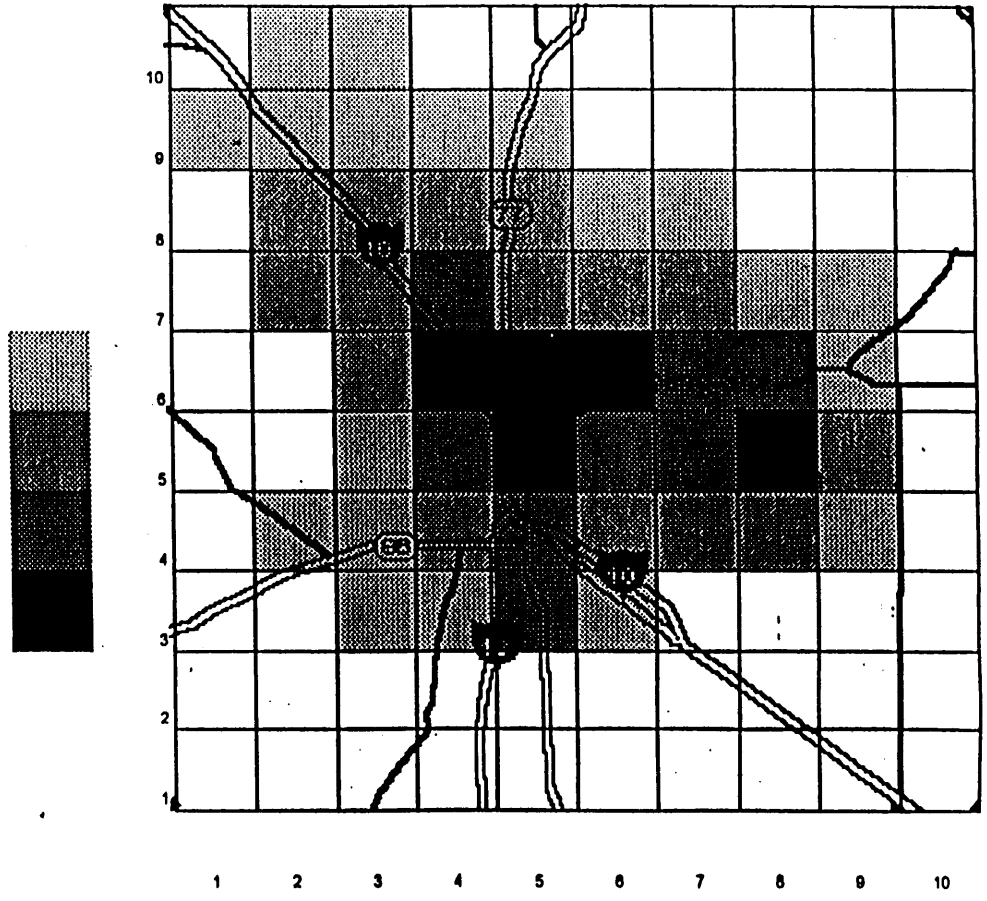
0.004-0.009

0.009-0.013

0.013-0.018

>0.018

Highest Cell = 0.024



TUCSON REGION

Trichloroethylene

Concentrations (ug/m3)

<0.05

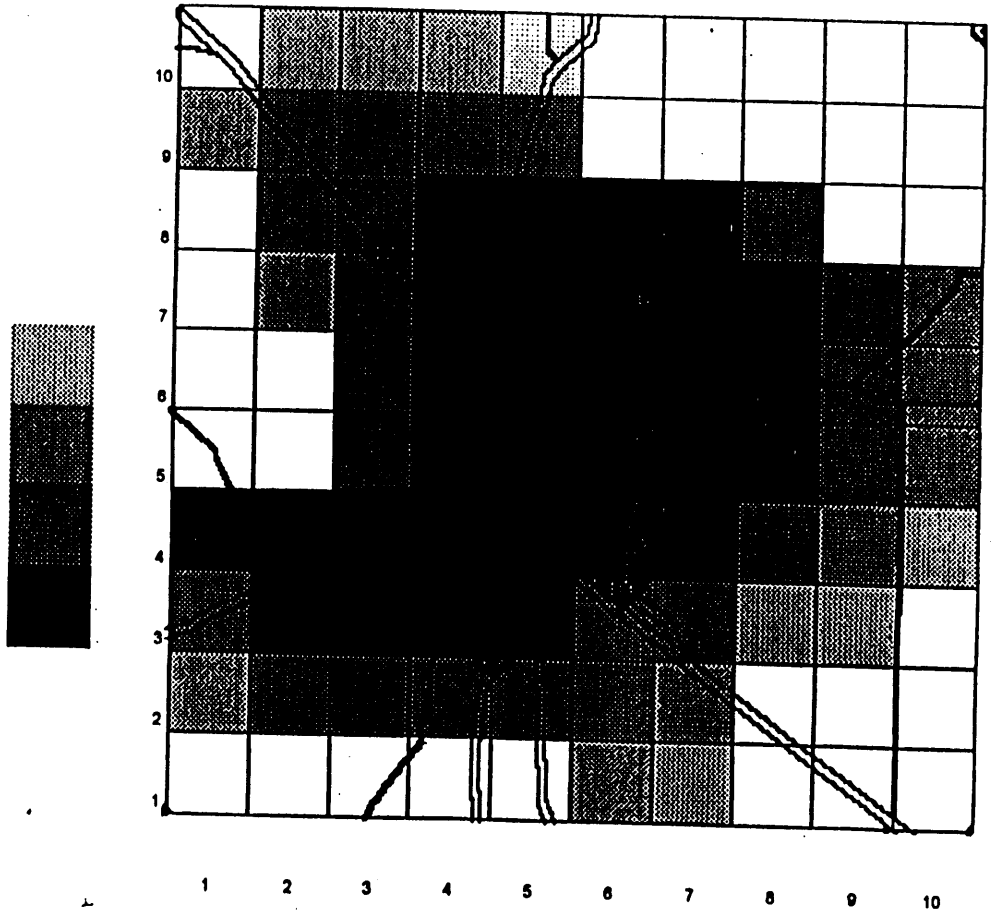
0.05-0.11

0.11-0.16

0.16-0.22

>0.22

Highest Cell = 0.27



TUCSON REGION

Toluene

Concentrations (ug/m3)

<1.9

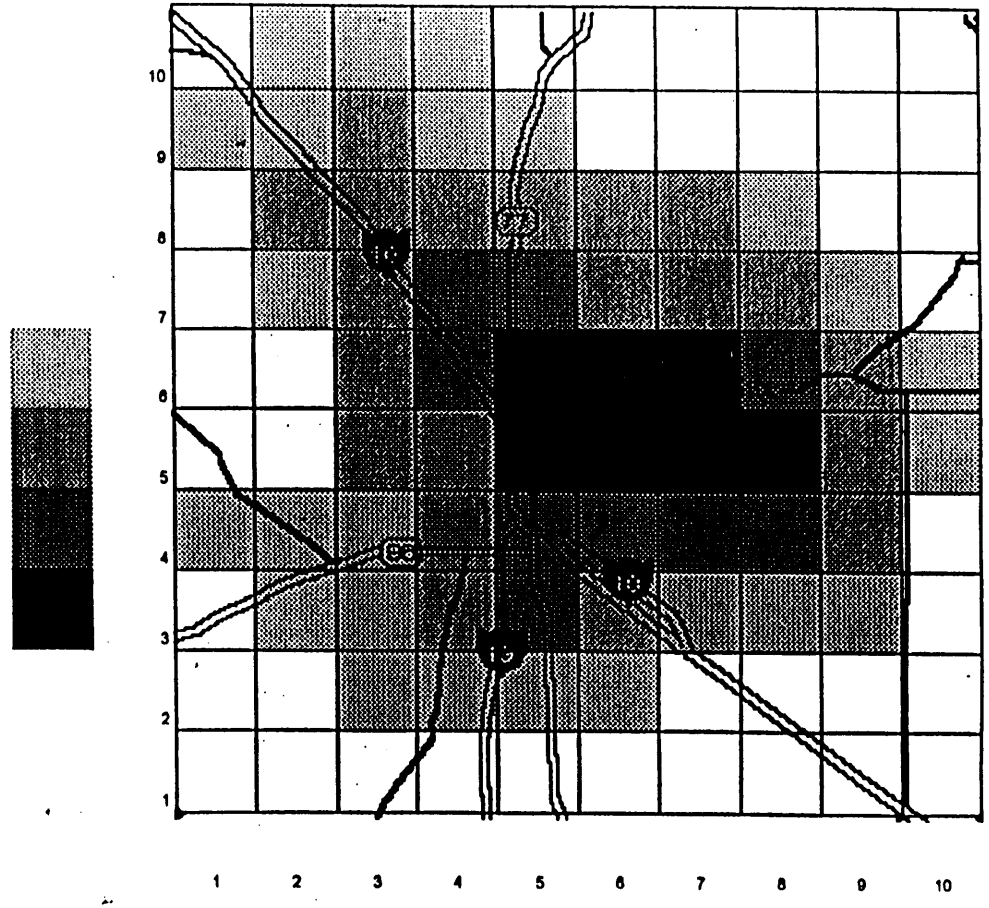
1.9-3.7

3.7-5.6

5.6-7.4

>7.4

Highest Cell = 9.7



TUCSON REGION

Tetrachloroethylene

Concentrations (ug/m3)

<0.18

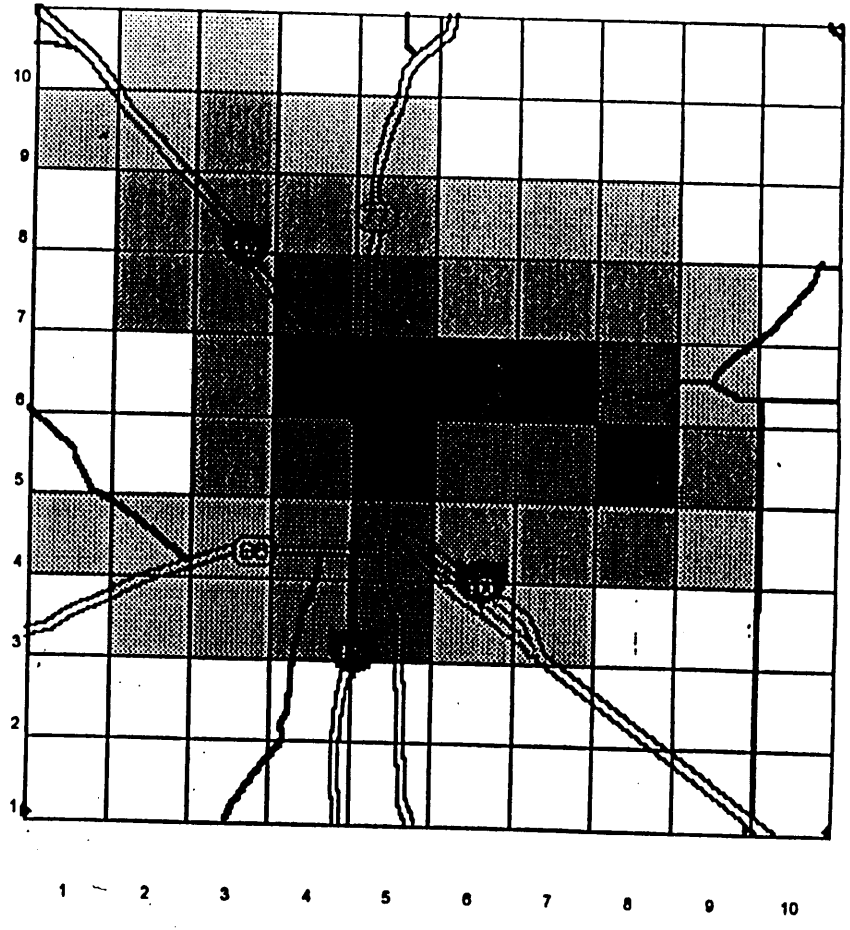
0.18-0.37

0.37-0.55

0.55-0.73

>0.73

Highest Cell = 0.97



TUCSON REGION

n-Hexane

Concentrations (ug/m3)

<1.3

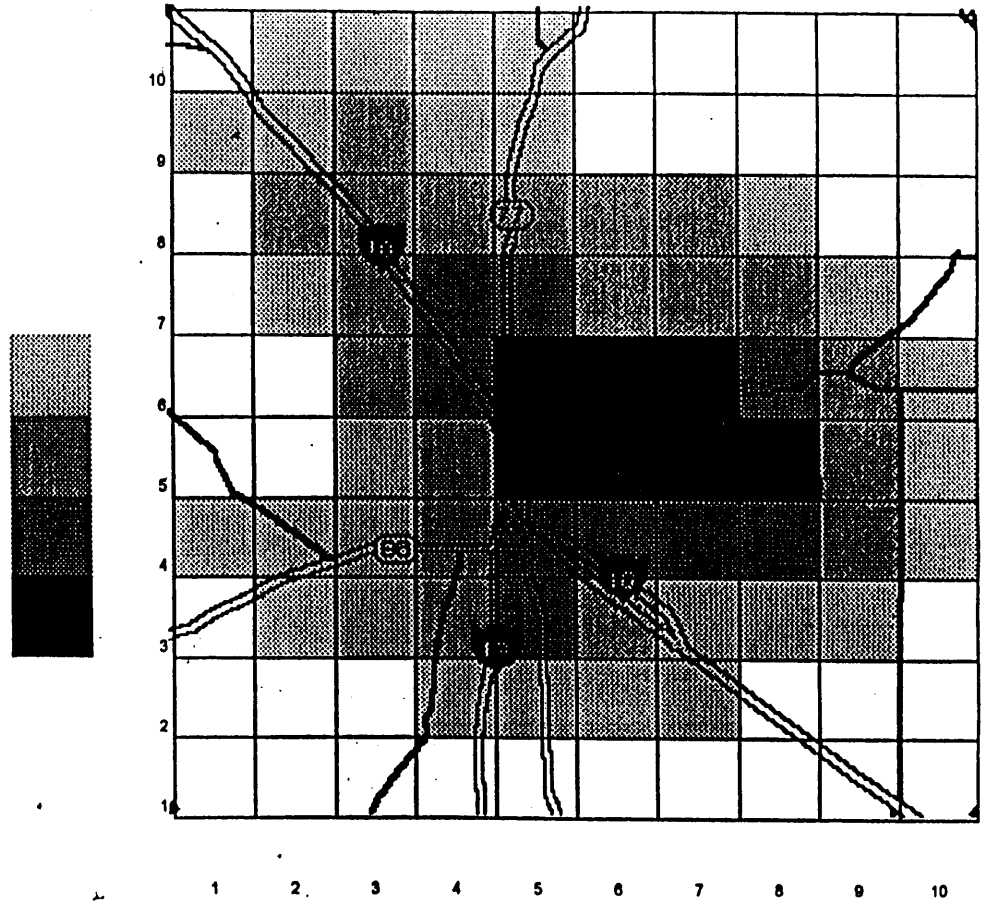
1.3-2.6

2.6-3.9

3.9-5.2

>5.2

Highest Cell = 6.9



TUCSON REGION

Trimethylbenzene

Concentrations (ug/m3)

<0.0047

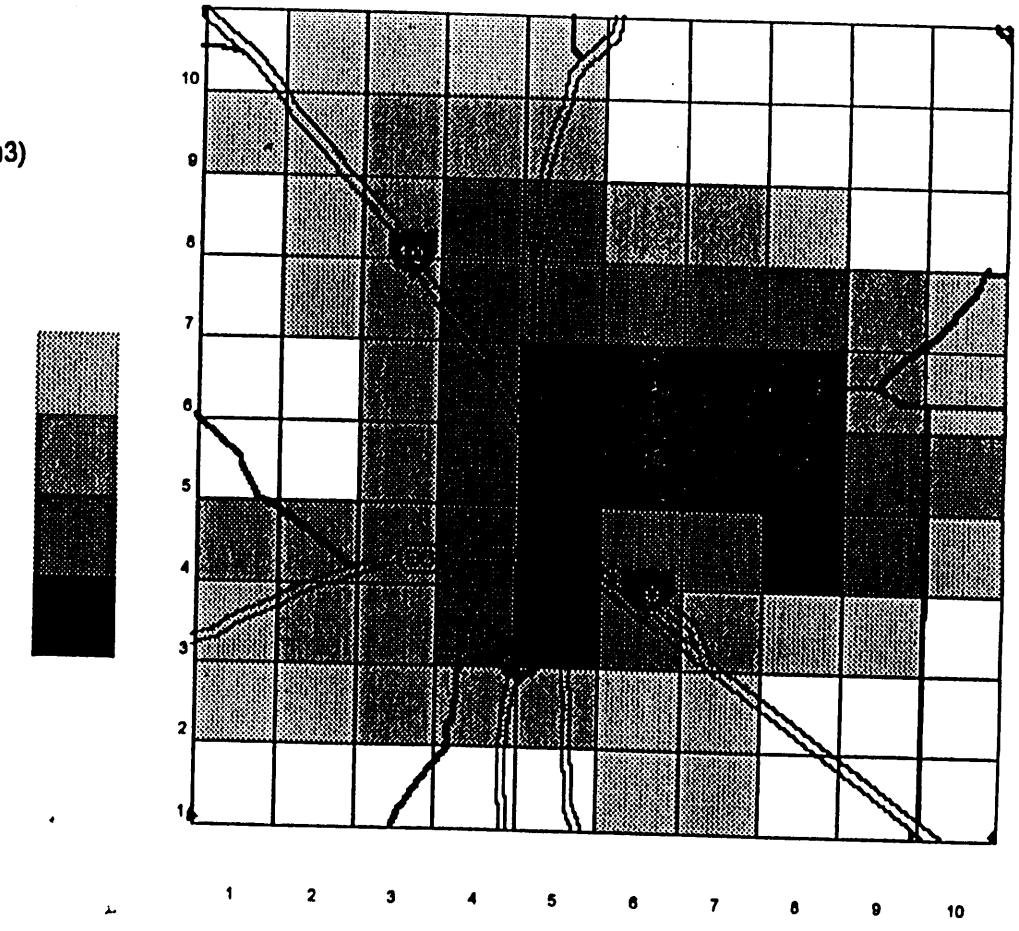
0.0047-0.0093

0.0093-0.0140

0.0140-0.0187

>0.0187

Highest Cell = 0.0244

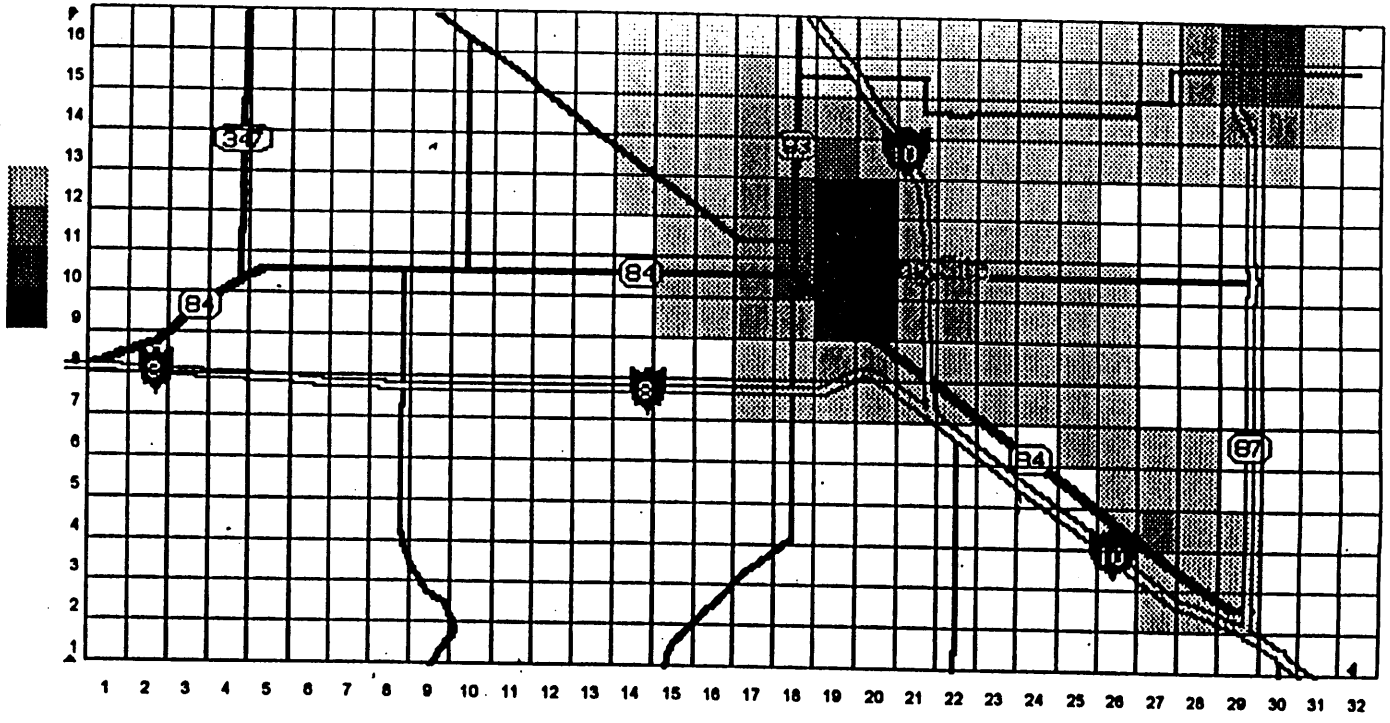


CASA GRANDE STUDY AREA

CASA GRANDE REGION

Acrolein
Concentrations (ug/m3)

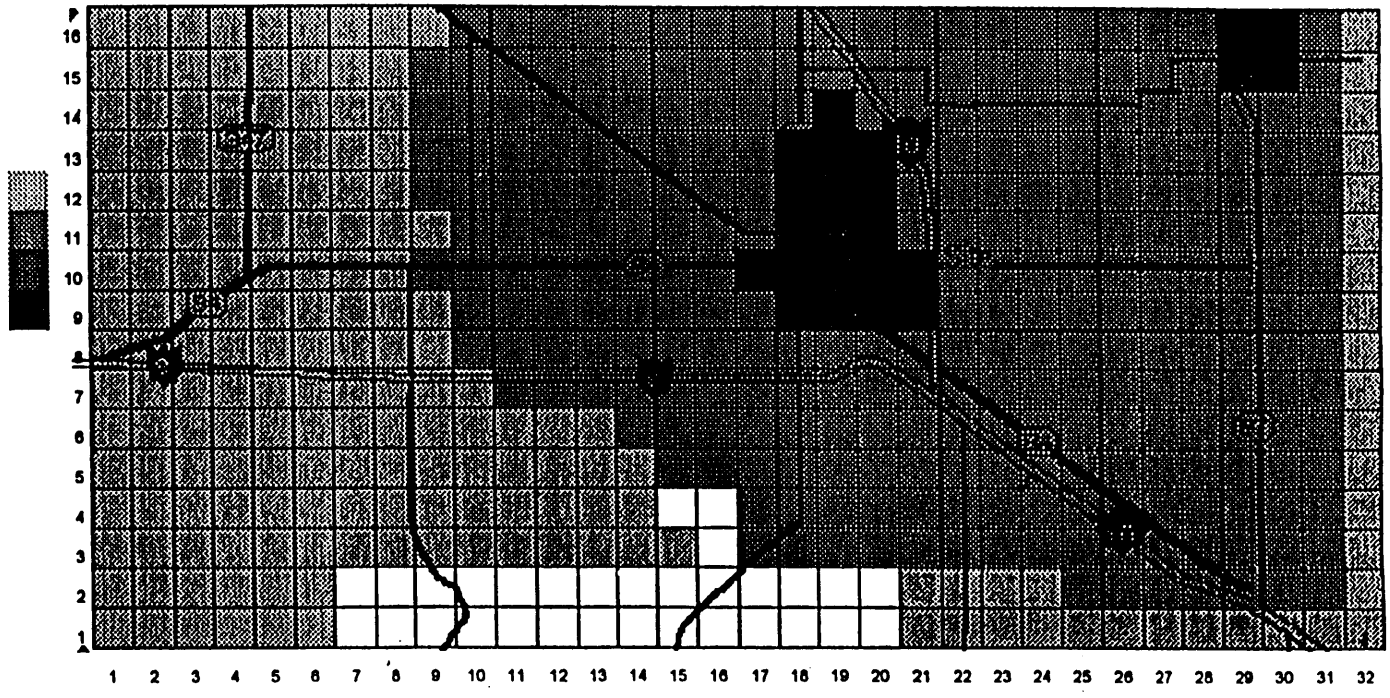
<0.005
0.005-0.010
0.010-0.015
0.015-0.021
>0.021
Highest Cell = 0.027



CASA GRANDE REGION

Acetaldehyde
Concentrations (ug/m3)

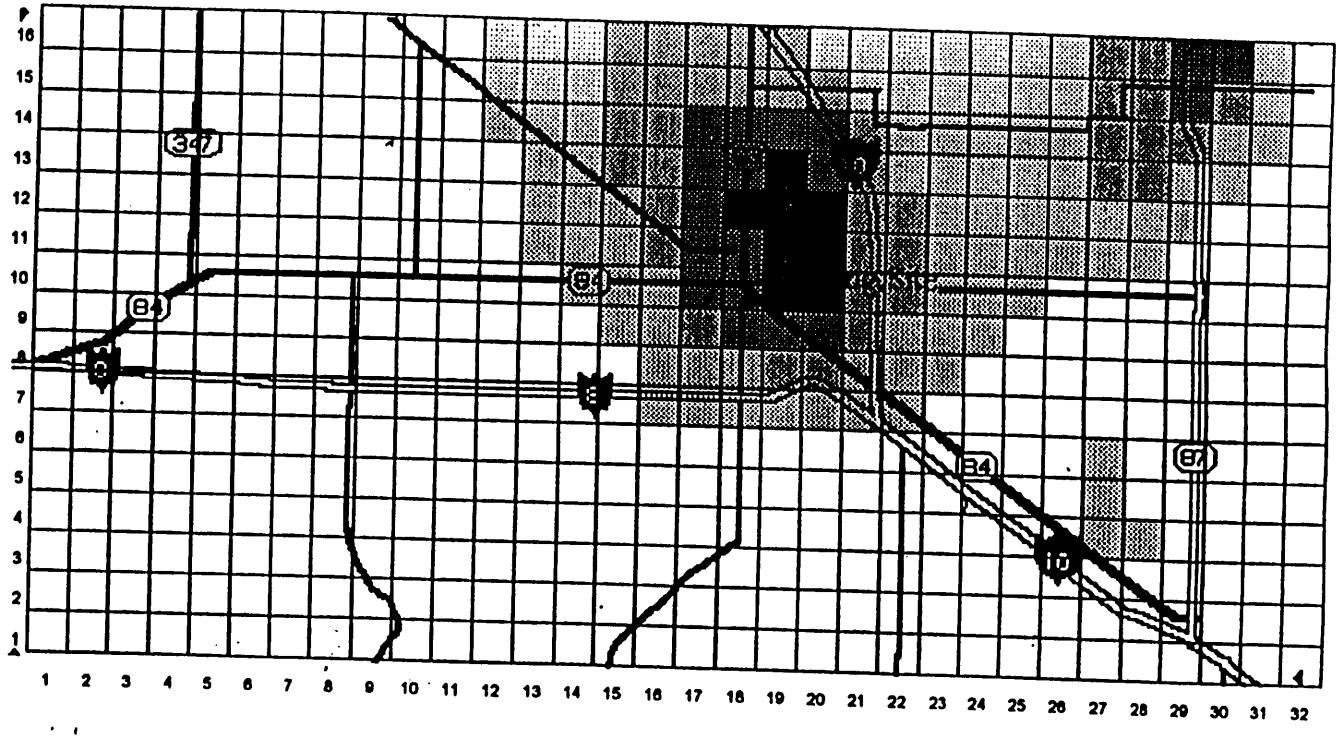
<0.5
0.5-1.0
1.0-1.6
1.6-2.1
>2.1
Highest Cell = 2.7



CASA GRANDE REGION

Arsenic
Concentrations (ug/m3)

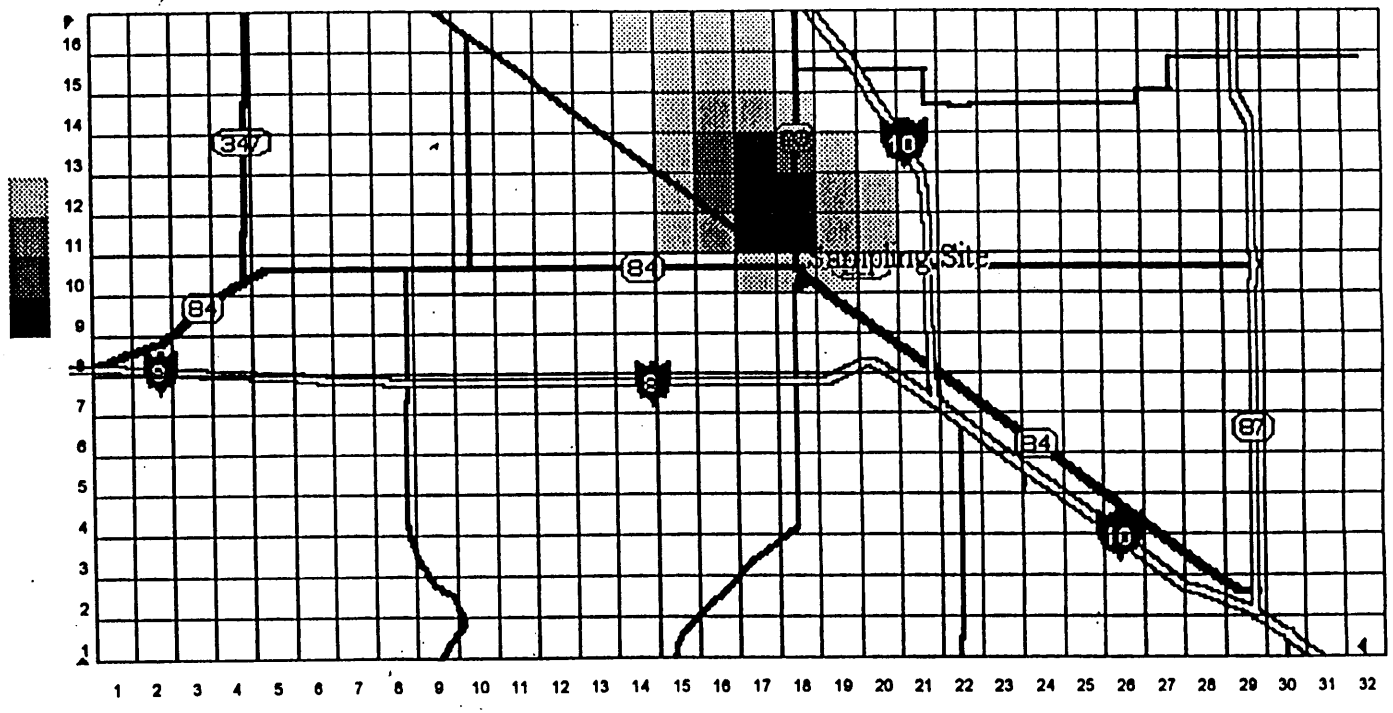
<0.00006
0.00006-0.00013
0.00013-0.00019
0.00019-0.00026
>0.00026
Highest Cell = 0.00032



CASA GRANDE REGION

Ammonia
Concentrations (ug/m3)

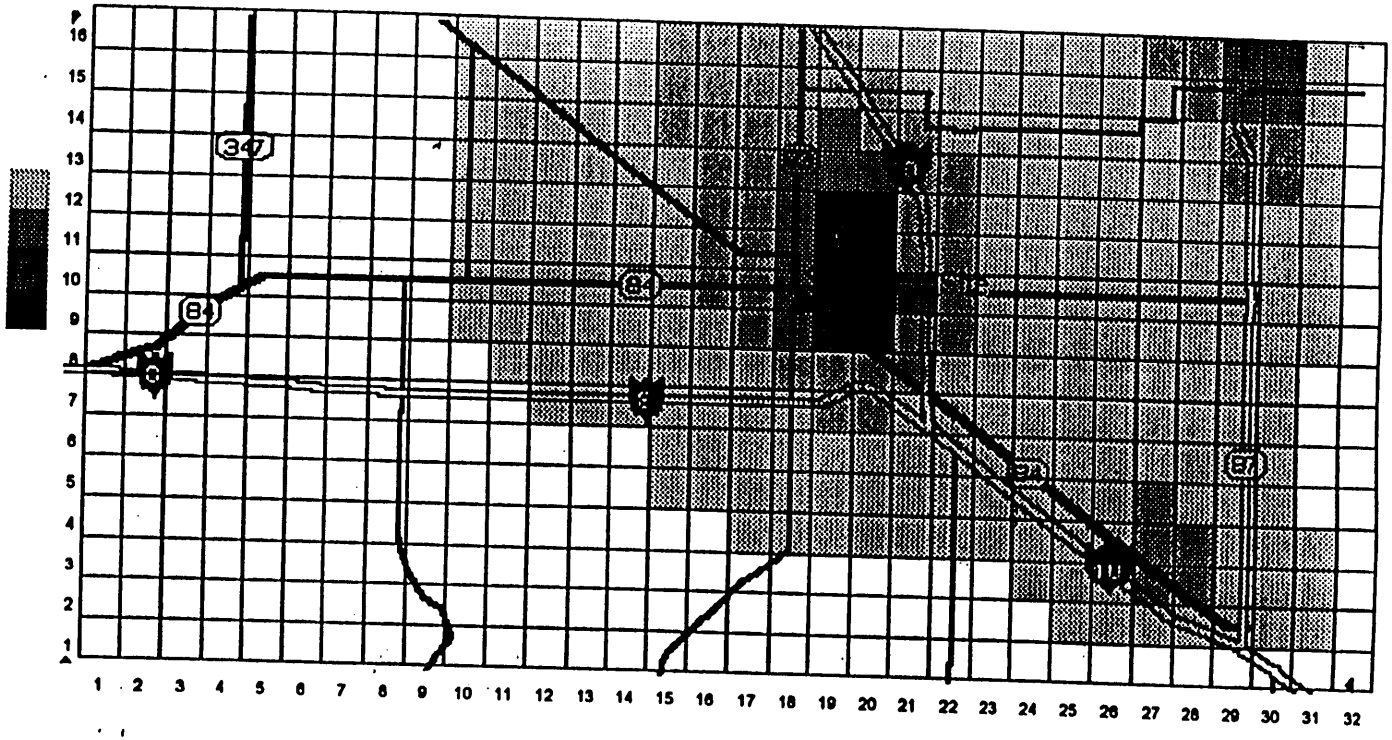
<1.3
1.3-2.7
2.7-4.0
4.0-5.3
>5.3
Highest Cell = 8.8



CASA GRANDE REGION

Benzene
Concentrations (ug/m3)

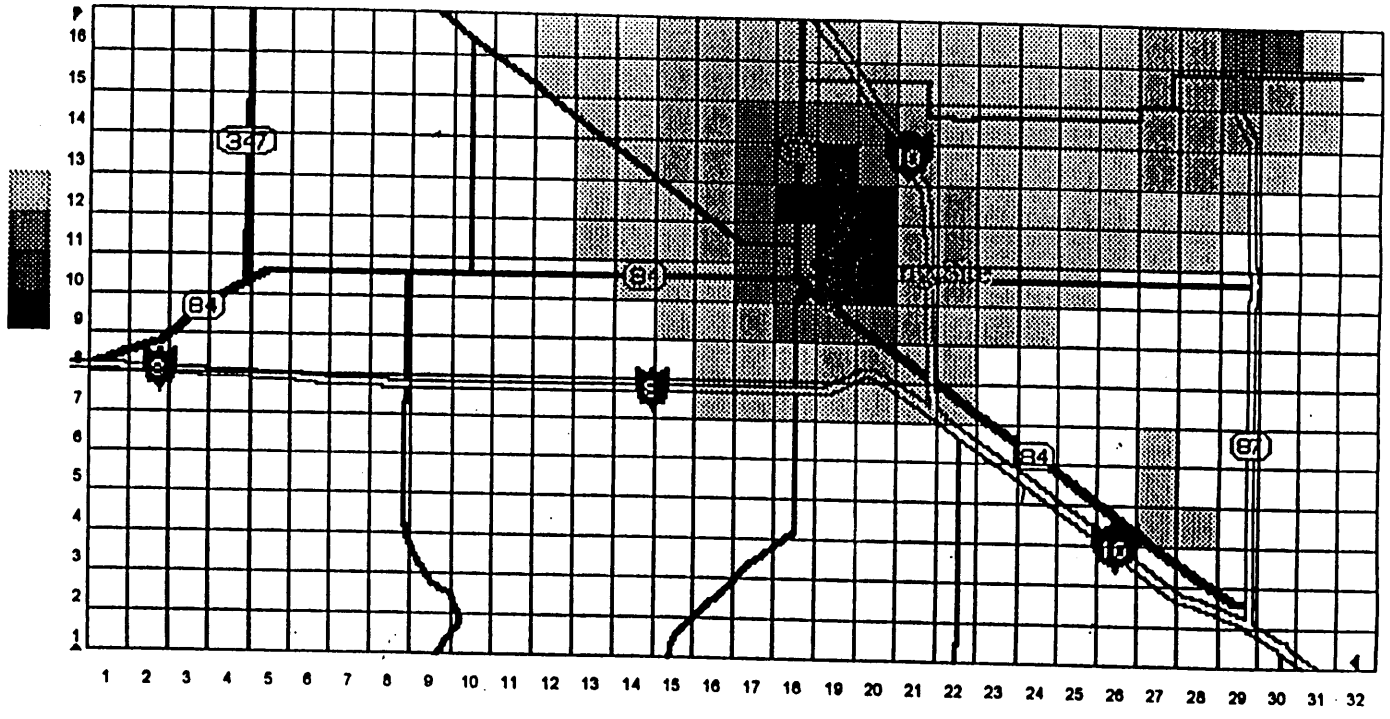
<0.43
0.43-0.87
0.87-1.30
1.30-1.73
>1.73
Highest Cell = 2.22



CASA GRANDE REGION

Barium
Concentrations (ug/m3)

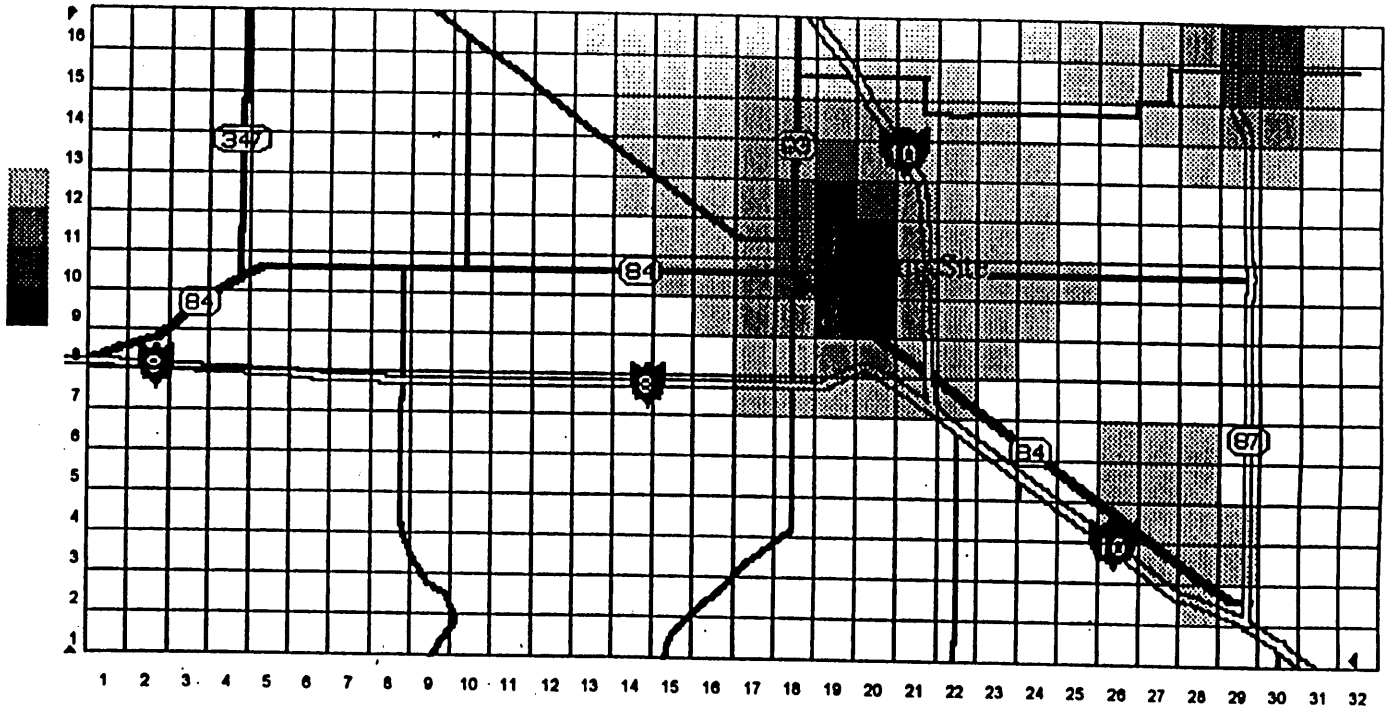
<0.00006
0.00006-0.00013
0.00013-0.00019
0.00019-0.00026
>0.00026
Highest Cell = 0.03695



CASA GRANDE REGION

1,3-Butadiene
Concentrations (ug/m3)

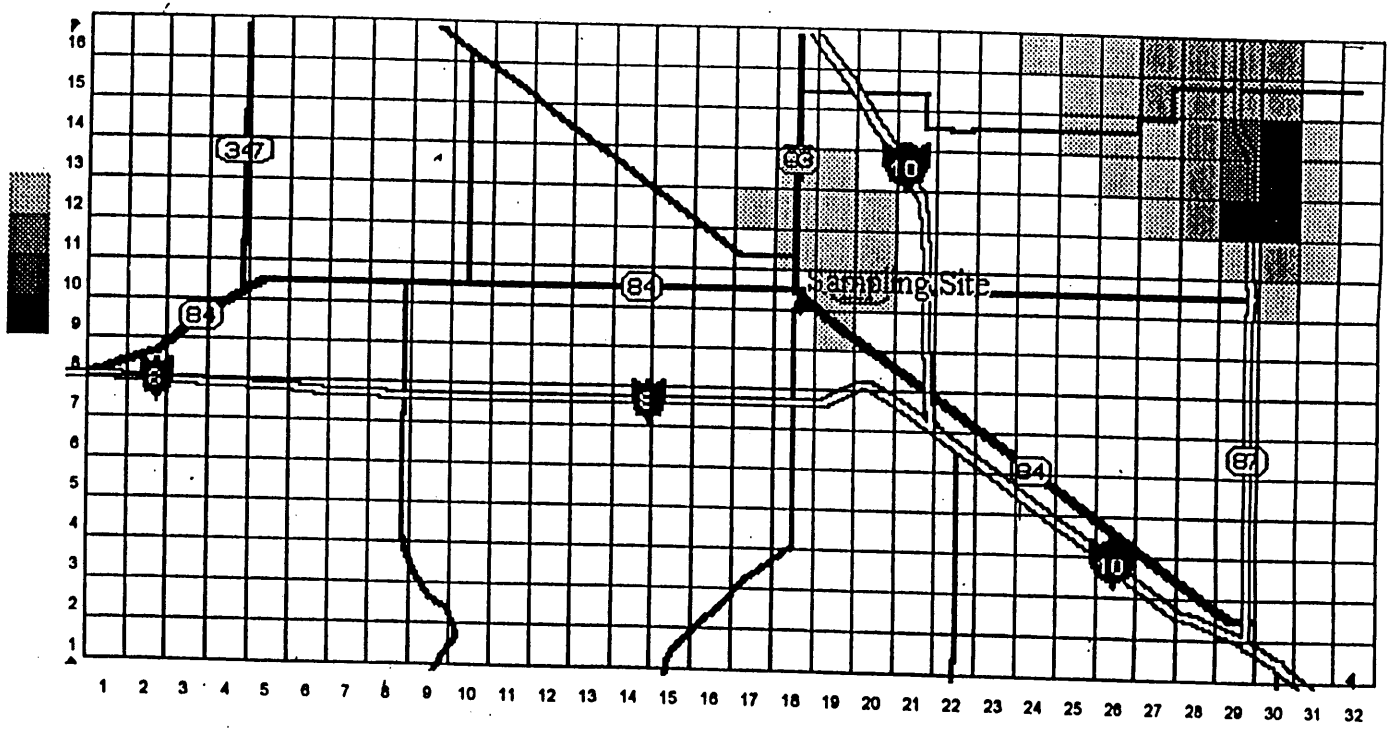
<0.05
0.05-0.11
0.11-0.16
0.16-0.22
>0.22
Highest Cell = 0.28



CASA GRANDE REGION

Benzo(a)pyrene
Concentrations (ug/m3)

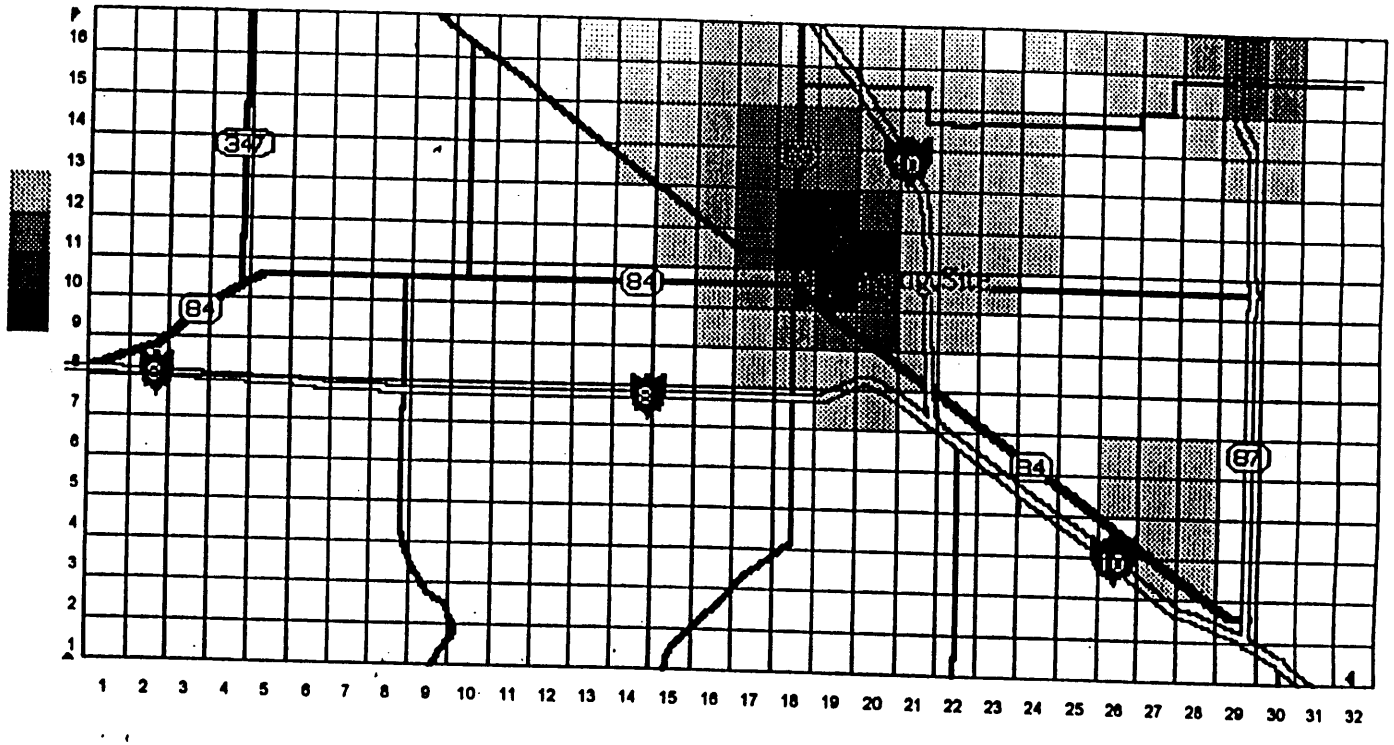
<0.00009
0.00009-0.00019
0.00019-0.00028
0.00028-0.00038
>0.00038
Highest Cell = 0.00053



CASA GRANDE REGION

Chloroform
Concentrations (ug/m3)

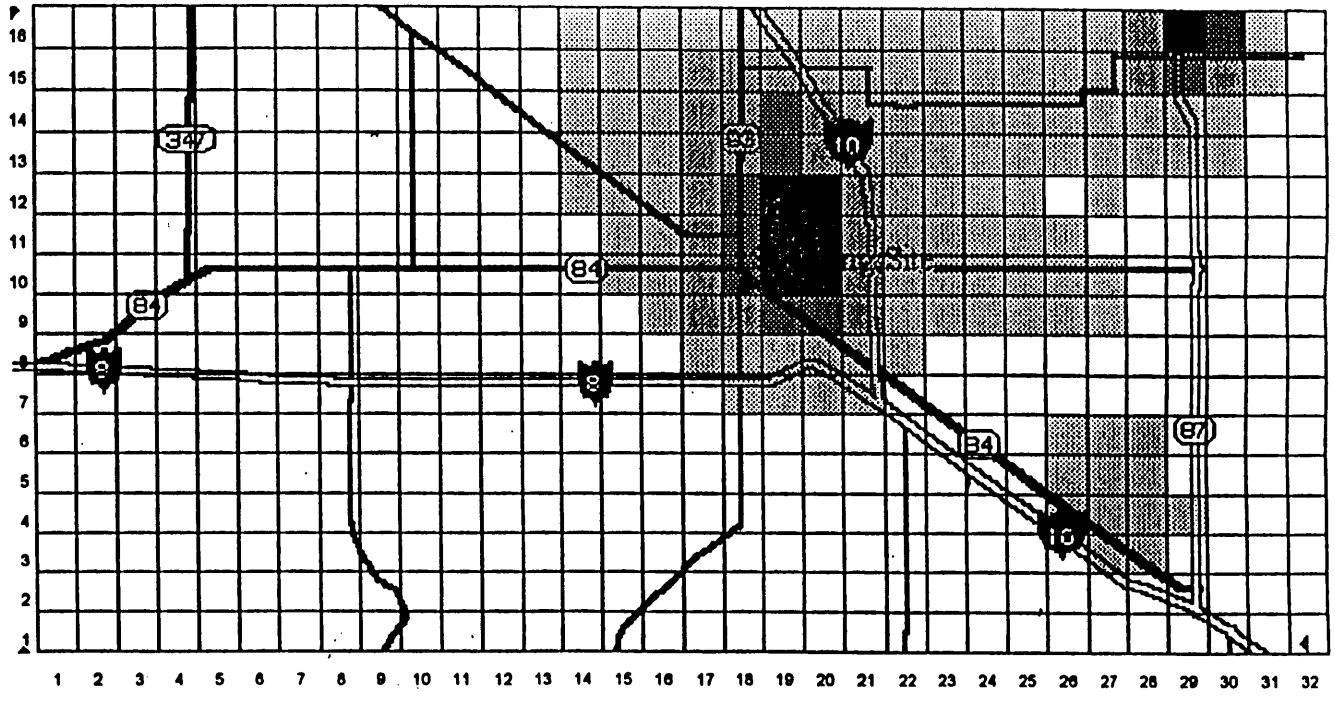
<0.04
0.04-0.09
0.09-0.13
0.13-0.18
>0.18
Highest Cell = 0.23



CASA GRANDE REGION

Cadmium
Concentrations (ug/m3)

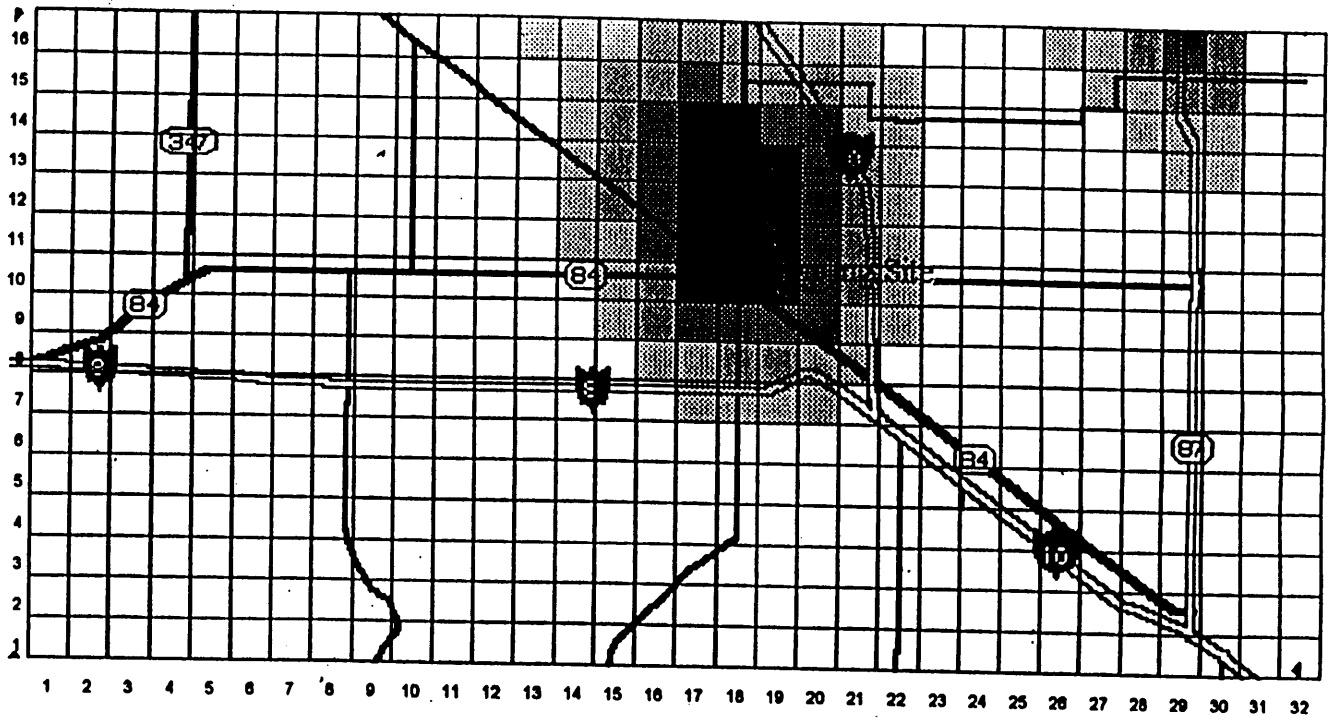
<0.00016
0.00016-0.00033
0.00033-0.00049
0.00049-0.00066
>0.00066
Highest Cell = 0.00085



CASA GRANDE REGION

Chromium (VI)
Concentrations (ug/m3)

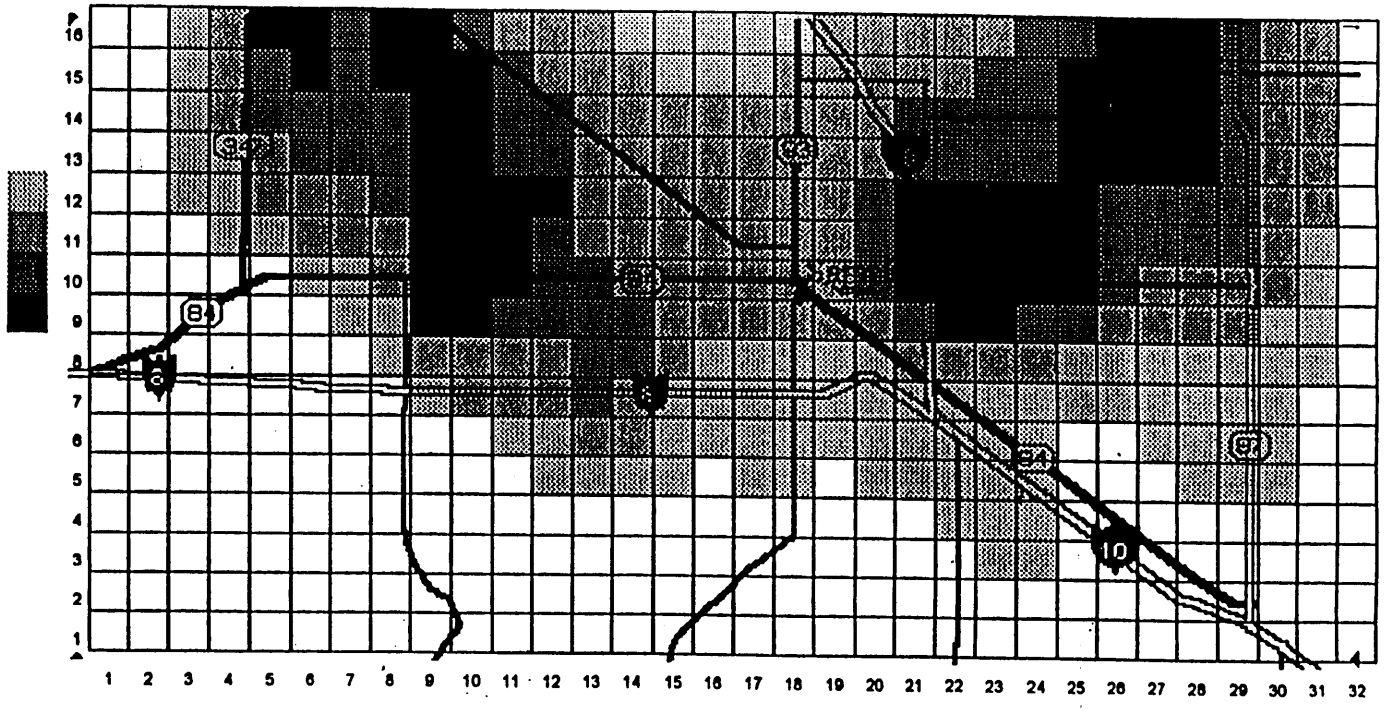
<0.00015
0.00015-0.00029
0.00029-0.00044
0.00044-0.00059
>0.00059
Highest Cell = 0.00075



CASA GRANDE REGION

Chlorpyrifos
Concentrations (ug/m3)

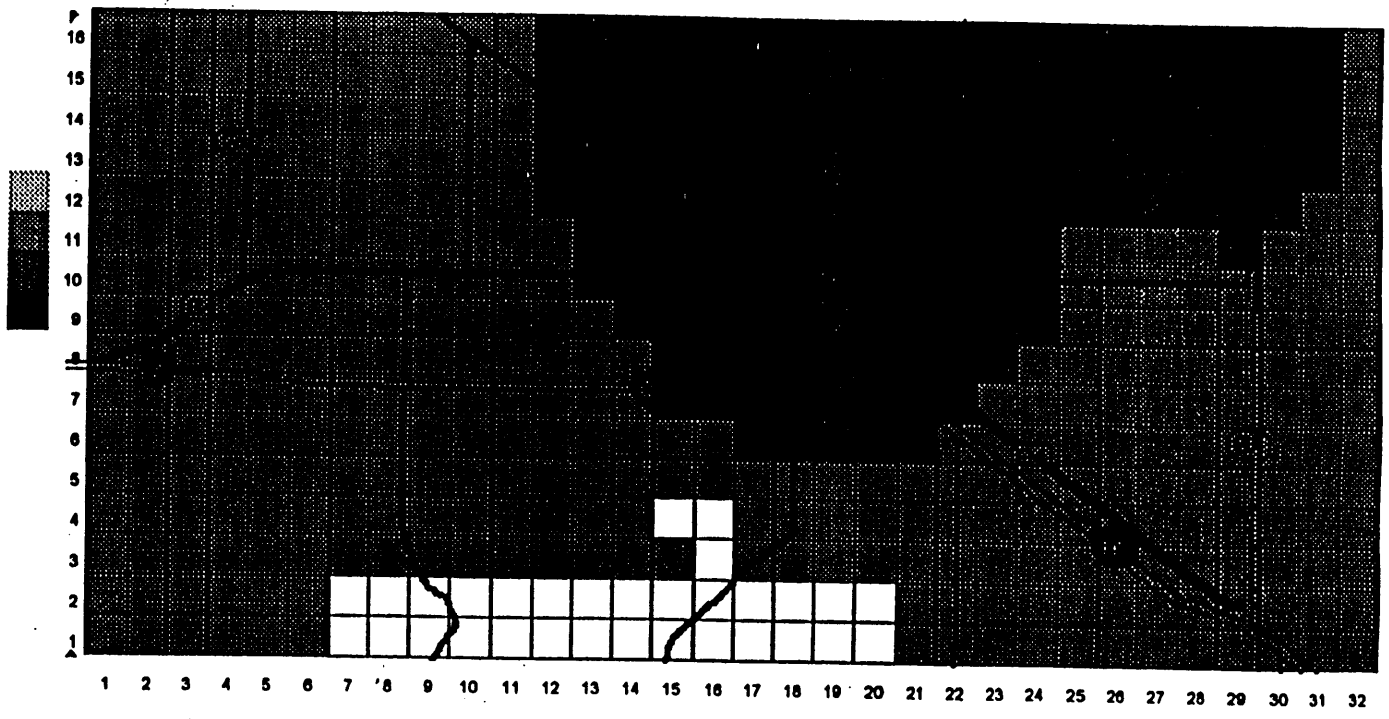
<0.014
0.014-0.029
0.029-0.043
0.043-0.057
>0.057
Highest Cell = 0.077



CASA GRANDE REGION

Dichloromethane
Concentrations (ug/m3)

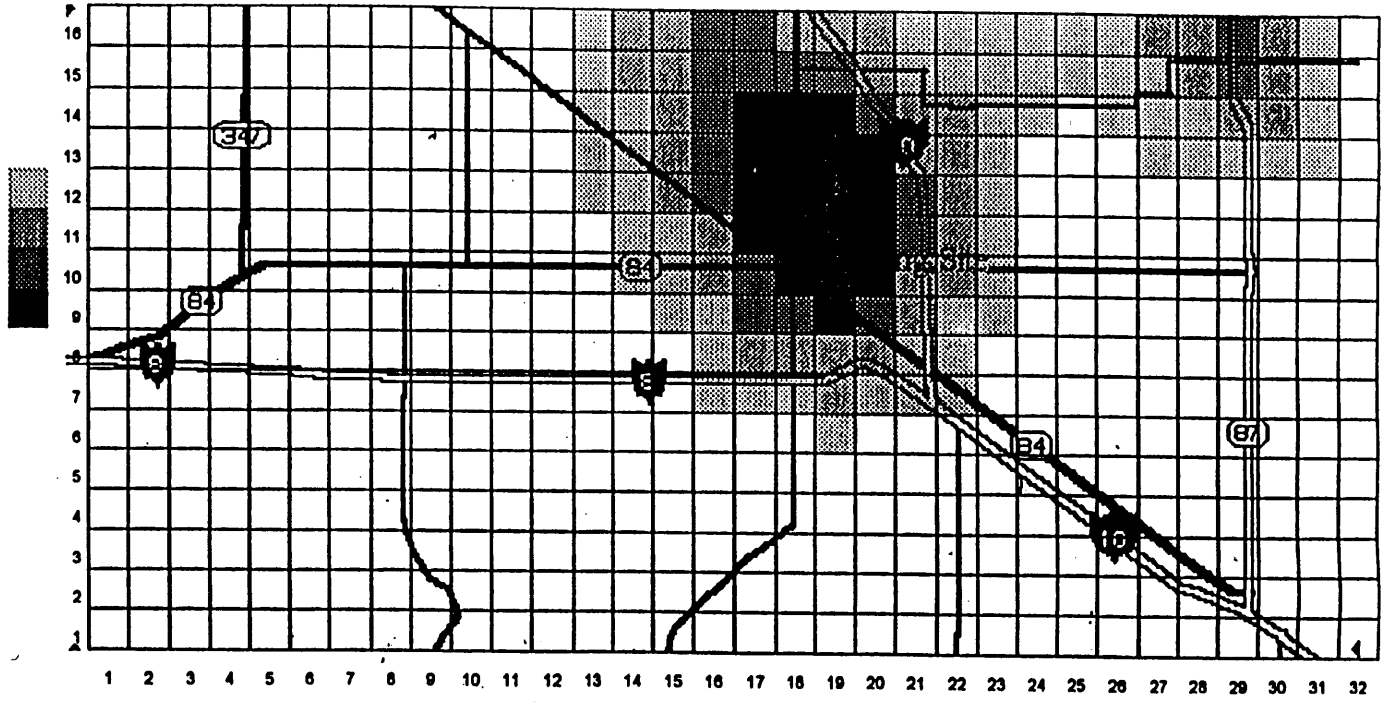
<0.65
0.65-1.31
1.31-1.96
1.96-2.61
>2.61
Highest Cell = 3.28



CASA GRANDE REGION

p-Dichlorobenzene
Concentrations (ug/m3)

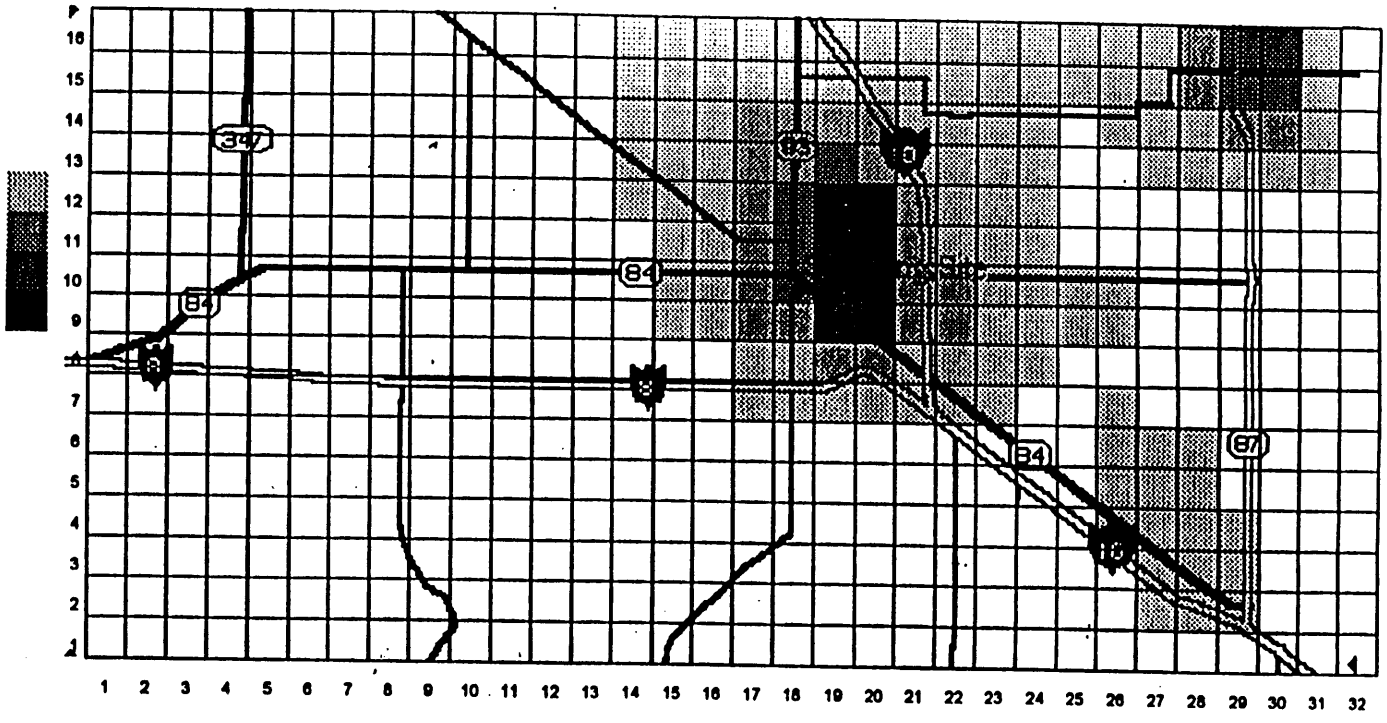
<0.014
0.014-0.028
0.028-0.042
0.042-0.056
>0.056
Highest Cell = 0.071



CASA GRANDE REGION

Formaldehyde
Concentrations (ug/m3)

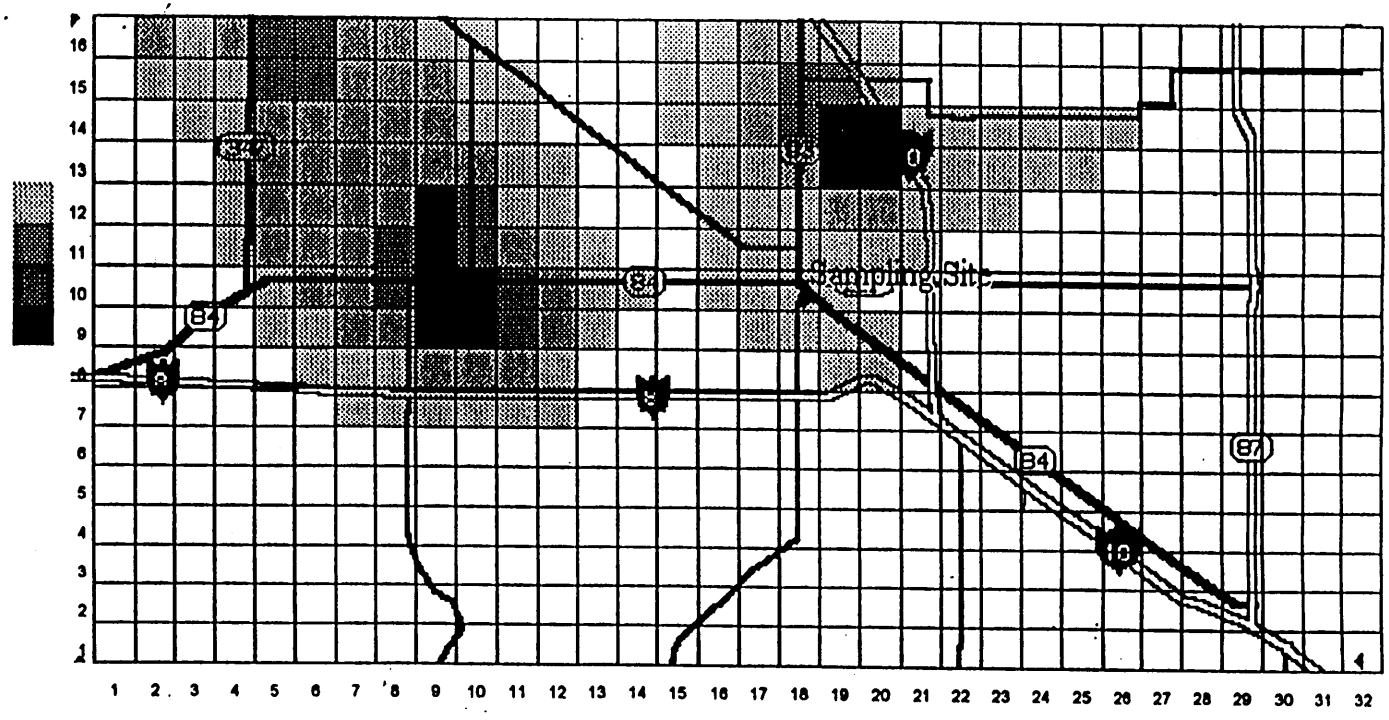
<0.24
0.24-0.48
0.48-0.72
0.72-0.96
>0.96
Highest Cell = 1.24



CASA GRANDE REGION

Disulfoton
Concentrations (ug/m3)

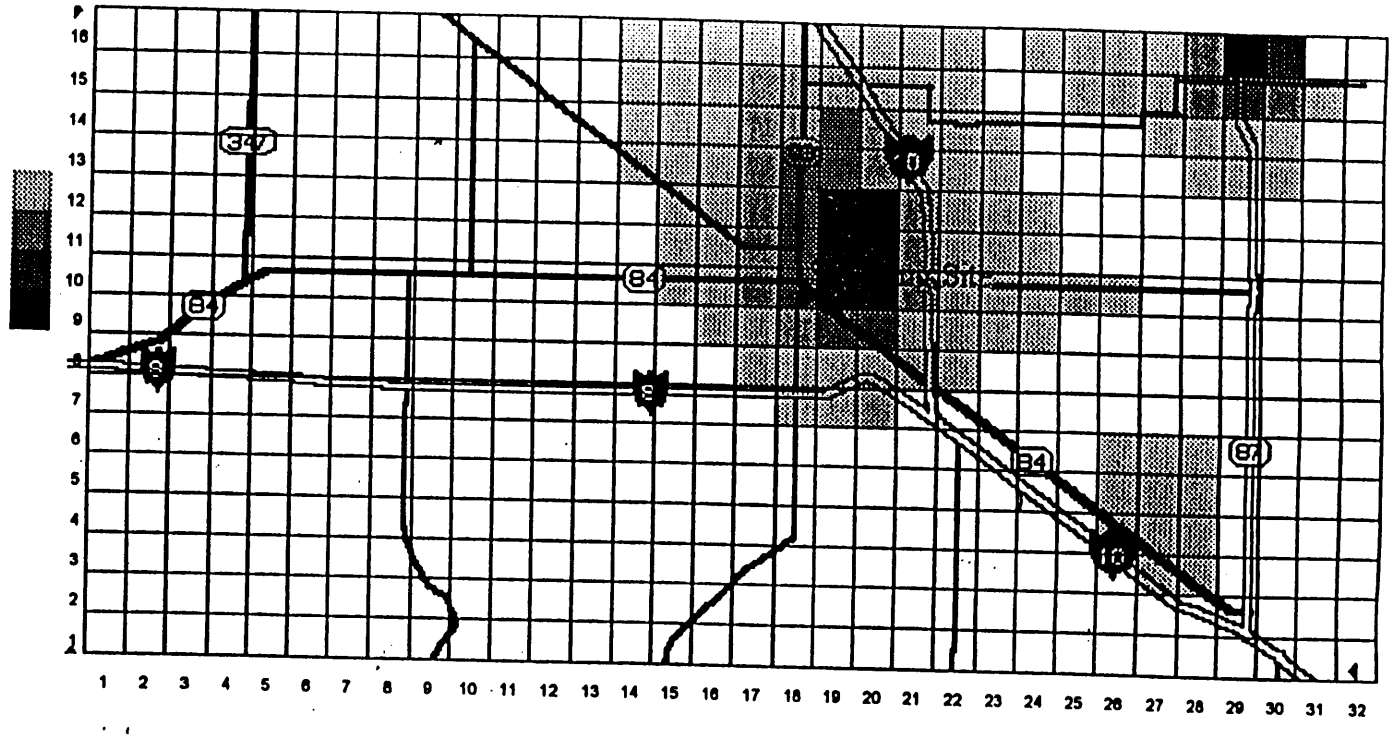
<0.0024
0.0024-0.0047
0.0047-0.0071
0.0071-0.0095
>0.0095
Highest Cell = 0.0147



CASA GRANDE REGION

Manganese
Concentrations (ug/m3)

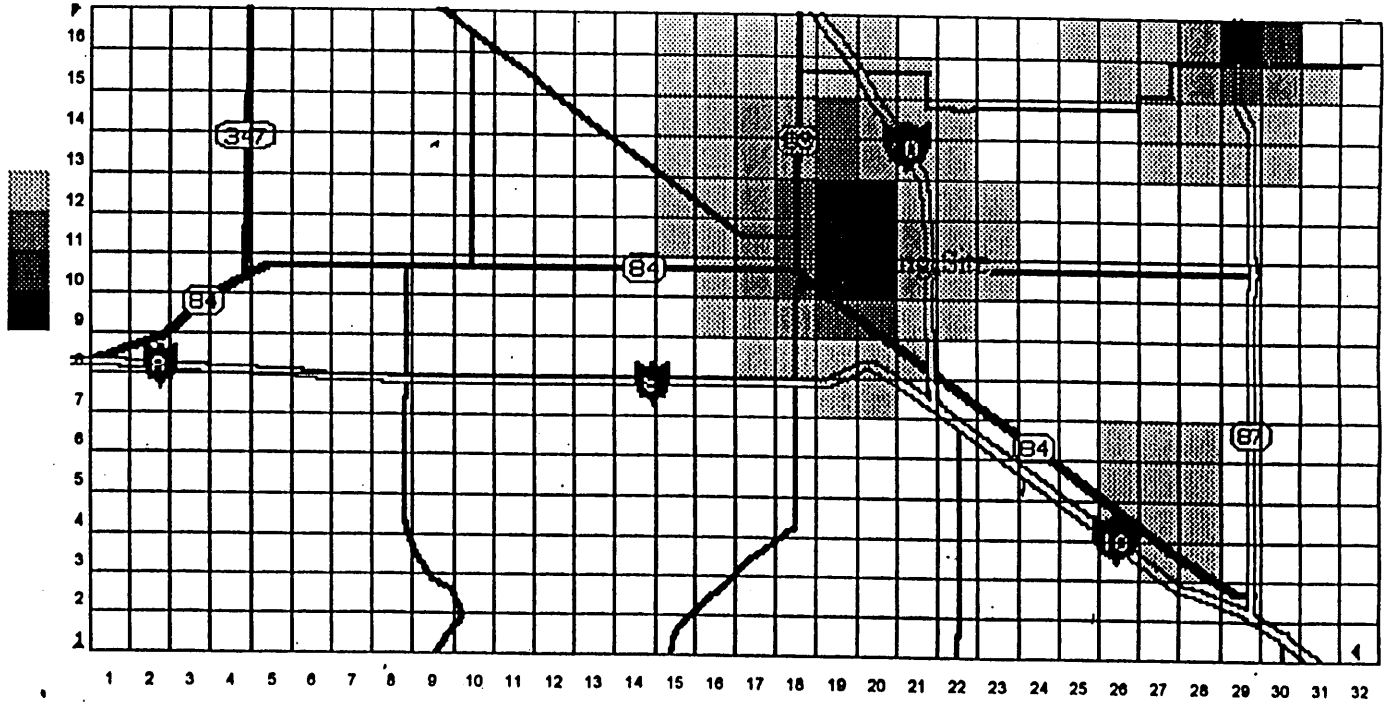
<0.006
0.006-0.012
0.012-0.018
0.018-0.025
>0.025
Highest Cell = 0.031



CASA GRANDE REGION

Lead
Concentrations (ug/m3)

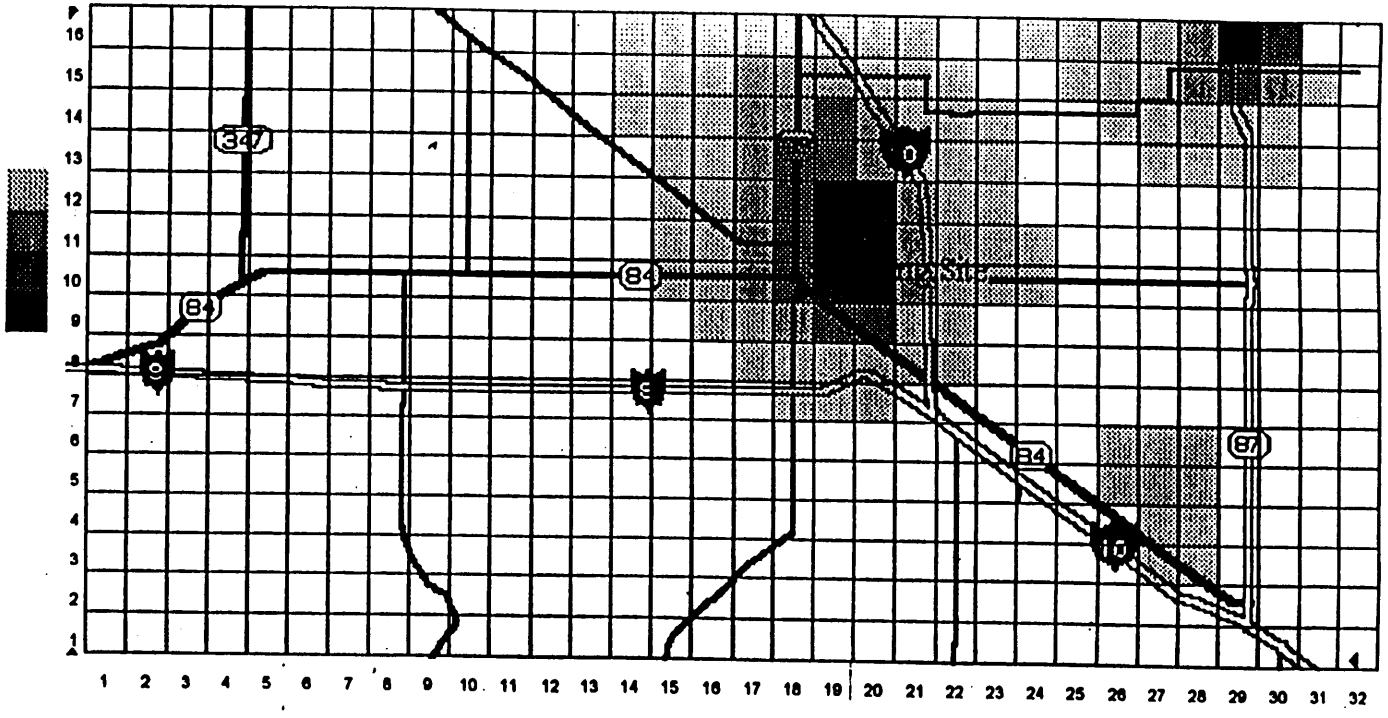
<0.0009
0.0009-0.0018
0.0018-0.0027
0.0027-0.0036
>0.0036
Highest Cell = 0.0046



CASA GRANDE REGION

Nickel
Concentrations (ug/m3)

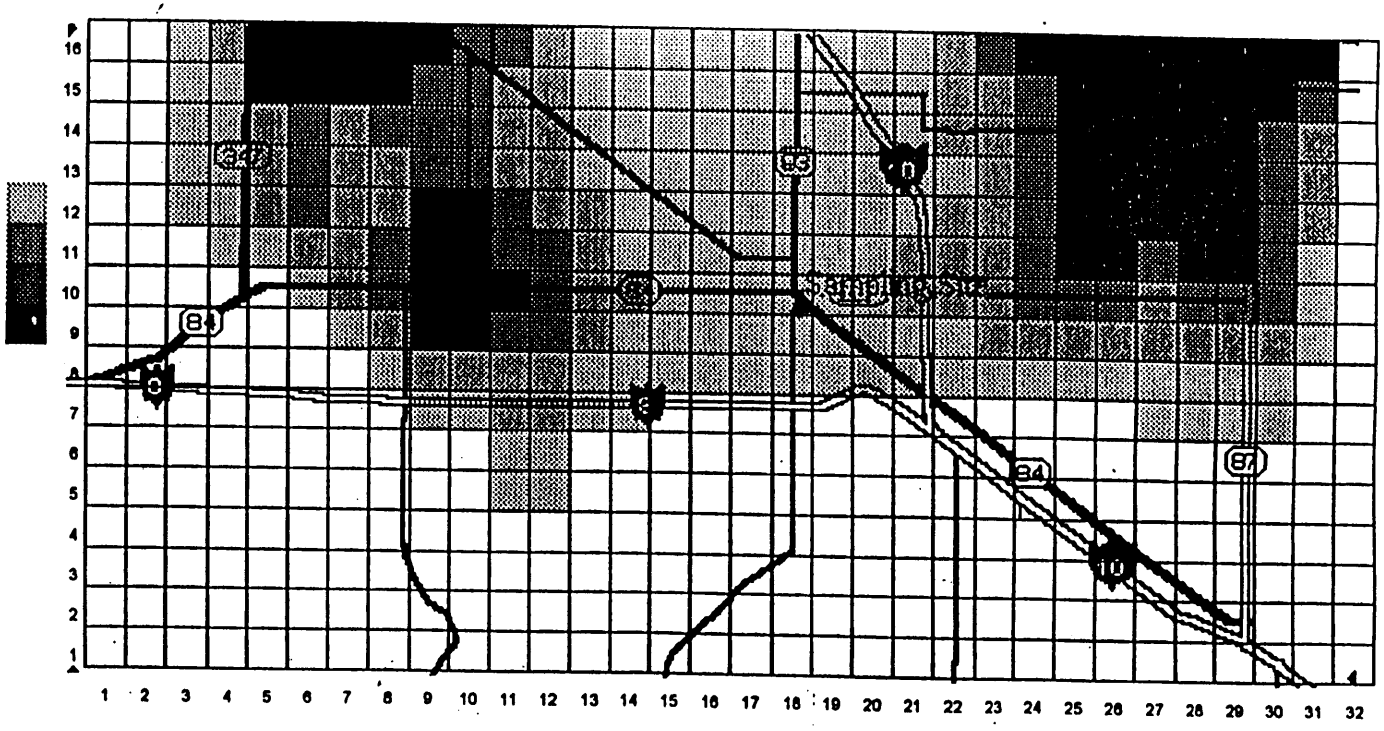
<0.0006
0.0006-0.0012
0.0012-0.0018
0.0018-0.0024
>0.0024
Highest Cell = 0.0030



CASA GRANDE REGION

Methyl Parathion
Concentrations (ug/m3)

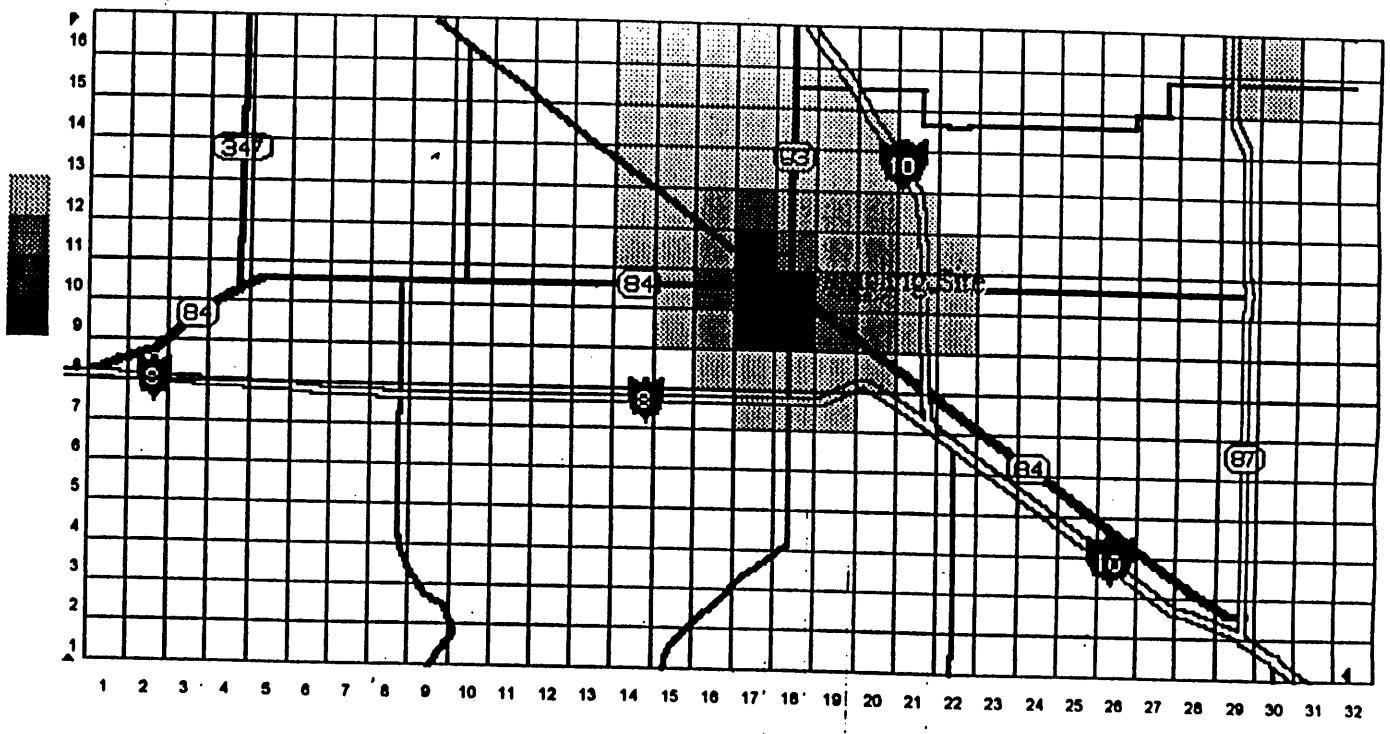
<0.015
0.015-0.030
0.030-0.045
0.045-0.060
>0.060
Highest Cell = 0.077



CASA GRANDE REGION

Toluene
Concentrations (ug/m3)

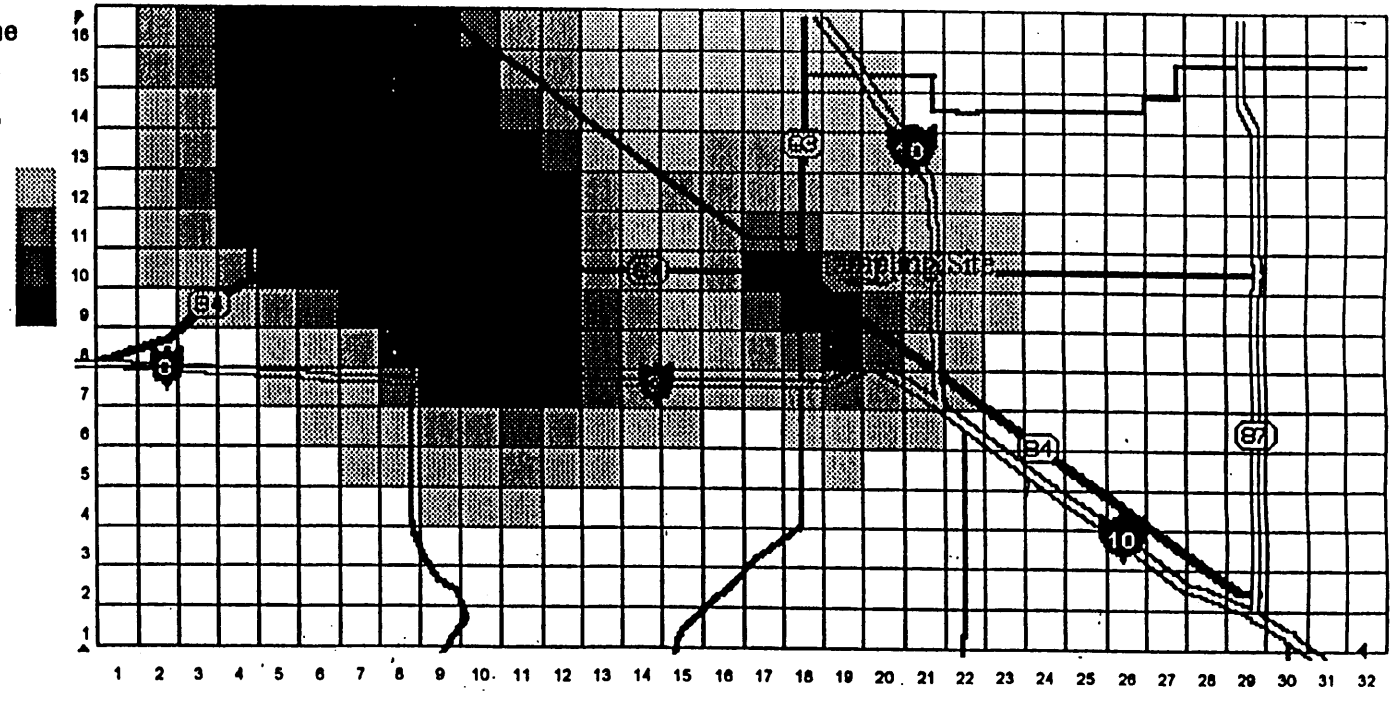
<2.5
2.5-5.1
5.1-7.6
7.6-10.2
>10.2
Highest Cell = 14.7



CASA GRANDE REGION

Pentachloronitrobenzene
Concentrations (ug/m3)

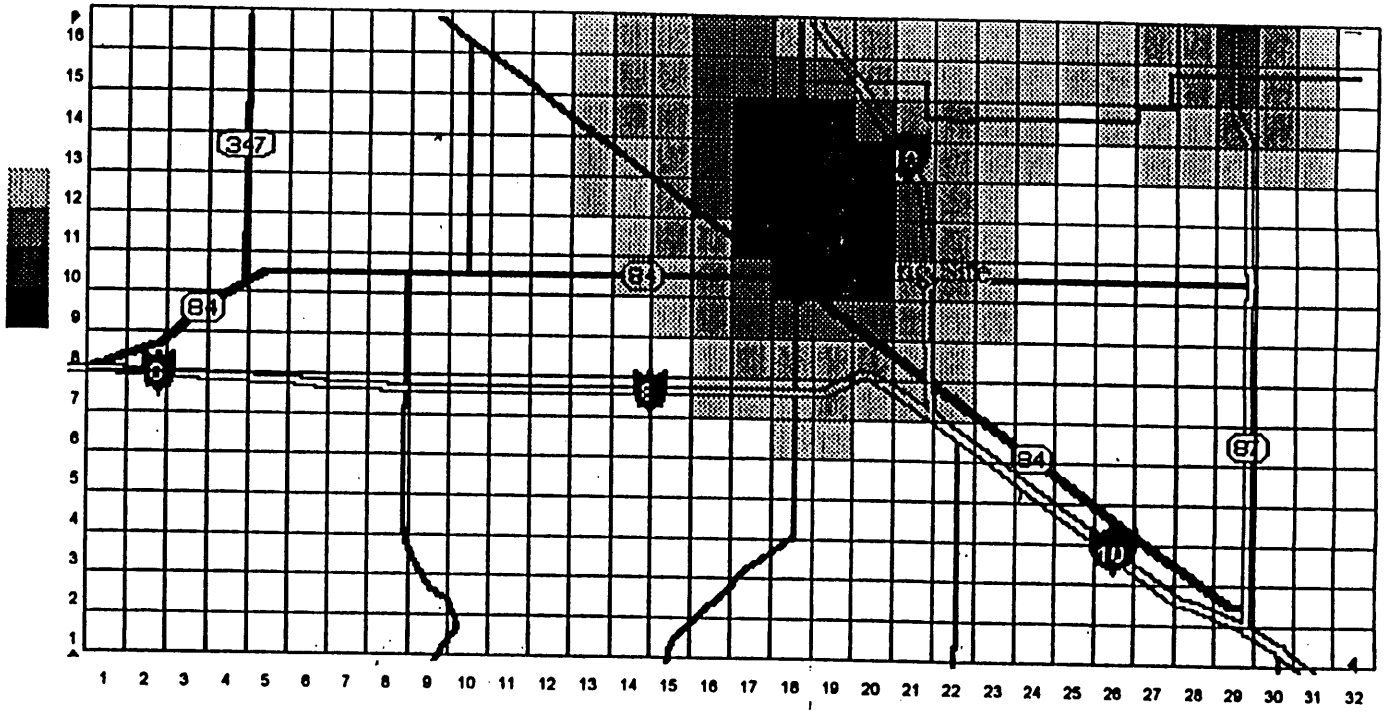
<0.0010
0.0010-0.0020
0.0020-0.0030
0.0030-0.0040
>0.0040
Highest Cell = 0.0052



CASA GRANDE REGION

Trichloroethylene
Concentrations (ug/m3)

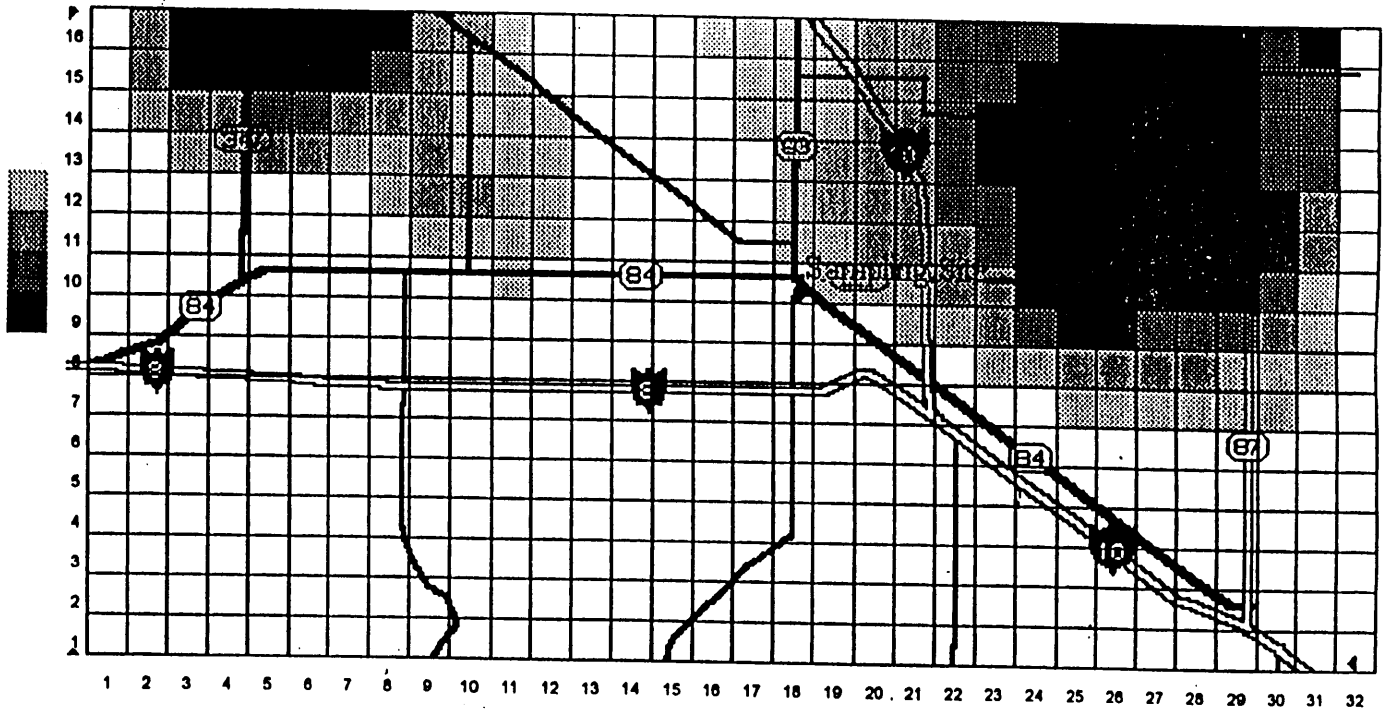
<0.17
0.17-0.33
0.33-0.50
0.50-0.67
>0.67
Highest Cell = 0.85



CASA GRANDE REGION

Trifluralin
Concentrations (ug/m3)

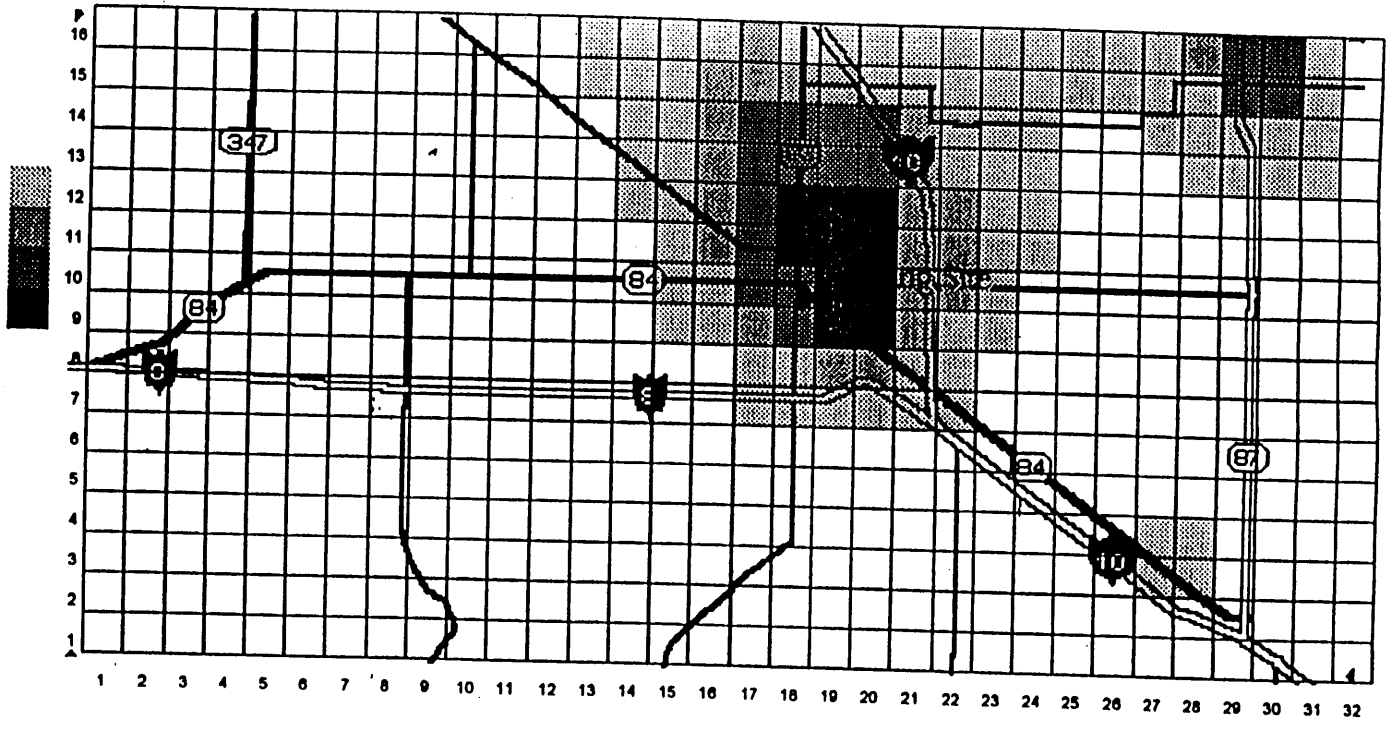
<0.004
0.004-0.007
0.007-0.011
0.011-0.015
>0.015
Highest Cell = 0.019



CASA GRANDE REGION

Trimethylbenzene
Concentrations (ug/m3)

<0.0009
0.0009-0.0018
0.0018-0.0027
0.0027-0.0036
>0.0036
Highest Cell = 0.0047



APPENDIX F

RISK ASSESSMENT

CONTENTS

1.0 INTRODUCTION 1-1

2.0 TOXICITY ASSESSMENT 2-1
- 2.1 Carcinogen Dose-Response 2-2
  - 2.1.1 Uncertainty in the Carcinogen Dose-Response Values 2-3
- 2.2 Noncarcinogen Dose-Response 2-8
  - 2.2.1 Uncertainty in Noncarcinogenic Dose-Response 2-9
- 2.3 Risk Assessment for Lead 2-12

3.0 EXPOSURE ASSESSMENT 3-1
- 3.1 Deterministic Exposure Assessment 3-1
  - 3.1.1 Probabilistic Exposure Assessment 3-3
- 3.2 Why Probabilistic Methods Are Useful 3-4
  - 3.2.1 Strengths of Probabilistic Methods 3-6
  - 3.2.2 Mechanics of Probabilistic Risk Assessment 3-7
  - 3.2.3 Computation Methods 3-8
- 3.3 Receptor-specific Exposure Parameter Distributions 3-9
  - 3.3.1 Body Weight 3-9
  - 3.3.2 Inhalation Rate 3-9
  - 3.3.3 Exposure Time 3-9
  - 3.3.4 Exposure Frequency 3-10
  - 3.3.5 Exposure Duration 3-10
  - 3.3.6 Averaging Period 3-10
- 3.4 Chemical-specific Exposure Parameters 3-10
  - 3.4.1 Air Concentration 3-11
- 3.5 Multi-Pathway Evaluation 3-13

4.0 RISK CHARACTERIZATION 4-1
- 4.1 Phoenix Study Region 4-2
- 4.2 Tucson Study Region 4-7
- 4.3 Casa Grande Study Region 4-20
- 4.4 Payson Study Region 4-37
- 4.5 Summary 4-40

CONTENTS
(Cont'd)

ATTACHMENTS

F-1 Dose Response Profiles for Elements, VOC, PAH, Pesticides plus ECAO risk Assessment Issue Paper

F-2 Probability Distributions

LIST OF TABLES

2-1 Dose-Response Information for Compounds with Potential Carcinogenic Effects - All Areas 2-4
2-2 Relative Potency Factors for Potentially Carcinogenic Polycyclic Aromat All Areas . . 2-7
2-3 Dose Response Information for Compounds with Potential Noncarcinogenic Effects from Chronic Exposure Through the Inhalation Route - All Areas 2-10
2-4 Summary of Toxicity Endpoints for Compounds with Potential Noncarcinogenic Effects - All Areas 2-11
3-1 Summary of Point Estimate Exposure Assumptions - All Areas 3-2
3-2 Ambient Air Concentrations - CTC and RME Scenario - Phoenix 3-14
3-3 Ambient Air Concentrations - CTC and RME Scenario - Tucson 3-15
3-4 Ambient Air Concentrations - CTC and RME Scenario - Casa Grande 3-16
3-5 Annual Average Air Concentrations Arizona HAPs, Payson, Air-Pathway 3-17
3-6 Evaluation of Multipathway Exposures to Particulate HAPS - Phoenix 3-18
4-1 Summary of Total Estimated Incremental Carcinogenic Risk and Total Estimated Noncarcinogenic Hazard Index for the REM Scenario - Phoenix 4-3
4-2 Summary of Total Estimated Incremental Carcinogenic Risk and Total Estimated NonCarcinogenic Hazard Index for the CTC Scenario - Phoenix 4-4
4-3 Toxicity Endpoints Analysis for NonCarcinogenic Effects of COI Due to Inhalation Young Child Resident - CTC Scenario - Phoenix 4-5
4-4 Toxicity Endpoints Analysis for Noncarcinogenic Effects of COI Due to Inhalation Young Child Resident - CTC Scenario - Phoenix 4-6
4-5 Arizona HAPs, Phoenix, Air Assessment Integrated Exposure Uptake Biokinetic Model for Lead in Children - Tucson 4-8

CONTENTS
(Cont'd)

4-7 Summary of Total Future Incremental Carcinogenic Risk and Total Future Noncarcinogenic Hazard Index for the RME Scenario - Tucson 4-12
4-8 Summary of Total Future Incremental Carcinogenic Risk and Total Future Noncarcinogenic Hazard Index for the CTC Scenario - Tucson 4-13
4-9 Summary of total Estimated Incremental Carcinogenic Risk and Total Estimated Noncarcinogenic Hazard Index for the RME Scenario - Tucson 4-14
4-10 Summary of Total Estimated Incremental Carcinogenic Risk and Total Estimated Noncarcinogenic Hazard Index for the CTC Scenario - Tucson 4-16
4-11 Toxicity Endpoints Analysis for Noncarcinogenic Effects of COI Due to Inhalation Young Child Resident RME Scenario - Tucson 4-17
4-12 Toxicity Endpoints Analysis for Noncarcinogenic Effects of COI Due to Inhalation Young Child Resident CTC Scenario - Tucson 4-18
4-13 Arizona HAPs, Tuscon, Air Assessment Integrated Exposure Uptake Biokinetic Model for Lead in Children - Tucson 4-19
4-14 Comparison of Deterministic and Probablistic Results for Estimated Carcinogenic Risk and Noncarcinogenic Hazard Index - Tucson 4-21
4-15 Summary of Total Future Incremental Carcinogenic Risk and Total Future Noncarcinogenic Hazard Index for the RME Scenario 4-24
4-16 Summary of Total Future Incremental Carcinogenic Risk and Total Noncarcinogenic Hazard Index for the CTC Scenario - Tuscon 4-25
4-17 Summary of Total Estimated Incremental Carcinogenic Risk and Total Estimated Noncarcinogenic Hazard Infex for the RME Scenario 4-26
4-18 Summary of Total Estimated Incremental Carcinogenic Risk and Total Estimated Noncarcinogenic Hazard Index for the CTC Scenario - Casa Grande 4-28
4-19 Toxicity Endpoints Analysis for Noncarcinogenic Effects of COI Due to Inhalation Young Child Resident RME Scenario - Casa Grande 4-29
4-20 Toxicity Endpoints Analysis for Noncarcinogenic Effects of COI Due to Inhalation Young Child Resident CTC Scenario - Casa Grande 4-30
4-21 Arizona HAPs, Casa Grande, Air Assessment Integrated Exposure Uptake Biokinetic Model for Lead in Children - Casa Grande 4-31
4-22 Comparison of Deterministic and Probablistic Results for Estimated Carcinogenic Risk and Noncarcinogenic Hazard Index - Casa Grande 4-34
4-23 Summary of Total Future Incremental Carcinogenic Risk and Total Future Noncarcinogenic Hazard Index for the RME Scenario - Casa Grande 4-35
4-24 Summary of Total Future Incremental Carcinogenic Risk and Total Future Noncarcinogenic Hazard Index for the CTC Scenario - Casa Grande 4-36

CONTENTS
(Cont'd)

4-25 Summary of Total Estimated Incremental Carcinogenic Risk and Total Estimated Noncarcinogenic Hazard Index for the RME Scenario - Payson 4-38
4-26 Summary of Total Estimated Incremental Carcinogenic Risk and Total Estimated Noncarcinogenic Hazard Index for the CTC Scenario - Payson 4-39
4-27 Toxicity Endpoints Analysis for Noncarcinogenic Effects of COI Due to Inhalation Young Child Resident RME Scenario - Payson 4-41
4-28 Toxicity Endpoints Analysis for Noncarcinogenic Effects of COI Due to Inhalation Young Child Resident CTC Scenario - Payson 4-42
4-29 Arizona HAPs, Casa Grande, Air Assessment Integrated Exposure Uptake Biokinetic Model for Lead in Children 4-43
4-30 Comparison of Deterministic and Probabilistic Results for Estimated Carcinogenic Risk and Noncarcinogenic Hazard Index - Payson 4-46
4-31 Summary of Total Future Incremental Carcinogenic Risk and Total Future Noncarcinogenic Hazard Index for the RME Scenario - Payson 4-47
4-32 Summary of Total Future Incremental Carcinogenic Risk and Total Future Noncarcinogenic Hazard Index for the CTC Scenario - Payson 4-48
4-33 Comparison of Deterministic and Probabilistic Results for Estimated Total Carcinogenic Risk - All Regions 4-49
4-34 Comparison of Deterministic and Probabilistic Results for Estimated Total Noncarcinogenic Hazard Index - All Regions 4-50

LIST OF FIGURES

3-1 Comparison of Deterministic (Point) Versus a Probabilistic Estimate of Exposure . . . 3-5
3-2 Representative Distributions for Adult Lifetime Resident Exposure Parameters 3-10
4-2 Distribution for Total HI - Young Child Resident, Tucson 4-11
4-3 Distribution for Total ELCR - Lifetime Resident, Tucson 4-22
4-4 Distribution for Total HI - Young Child Resident, Tucson 4-23
4-5 Distribution for Total ELCR - Lifetime Resident, Casa Grande 4-32
4-6 Distribution for Total HI - Young Child Resident, Casa Grande 4-33
4-7 Distribution for Total ELCR - Lifetime Resident, Payson 4-44
4-8 Distribution for Total HI - Young Child Resident, Payson 4-45

APPENDIX F - RISK ASSESSMENT

1.0 INTRODUCTION

This appendix presents the results of the risk assessments conducted for each of the Arizona study regions. The risk assessments have been conducted according to the four step paradigm developed by the National Research Council (NRC, 1983, 1994), as follows:

1. Identify the Hazard. Select chemical compounds for which a quantitative risk assessment should be performed because of their toxicity, prevalence, mobility, and/or persistence.

2. Assess Toxicity. For the selected compounds, determine the relationship between the magnitude of exposure (dose) and the occurrence of specific health effects (response).

3. Assess Exposure. Determine the magnitude and frequency of potential human exposure to the compounds of interest.

4. Characterize Risk. Estimate potential risks to human health by combining the information in the toxicity assessment and the exposure assessment.

The risk assessment method has been described in detail in Volume I of this Final Report. The purpose of this appendix is to provide additional information, evaluate uncertainties in the available information, describe additional methods employed in the risk assessment, and present the detailed results of the risk assessment.

The development of the Research HAPs List (in Appendix D of Volume I) and the selection of the Chemicals of Interest (COI) (addressed in Section 3.0 in the main body of this volume) constituted the hazard identification step.

The COI identified for each region were quantitatively evaluated for potential carcinogenic and noncarcinogenic health risks. The numerical toxicity values for each of the COI, for both potentially carcinogenic and noncarcinogenic effects are provided in the Toxicity Assessment in Section 2.0 of this Appendix. The toxicity assessment methodology was presented in Volume I of the Final Report; however, some of that text has been included here in order to reinforce the main concepts upon which the discussion of the uncertainties in the toxicity values, also presented in Section 2.0, is based.

The exposure assessment in Volume I of the Final Report presented the receptors and exposure parameters used to evaluate receptor exposure for both a reasonable maximal exposure scenario and a central tendency case scenario. The method described in Volume I represents a deterministic, or point estimate, approach to the calculation of risk. This approach is summarized in Section 3.1 below.

To augment the deterministic risk assessment for each region, a probabilistic analysis has been performed to calculate distributions of receptor risk. The probabilistic analysis is discussed in detail in Section 3.2.

The results of both the deterministic and probabilistic analyses for all four regions are presented in the risk characterizations in Section 4.0.

2.0 TOXICITY ASSESSMENT

In the toxicity assessment, the relationship between the magnitude of exposure (dose) and the occurrence of the specific health effect (response) for each COI is evaluated. Two categories of health effects are identified in health risk assessments, potentially carcinogenic and noncarcinogenic health effects, and both are evaluated very differently in the risk assessment process. Separate dose-response values are derived for potentially carcinogenic and noncarcinogenic health effects.

Sources of the published dose-response values that were used in the risk assessment include U.S. EPA's Integrated Risk Information System (IRIS) (U.S. EPA, 1995a), the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b) and values provided by the U.S. EPA Environmental Criteria and Assessment Office (ECAO) in Cincinnati, Ohio, and, for chemicals that do not have U.S. EPA-derived values, the California Environmental Protection Agency (CalEPA) (CalEPA, 1994).

The primary source of dose-response values was the U.S. EPA Integrated Risk Information System (IRIS) which is an on-line computer database of toxicological information (U.S. EPA, 1995a). The IRIS database is updated monthly to provide the most current U.S. EPA-verified dose-response values. As defined by the U.S. EPA (U.S. EPA, 1995b), a toxicity value or cancer value is "Work Group-verified" if all available information on the value has been examined by an Agency Work Group, the value has been calculated using current Work Group methodology, a unanimous consensus has been reached on the value by the Work Group, and the value appears on IRIS.

Another source of dose-response values was the U.S. EPA Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b). HEAST is published annually by the U.S. EPA and provides a compilation of dose-response values available at the time of publishing. Because HEAST is published annually, the dose-response values provided may not represent the most current values available. In addition, the dose-response values provided by HEAST are considered to be provisional, i.e., the value has had some form of agency review, but does not appear on IRIS. These values may or may not have been generated through the Agency Work Group process, but the values generally use all available information, use current methodology and a consensus was reached by Agency scientists on the value. HEAST is therefore considered to be an unverified source of dose-response values and should be used only if no dose-response value is available on IRIS.

When a dose-response value was not available from IRIS or HEAST, the U.S. EPA Environmental Criteria and Assessment Office (ECAO) in Cincinnati was consulted for a provisional dose-response value. These dose-response values may or may not meet the HEAST criteria. Therefore, the hierarchy of dose-response value sources correlates in general with the level of confidence in the values, with the values provided directly by ECAO having the lowest level of scientific review and approval and thus the least level of confidence.

Finally, CalEPA provides CSF for a list of chemicals. CalEPA CSF were used in this risk assessment if the U.S. EPA sources did not provide a value. This was the case for both 1,4-dichlorobenzene, and the potentially carcinogenic PAH.

The development of dose-response values is described in Sections 2.1 and 2.2 for potentially carcinogenic and noncarcinogenic effects, respectively. Attachment F-1 presents detailed information concerning the derivation of the dose-response values for each of the COI.

2.1 Carcinogen Dose-Response

The risk assessment for potentially carcinogenic effects estimated the probability over a lifetime of contracting any type of cancer due to exposure to a specific chemical, in excess of those cancers that would result anyway in the absence of exposure to that chemical. Although there is recent evidence to the contrary, it was assumed in the toxicity assessment for potential carcinogens that a finite level of risk of contracting cancer is associated with any dose above zero, theoretically even with exposure to a single molecule. This assumption, termed the no-threshold assumption, is a controversial one, but it is typically used in regulatory risk assessment because it maximizes the protection of health.

The numerical estimate of a chemical's carcinogenicity that was used in this risk assessment is the cancer slope factor (CSF). The CSF can be thought of as the risk of contracting cancer, due to exposure to the chemical of interest, if the intake of the chemical into the body is 1 milligram (mg) per day for each kilogram (kg) of body weight for every day of a 70-year lifetime. (A milligram is about 1/29th of an ounce, and a kilogram is about 2.2 pounds.) Because a milligram is 1/1000 of a gram and a kilogram is 1000 grams, an alternative interpretation is that the CSF is the risk of contracting cancer if an amount equal to one-millionth of the body weight enters the body every day for 70 years. The estimated risks in the risk assessment are determined by multiplying the CSF by the estimated exposure dose. CSFs are expressed in terms of $(mg/kg\text{-}day)^{-1}$. When multiplied by the lifetime average daily dose estimated in the exposure assessment and expressed in terms of mg/kg-day, the product is the unitless risk estimate.

Data upon which to base the development of a CSF is limited. Data on the chemical causes of cancer in humans is very limited. Cancer development in humans is usually inferred from experiments with laboratory animals. The human carcinogenic potential of a chemical is classified based on the weight of the scientific evidence. Accordingly, the U.S. EPA classifies chemicals into one of the following groups:

- Group A: Human Carcinogen (sufficient evidence of carcinogenicity in humans)
- Group B: Probable Human Carcinogen (B1 = limited evidence of carcinogenicity in humans; B2 = sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- Group C: Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data)
- Group D: Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)
- Group E: Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies)

The CSFs used in this risk assessment are presented in Table 2-1. These are CSF associated with exposure to COI by inhalation. For each COI evaluated in the risk assessment, the table presents the carcinogen classification, the CSF, and the study animal and route of exposure used in the experiment upon which the CSF was based.

2.1.1 Uncertainty in the Carcinogen Dose-Response Values

There are many uncertainties in the dose response values for carcinogenic effects. COI such as arsenic, benzene, hexavalent chromium, and nickel are classified as Group A carcinogens based on studies of workers exposed to these chemicals at the workplace. Therefore, there is more confidence in these CSF than for other compounds. However, workplace exposure levels are generally much higher than predicted environmental levels, and it is not certain that these chemicals are as potent carcinogens at low environmental levels as they are at the higher workplace levels. Therefore, even for these COI there is some uncertainty about the predicted risk levels.

The potential carcinogenic risks calculated for cadmium and acrylonitrile are also based on human occupational exposure studies. These two COI are classified as B1 carcinogens based on limited evidence of carcinogenicity in humans. Therefore, less confidence can be placed on

TABLE 2-1
DOSE-RESPONSE INFORMATION FOR COMPOUNDS WITH POTENTIAL CARCINOGENIC EFFECTS
ALL AREAS: PAYSON, TUCSON, CASA GRANDE, PHOENIX
HUMAN HEALTH RISK ASSESSMENT

Compound	CAS Number	EPA Carc. Class	Inhalation CSF [1/(mg/kg-day)]	Inh. CSF Ref. (Last Verified)	Inhal. CSF Study Animal	Inhal. CSF Study Method
ARSENIC	7440-38-2	A	1.50E+01	IRIS (10/95)	Human	Inhalation:Occupational
BARIUM	7440-39-3	NA	NA	NA	NA	NA
CADMIUM	7440-43-9	B1	6.30E+00	IRIS (10/95)	Human	Inhalation:Occupational
CHROMIUM (HEXAVALENT)	7440-47-3	A	4.10E+01	HEAST (1995)	Human	Inhalation:Occupational
MANGANESE	7439-96-5	D	NA	NA	NA	NA
MERCURY	7439-97-6	D	NA	NA	NA	NA
NICKEL	7440-02-0	A	8.40E-01	IRIS (10/95)	Human	Inhalation
ACETALDEHYDE	75-07-0	B2	7.70E-03	IRIS (10/95)	Rat	Inhalation
ACROLEIN	107-02-8	C	NA	NA	NA	NA
ACRYLONITRILE	107-13-1	B1	2.40E-01	HEAST (1995)	Human	Inhalation:Occupational
AMMONIA	7664-41-7	NA	NA	NA	NA	NA
BENZENE	71-43-2	A	2.90E-02	HEAST (1995)	Human	Inhalation:Occupational
1,3-BUTADIENE	106-99-0	B2	9.80E-01	IRIS (10/95)	Mouse	Inhalation
CARBON TETRACHLORIDE	56-23-5	B2	5.30E-02	IRIS (10/95)	Hamster, Mouse, Rat	Oral:Gavage
CHLOROBENZENE	108-90-7	D	NA	NA	NA	NA
CHLOROFORM	67-66-3	B2	8.10E-02	HEAST (1995)	Mouse	Oral:Gavage
1,4-DICHLOROBENZENE	106-46-7	B2	4.00E-02	CA 1994	Mouse	Oral:Gavage
DICHLOROMETHANE	75-09-2	B2	1.60E-03	IRIS (10/95)	Mouse	Inhalation
ETHYLENE DIBROMIDE	106-93-4	B2	7.70E-01	IRIS (10/95)	Rat	Inhalation
ETHYLENE OXIDE	75-21-8	B1	3.50E-01	HEAST (1995)	Rat	Inhalation:Intermittent
FORMALDEHYDE	50-00-0	B1	4.50E-02	IRIS (10/95)	Rat	Inhalation
N-HEXANE	110-54-3	NA	NA	NA	NA	NA
TETRACHLOROETHYLENE	127-18-4	B2	2.00E-03	ECAO (10/93)	NA	NA
TOLUENE	108-88-3	D	NA	NA	NA	NA
TRICHLOROETHYLENE	79-01-6	B2	6.00E-03	ECAO (3/93)	Mouse	Inhalation
TRIMETHYLBENZENE (TOTAL)	25551-13-7	NA	NA	NA	NA	NA
VINYL CHLORIDE	75-01-4	A	3.00E-01	HEAST (1995)	Rat	Inhalation:Intermittent
ACENAPHTHENE	83-32-9	NA	NA	NA	NA	NA
ACENAPHTHYLENE	208-96-8	NA	NA	NA	NA	NA
ANTHRACENE	120-12-7	NA	NA	NA	NA	NA
BENZO(A)ANTHRACENE	56-55-3	B2	3.90E-01	CA (1994), US EPA (1993)	Hamster	Inhalation
BENZO(A)PYRENE	50-32-8	B2	3.90E+00	CA (1994), US EPA (1993)	Hamster	Inhalation
BENZO(B)CHRYSENE	BBCHRN	NA	NA	NA	NA	NA
BENZO(B+J+K)FLUORANTHENE	205-99-2	B2	3.90E-01	CA (1994), US EPA (1993)	Hamster	Inhalation
BENZO(G,H,I)PERYLENE	191-24-2	NA	NA	NA	NA	NA
BIPHENYL	95-52-4	D	NA	NA	NA	NA
CHRYSENE	218-01-9	B2	3.90E-03	CA (1994), US EPA (1993)	Hamster	Inhalation
DIBENZ(A,H+A,C)ANTHRACENE	53-70-3	B2	3.89E+00	CA (1994), US EPA (1993)	Hamster	Inhalation
1,2-DIMETHYLNAPHTHALENE	DMN12	D	NA	NA	NA	NA
1,3+1,6+1,7-DIMETHYLNAPHTHALENE	DM1367	NA	NA	NA	NA	NA
1,4+1,5+2,3-DIMETHYLNAPHTHALENE	DM14523	D	NA	NA	NA	NA
1,8-DIMETHYLNAPHTHALENE	DMN18	NA	NA	NA	NA	NA
2,6+2,7-DIMETHYLNAPHTHALENE	DMN267	D	NA	NA	NA	NA
FLUORANTHENE	206-44-0	NA	NA	NA	NA	NA
FLUORENE	86-73-7	NA	NA	NA	NA	NA
INDENO(1,2,3-CD)PYRENE	193-39-5	B2	3.90E-01	CA (1994), US EPA (1993)	Hamster	Inhalation
1-METHYLNAPHTHALENE	MNAPH1	NA	NA	NA	NA	NA
2-METHYLNAPHTHALENE	91-57-6	D	NA	NA	NA	NA
NAPHTHALENE	91-20-3	NA	NA	NA	NA	NA
PHENANTHRENE	85-01-8	NA	NA	NA	NA	NA
PYRENE	129-00-0	D	NA	NA	NA	NA
CHLORPYRIFOS	2921-88-2	NA	NA	NA	NA	NA
DISULFOTON	298-04-4	NA	NA	NA	NA	NA
METHYL PARATHION	298-00-0	NA	NA	NA	NA	NA
PENTACHLORONITROBENZENE	82-68-8	C	2.60E-01 (a)	HEAST (1995)	Mouse	Oral
TRIFLURALIN	1582-09-8	C	7.70E-03 (a)	IRIS (10/95)	Rat	Oral:Diet

Notes:
CAS - Chemical Abstracts Service.
CSF - Cancer Slope Factor.
IRIS - Integrated Risk Information System, an online computer database of toxicological infomation (U.S. EPA, 1995).
HEAST - Health Effects Assessment Summary Tables, published annually by the U.S. EPA (1995).
CA 1994 - California EPA. 1994. California Cancer Potency Factors: Update Memorandum. November 1, 1994.
US EPA. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. July 1993.
NA - Not Applicable
(a) - Oral dose-response value used to evaluate inhalation exposures.

Source: ENSR 1995 DRCALL.WQ1 RN: 1

the risk estimates for these two COI than for the COI classified in Group A, but more confidence can be placed on these risk estimates than for risk estimates based on animal study results.

For many chemicals, animal studies provide the only reliable information upon which to base an estimate of adverse human health effects. Extrapolation from animals to humans introduces a great deal of uncertainty into the risk characterization. In most instances it is not known how differently a human may react to the chemical compared to the animal species used to test the chemical. The CSF for the COI not mentioned above are all based on animal studies. If a compound's fate and the mechanisms by which it causes adverse effects is known in both animals and humans, uncertainty is reduced. When the fate and mechanism for the chemical are unknown, uncertainty increases.

Often there is only one study of the carcinogenicity of a chemical upon which to base the CSF. When there is a choice of studies, or when more than one species has been tested in a study, the U.S. EPA bases the CSF on the species and tumor site that shows the most significant increases in tumor incidence with increasing dose. In some cases this selection is done in spite of tumor incidence in other organs and total tumor incidence showing significant decreases with increasing dose. In the case of 1,3-butadiene, both mice and rats have been used in carcinogenicity tests. Mice exhibited higher tumor incidences at lower exposure levels than rats upon inhalation exposure to 1,3-butadiene. The EPA CSF is based upon the mouse data. However, recent research indicates that mice and rats metabolize this compound much differently, and that humans are much more like rats in their metabolism of 1,3-butadiene. Therefore, use of the EPA CSF for 1,3-butadiene is likely to overestimate potential human risks. For many other COI, it is not known if use of the animal model upon which the CSF is based results in an over- or under-estimation of human health risks. Therefore, this contributes to the uncertainty.

The U.S. EPA converts doses administered to experimental animals to obtain human equivalent doses by using the assumption that humans and animals are equally sensitive to the toxic effects of a substance if the same dose per unit body surface area is absorbed by each species. While this assumption holds for certain types of chemicals (i.e., direct acting genotoxins), it is not appropriate for all chemicals. This assumption may over-estimate human health risk by a factor of 6 to 12 depending on the study species.

An additional extrapolation is required when predicting human health effects from the high experimental doses used in animal studies to the low doses expected from environmental exposures. The U.S. EPA uses statistical models to accomplish this. The one most often used is the linearized multistage model. The CSF calculated from this model is the upper 95% confidence limit of the dose-response slope. U.S. EPA states that this upper-bound estimate

leads to a plausible upper limit to the risk, but the true risk, however, is unknown, and may be as low as zero. Therefore, use of this statistical model to predict health risk introduces uncertainty into the assessment, and the risk results are more likely to be an over-estimation than an underestimation of the true risk.

Additional uncertainty is introduced when animal data from experiments in which the chemicals were administered to the study animal orally are used to derived CSF for inhalation exposures. This is the case for the following COI in this risk assessment: carbon tetrachloride, 1,4-dichlorobenzene, and chloroform. In addition, the U.S. EPA does not provide any inhalation CSF values for the pesticide COI evaluated in the risk assessment for the Casa Grande region. However, two of these COI do have oral CSF values: pentachloronitrobenzene, and trifluralin. The decision was made to use the oral CSF to evaluate inhalation exposures rather than omit these chemicals from further consideration in the risk assessment, due to the importance of agricultural chemicals in this region. The use of the oral CSF for these COI introduces an unknown amount of uncertainty into the risk assessment.

As stated above, it is assumed in the toxicity assessment for potential carcinogens that a finite level of risk of contracting cancer is associated with any dose above zero. However, this assumption has been challenged based on recent experimental results from studies designed to elucidate the mechanism of carcinogenesis of chloroform. As discussed in greater detail in Attachment A, there is evidence that a threshold for carcinogenic effects does exist for chloroform, and that an air concentration of 0.049 mg per cubic meter of air is a "virtually safe dose" for exposure to chloroform. It should be noted that all of the predicted or measured air concentrations for chloroform in each region are well below this value. Therefore, it is likely that the risks predicted for chloroform in this risk assessment greatly over-estimate the true risk.

Polycyclic aromatic hydrocarbons (PAH) are identified as COI in the Payson region. While the emissions estimates were classified only as a class of compounds (POM or polycyclic organic matter), the air monitoring data from the Payson region provided air concentrations of a large number of PAH compounds. The U.S. EPA has classified seven PAH as probable human carcinogens (Class B2), and the California EPA (CalEPA) has classified 21 as potential carcinogens. The U.S. EPA does not have an inhalation CSF for carcinogenic PAH, however, CalEPA does provide a value for benzo(a)pyrene. To perform carcinogenic risk assessment for the other potentially carcinogenic PAH, an estimate of their potency is needed. Comparative potency approaches have been developed by both U.S. EPA and CalEPA. Experimental studies on benzo(a)pyrene and other PAH were used to derive comparative potency factors that relate the potency of other PAH to benzo(a)pyrene. The comparative potency factors used in this assessment are presented in Table 2-2. Although CalEPA provides potency estimates for additional PAH, none of those PAH were identified in Payson. The comparative potency factors

TABLE 2-2

Relative Potency Factors for Potentially Carcinogenic Polycyclic Aromatic Hydrocarbons - All Areas: Payson, Tucson, Casa Grande, Phoenix

Constituent	Benzo(a)pyrene Toxic Equivalent Factor
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenzo(a,h)anthracene	1.0
Indeno(1,2,3-cd)pyrene	0.1

TEF.WQ1 RN:0

Source: U.S. EPA, 1993

were combined with the CalEPA inhalation CSF for benzo(a)pyrene as shown in Table 2-1, for the evaluation of potential carcinogenic health risks. Use of the comparative potency approach introduces uncertainty into the risk estimates, but it cannot be known whether the predicted health risks are over- or under-estimating the true health risks.

2.2 Noncarcinogen Dose-Response

The term noncarcinogenic is used to describe toxic properties of chemicals other than carcinogenicity. Just as the term carcinogenic can be associated with a variety of different types of cancer, there are many health endpoints included in the noncarcinogenic category, such as liver, kidney or respiratory effects. Chemicals that are classified as carcinogens can also have noncarcinogenic properties and dose-response values. Fifteen of the COI in this risk assessment were evaluated for both potentially carcinogenic and noncarcinogenic health effects.

Compounds that have effects that are not carcinogenic are assumed to have a dose level below which no adverse effect occurs or, conversely, above which an adverse effect may be seen. This dose is called the threshold dose. An estimate of the threshold dose that is based on experimental determination that no effect occurs at a given dose is called a No Observed Adverse Effect Level (NOAEL). The true threshold can be assumed to be at or above the NOAEL. The lowest dose at which an adverse effect has been observed in an experiment is called a Lowest Observed Adverse Effect Level (LOAEL). The true threshold dose is somewhere below the LOAEL. The type of adverse effect, such as liver damage, change in kidney function, altered respiratory function, or a change in a blood parameter, with which the NOAEL or LOAEL is associated, is termed the critical effect for the study.

By applying uncertainty factors to the NOAEL or the LOAEL, References Doses (RfDs) and Reference Concentrations (RfCs) for chronic exposures to chemicals with noncarcinogenic effects have been developed by the U.S. EPA (1995a, 1995b). Uncertainty factors are applied to the NOAEL or LOAEL to account for uncertainties associated with the dose-response relationship due to using an animal study to derive a human dose-response value, extrapolating from short term studies to long term exposures, and to reflect effects on sensitive subpopulations. Generally, a 10-fold factor is used to account for each of these uncertainties; thus, the total uncertainty factor can range from 10 to 10,000.

This use of uncertainty factors assumes that humans are more sensitive to the toxic effects of chemicals than animals, and therefore, the estimated human threshold is assumed to be up to several orders of magnitude below the threshold defined in an animal study. For compounds with noncarcinogenic effects, an RfD or RfC that is thus derived provides reasonable certainty

that noncarcinogenic health effects are not expected to occur even if daily exposures were to occur at the RfD or RfC level for a lifetime.

RfDs (and exposure doses) are expressed in units of milligrams of chemical per kilogram of body weight per day (mg/kg-day). RfCs are dose-response values specifically for the inhalation route of exposure and are expressed as milligrams of compound per cubic meter of air (mg/m^3). In order to use these dose-response values to calculate an average daily exposure dose (as described in Section 3.0, Exposure Assessment), the RfCs are converted to RfDs for a 70 kg (about 155 lb) body weight and assuming that 20 m^3 of air is inhaled during one day. Thus the conversion from RfC to RfD follows the formula cited in HEAST (U.S. EPA, 1995b):

$$RfC\ (mg/m^3) \times (1/70\ kg) \times (20\ m^3/day) = RfD\ (mg/kg\text{-}day).$$

The inhalation RfDs are presented in Table 2-3. For each COI, the table presents the RfD, the uncertainty factor, the EPA confidence level, the target organ/critical effect at the LOAEL, the study animal, and the route of exposure. The toxic endpoint categories associated with each COI are presented in Table 2-4. These grouping were used for performing the toxic endpoint analyses for the study regions.

2.2.1 Uncertainty in Noncarcinogenic Dose-Response

In accordance with U.S. EPA ECAO policy, the evaluation of noncarcinogenic effects following inhalation exposures was being made only for those chemicals whose toxic effects have been studied following inhalation exposures, and for which U.S. EPA has developed RfC values. All of the inhalation RfDs are based on inhalation studies in humans or animals. However, an exception was made for the evaluation of pesticides in the Casa Grande study region because of the agricultural importance of these chemicals. For these chemicals, the oral RfD values were used to evaluate inhalation exposures. This assumption introduces some uncertainty into the risk assessment, and it cannot be known whether use of these values will tend to over- or under-estimate the potential for human health effects to occur.

As with the carcinogenic dose-response values, uncertainty in the reference doses depends on the study animal used for evaluation, the route of exposure, and additionally on the Uncertainty Factor used to derive the RfD. For those values available on IRIS or from the ECAO, a level of confidence in the dose-response value is provided.

Human occupational studies served as the basis for seven of the inhalation RfDs: manganese, mercury, ammonia, ethylenedibromide, n-hexane, toluene, and trimethylbenzene. The remaining RfDs are based on animal studies. As shown in Table 2-2, the confidence levels for the

DOSE-RESPONSE INFORMATION FOR COMPOUNDS WITH POTENTIAL NONCARCINOGENIC EFFECTS FROM CHRONIC EXPOSURE THROUGH THE INHALATION ROUTE
ALL AREAS: PAYSON, TUCSON, CASA GRANDE, PHOENIX
HUMAN HEALTH RISK ASSESSMENT

Compound	Inh. Dose-Response Value (mg/kg-day)		Reference (Last Verified), Type	Uncertainty Factors	EPA Confidence Level	Target Organ / Critical Effect at LOAEL	Study Animal	Study Method
ARSENIC	NA		NA	NA	NA	NA	NA	NA
BARIUM	1.43E-04	(a)	HEAST (1995)	1000	NA	Fetotoxicity	Rat	Inhalation:Intermittent
CADMIUM	NA		NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	NA		NA	NA	NA	NA	NA	NA
MANGANESE	1.43E-05	(a)	IRIS (10/95)	300	Medium	Increased respiratory symptoms and psychomotor effects	Human	Inhalation:Occupational
MERCURY	8.57E-05	(a)	IRIS (10/95)	30	Medium	Membrane disturbances: autonomic disfunction	Human	Inhalation:Occupational
NICKEL	NA		NA	NA	NA	NA	NA	NA
ACETALDEHYDE	2.87E-03	(a)	IRIS (10/95)	1000	Low	Degeneration of olfactory epithelium	Rat	Inhalation:Intermittent
ACROLEIN	5.71E-06	(a)	IRIS (10/95)	1000	Medium	Squamous metaplasia; neutrophilic infiltration of nasal epithelium	Rat	Inhalation:Intermittent
ACRYLONITRILE	5.71E-04	(a)	IRIS (10/95)	1000	Medium	Degeneration and inflamation of nasal respiratory epithelium: hyperplas	Rat	Inhalation
AMMONIA	2.86E-02	(a)	IRIS (10/95)	30	Medium	No adverse effects observed	Human	Inhalation:Occupational
BENZENE	1.71E-03	(a)	ECAO (6/95)	1000	Low	Hematopoietic effects	Mice	Inhalation:Intermittent
1,3-BUTADIENE	NA		NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	5.71E-04	(a)	ECAO (11/95)	3000	Low	Increased liver weight	Guinea Pig	Inhalation:Intermittent
CHLOROBENZENE	5.70E-03	(a)	HEAST (1995)	10000	NA	Liver and kidney effects	Rat	Inhalation:Intermittent
CHLOROFORM	NA		NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	2.30E-01	(a)	IRIS (10/95)	100	Medium	Increased liver weight	Rat	Inhalation
DICHLOROMETHANE	8.60E-01	(a)	HEAST (1995)	100	NA	Liver toxicity	Rat	Inhalation:Intermittent
ETHYLENE DIBROMIDE	5.71E-05	(a)	HEAST (1995)	1000	NA	Sperm effects	Human	Inhalation:Intermittent
ETHYLENE OXIDE	NA		NA	NA	NA	NA	NA	NA
FORMALDEHYDE	NA		NA	NA	NA	NA	NA	NA
N-HEXANE	8.71E-02	(a)	IRIS (10/95)	300	Medium	Neurotoxicity; electrophysiological alterations	Human	Inhalation:Occupational
TETRACHLOROETHYLENE	1.14E-01	(a)	ECAO (7/95)	300	Medium	Kidney effects	Rat	Inhalation:Intermittent
TOLUENE	1.14E-01	(a)	IRIS (10/95)	300	Medium	Neurological effects	Human	Inhalation:Occupational
TRICHLOROETHYLENE	NA		NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	1.71E-03	(a)	ECAO (7/95)	3000	Low	Adverse respiratory, nuerological, and hematological effects.	Human	Inhalation:Occupational
VINYL CHLORIDE	NA		NA	NA	NA	NA	NA	NA
ACENAPHTHENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
ACENAPHTHYLENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
ANTHRACENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
BENZO(A)ANTHRACENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
BENZO(A)PYRENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
BENZO(B)CHRYSENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
BENZO(B+J+K)FLUORANTHENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
BENZO(G,H,I)PERYLENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
BIPHENYL	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
CHRYSENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
DIBENZ(A,H+A,C)ANTHRACENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
1,2-DIMETHYLNAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
1,3+1,6+1,7-DIMETHYLNAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
1,4+1,5+2,3-DIMETHYLNAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
1,6-DIMETHYLNAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
2,6+2,7-DIMETHYLNAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
FLUORANTHENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
FLUORENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
INDENO(1,2,3-CD)PYRENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
1-METHYLNAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
2-METHYLNAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
NAPHTHALENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
PHENANTHRENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
PYRENE	1.14E-03	(a,b)	ECAO (10/95)	3000	Low	Nasal effects	Mice	Inhalation:Intermittent
CHLORPYRIFOS	3.00E-03	(c)	IRIS (10/95)	10	Medium	Cholinesterase inhibition	Human	Oral
DISULFOTON	4.00E-05	(c)	IRIS (10/95)	1000	Medium	Optic nerve degeneration; cholinesterase inhibition	Rat	Oral Diet
METHYL PARATHION	2.50E-04	(c)	IRIS (10/95)	100	Medium	Reduced hemoglobin, hematocrit, and RBCs	Rat	Oral:Diet
PENTACHLORONITROBENZENE	3.00E-03	(c)	IRIS (10/95)	300	Medium	Microscopic cholestatic hepatosis	Dog	Oral Diet
TRIFLURALIN	7.50E-03	(c)	IRIS (10/95)	100	High	Increased liver weight; increased methemoglobin	Dog	Oral:Diet

Notes:
CAS - Chemical Abstracts Service.
LOAEL - Lowest Observed Adverse Effects Level.
RIC - Reference Concentration.
IRIS - Integrated Risk Information System, an on-line computer database of toxicological information (U.S. EPA, 1995).
HEAST - Health Effects Assessment Summary Tables, published annually by the U.S. EPA (1995).
NA - Not Applicable
(a) - Calculated from the reference concentration assuming a 70 kg adult inhales 20 m^3/day.
(b) - Due to structural similarity, calculated from the reference concentration for naphthalene of 4E-03 mg/m^3.
(c) - Oral RfD used to evaluate inhalation exposures.

Source: ENSR 1995 RN:1

TABLE 2-4
SUMMARY OF TOXICITY ENDPOINTS FOR COMPOUNDS WITH POTENTIAL NONCARCINOGENIC EFFECTS
FROM CHRONIC EXPOSURE THROUGH THE INHALATION ROUTE
ALL AREAS: PHOENIX, TUCSON, CASA GRANDE AND PAYSON
HUMAN HEALTH RISK ASSESSMENT

Compound	Blood Effects	Developmental Effects	Kidney Effects	Liver Effects	Neurological Effects	No Adverse Effects	Reproductive Effects	Respiratory Effects
ARSENIC								
BARIUM		X						
CADMIUM								
CHROMIUM (HEXAVALENT)								
MANGANESE					X			
MERCURY					X			X
NICKEL								
ACETALDEHYDE								X
ACROLEIN								X
ACRYLONITRILE								X
AMMONIA						X		
BENZENE	X							
1,3-BUTADIENE								
CARBON TETRACHLORIDE				X				
CHLOROBENZENE			X	X				
CHLOROFORM								
1,4-DICHLOROBENZENE				X				
DICHLOROMETHANE				X				
ETHYLENE DIBROMIDE							X	
ETHYLENE OXIDE								
FORMALDEHYDE					X			
N-HEXANE			X					
TETRACHLOROETHYLENE					X			
TOLUENE								
TRICHLOROETHYLENE								
TRIMETHYLBENZENE (TOTAL)	X				X			X
VINYL CHLORIDE								
ACENAPHTHENE								X
ACENAPHTHYLENE								X
ANTHRACENE								X
BENZO(A)ANTHRACENE								X
BENZO(A)PYRENE								X
BENZO(B)CHRYSENE								X
BENZO(B+J+K)FLUORANTHENE								X
BENZO(G,H,I)PERYLENE								X
BIPHENYL								X
CHRYSENE								X
DIBENZ(A,H+A,C)ANTHRACENE								X
1,2-DIMETHYLNAPHTHALENE								X
1,3+1,6+1,7-DIMETHYLNAPHTHALENE								X
1,4+1,5+2,3-DIMETHYLNAPHTHALENE								X
1,8-DIMETHYLNAPHTHALENE								X
2,6+2,7-DIMETHYLNAPHTHALENE								X
FLUORANTHENE								X
FLUORENE								X
INDENO(1,2,3-CD)PYRENE								X
1-METHYLNAPHTHALENE								X
2-METHYLNAPHTHALENE								X
NAPHTHALENE								X
PHENANTHRENE								X
PYRENE								X
CHLORPYRIFOS	X							
DISULFOTON	X				X			
METHYL PARATHION	X							
PENTACHLORONITROBENZENE				X				
TRIFLURALIN	X			X				

TOXPOINT.WQ1 RN: 1
Source: ENSR 1995

inhalation RfDs are either medium or low, and the uncertainty factors span a wide range from a low of 30 for ammonia and mercury to a high of 10,000 for chlorobenzene. These uncertainties need to be kept in mind when evaluating the results of the risk assessment.

2.3 Risk Assessment for Lead

Although the U.S. EPA has classified lead as a B2 (probable) human carcinogen, no CSF has been developed. Because of the uncertainties in the dose-response relationship between exposure to lead and biological effects, it is unclear whether the noncarcinogenic effects of lead exhibit a threshold response, and an RfD for lead is not available. Therefore, potential exposures to lead cannot be evaluated using the traditional methods of risk assessment. However, the U.S. EPA has developed an Integrated Exposure Uptake Biokinetic (IEUBK) model that correlates lead levels in the environment to blood lead levels in children (U.S. EPA, 1994). This model has been used to evaluate potential exposures to lead in each of the study regions. This section presents a discussion of the model and how it was applied in this risk assessment.

It is generally believed that increasing blood lead concentrations in children correlate with adverse neurological effects. The IEUBK model links typical risk assessment exposure analysis with a biokinetic model of lead uptake and distribution in the body to enable estimates of blood lead levels that may occur due to overall exposure to lead in the environment. The model is designed to consider differing exposure patterns and physiological changes in various age groups in calculating estimates of blood lead levels.

The IEUBK model predicts blood lead levels in children 0-7 years of age due to exposures to lead from multiple sources, including air, water, soil/dust, and diet, as well as contribution from maternal sources. The model's default parameters define health-protective but plausible exposure estimates to lead in ambient air, drinking water, diet, and soil/dust for children 0-7 years of age based on their behavior patterns under residential circumstances. The model incorporates age-specific estimates of amounts of drinking water and diet consumed along with variations in respiratory rates and activity patterns between infants, and young and older children.

The health effects of most concern from lead exposures are impaired mental and physical development in young children. Available evidence suggests that a threshold for these effects lies between 10 to 15 micrograms per deciliter of blood (μg/dL) (U.S. EPA, 1994). (A microgram is 1/1000 of a milligram, or 1/1,000,000 of a gram, and a deciliter is 1/10 of a liter.) Evidence for adverse effects below the 10 μg/dL level is uncertain and remains controversial. Regulatory agencies such as the U.S. EPA and the Center for Disease Control (CDC) have designated the 10 μg/dL blood lead level as a "level of concern." This designation does not imply that adverse

health effects are present in individuals with this blood concentration, but raises concern that additional exposures may result in adverse effects.

The CDC's most recent recommendations on preventing lead poisoning in young children state, as a goal, the reduction of children's blood lead levels to below 10 μg/dl. Surveys have found that in many settings a portion of children will have blood lead levels greater than this value. Because of the multiple source nature of lead exposure, it is impractical to eliminate all sources of lead exposure and achieve the goal of having 100% of children with blood lead levels less than 10 μg/dL. To address this situation, the U.S. EPA has selected a level of overall protectiveness for lead. The Agency's policy, used since the 1970s with the introduction of the ambient air standard for lead of 15 μg per cubic meter of air, is that regulatory criteria should allow the probability that only a small fraction of the exposed population would exceed the 10 μg/dL blood lead level. An exceedance probability of no more than 5% has been used historically by the Agency in evaluating cleanup actions. This criterion has been applied to the IEUBK model results presented here to evaluate potential exposure to lead in ambient air.

The IEUBK model was used to evaluate potential exposures to lead in ambient air in each study region. The default exposure levels defined in the model for exposures other than inhalation (e.g., water, diet) were used with the exception of soil/dust. The average background concentration of lead in Arizona soils of 7.7 mg/kg (ADEQ, 1991) was used in the model for the soil/dust concentrations.

3.0 EXPOSURE ASSESSMENT

The purpose of the exposure assessment is to estimate the amounts of COI to which humans are exposed over various periods of time. It consists of several components that lead to the calculation of an average daily dose (ADD) for chronic exposures for each receptor evaluated, expressed as mass of substance per unit body weight per day for each COI by each pathway. The exposure assessment includes the following steps:

1. Receptor identification,
2. Identification of exposure pathways,
3. Definition of exposure calculation equations,
4. Definition of chemical-specific exposure parameters, and
5. Definition of receptor-specific exposure parameters.

3.1 Deterministic Exposure Assessment

Volume I of this report presented the method to be used to conduct the exposure assessment. The exposure assessment was designed to evaluate potential exposures to COI in each study region via the inhalation pathway by the following receptors:

1. The lifetime resident, who is assumed to be exposed as a young child aged 0-6 years, as an older child aged 6-18 years, and as an the adult, and evaluated for both potentially carcinogenic and noncarcinogenic health effects;

2. The adult resident, who is assumed to be exposed to COI only during the adult years, and evaluated for both potentially carcinogenic and noncarcinogenic health effects; and

3. The young child resident, who is assumed to be exposed to COI from 0-6 years of age, and evaluated for potential noncarcinogenic health effects.

Exposure to COI via inhalation, in terms of ADD, is estimated as follows:

TABLE 3-1
SUMMARY OF POINT EXTIMATE EXPOSURE ASSUMPTIONS
ARIZONA HAPS, ALL STUDY REGIONS, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

	RME				CTC				
	Lifetime Resident				Lifetime Resident			Young Child Resident (0-6 yr)	Adult Resident (18-39 yr)
Parameter	Young Child (0-6 yr)	Older Child (6-18 yr)	Adult (18-59 yr)	Adult Resident (18-77 yr)	Young Child (0-6 yr)	Older Child (6-18 yr)	Adult (8-21 yr)		
Parameters Used in the Inhalation Pathway									
Exposure Time (hr/24 hr)	24	24	24	24	24	24	24	24	24
Exposure Frequency (days/365 days)	365	365	365	365	350	350	350	350	350
Exposure Duration (yr)	6	12	41	59	6	12	3	6	21
Inhalation Rate (m^3/day)	9.3	16.8	18	17.6	6.6	11.5	14.8	6.6	13.5
Body Weight (kg)	14	42	70	70	14	42	70	14	70

Notes:
RME - Reasonable Maximum Exposure.
CTC - Central Tendency Case.

Source: ENSR 1995 ASSUM.WQ1 R.N.: 1

ADD (mg/kg-day) = Concentration in air (mg/m^3)
x Inhalation rate (m^3/day)
x Exposure time per day (hr/24 hr)
x Days exposed per year (day/365 days)
x Exposure duration (years)
/ Body weight (kg)
/ Averaging period (years)

The type of risk assessment described in Volume I is termed a point-estimate, or deterministic, risk assessment, because for each parameter in the exposure calculation equation, a single point-estimate was selected for that value for each receptor evaluated. For each receptor, a reasonable maximal exposure (RME) scenario and a central tendency case (CTC) scenario was defined in order to provide estimates of upper-bound and average exposures, respectively. Table 3-1 presents the point-estimate parameters selected for each receptor. Their derivation was presented in Volume I.

3.1.1 Probabilistic Exposure Assessment

To augment the deterministic, or point estimate, risk assessment for each region, a probabilistic analysis was performed to calculate distributions of risk for each receptor. While the RME and CTC point values for risk provide a rough estimate of the range of risks that may be incurred in each region, the probabilistic analysis can better define this risk range and provides the added benefit of being able to define risks associated with any percentile of risk. The probabilistic analysis was performed to evaluate potential carcinogenic risks for the lifetime resident, and to evaluate potential noncarcinogenic hazards for the young child receptor. Based on the point-estimate results presented in Section 4.0, these receptors were chosen for the probabilistic analysis because they represent the most sensitive receptors for each health endpoint.

The probabilistic analyses were performed to provide an alternative set of risk estimates for the inhalation scenarios for each of the study regions. Deterministic risk assessments require the use of single point estimates to quantify each parameter of exposure and toxicity in the equations used to calculate risk or hazard. However, for each exposure assumption, there is a range of possible and valid values. For those variables with a broad range of possible but uncertain values, current risk assessment guidance recommends the use of upper-bound values in the risk calculation. This accounts for variability and uncertainty in our knowledge base and ensures that the resulting risk estimate is likely to overstate actual risk to any one individual. Rather than debate which value is best, the same calculation can be performed using a statistical approach that uses distributions containing all valid point estimates. Instead of calculating one risk estimate, a distribution of risks can be generated.

The principal advantage of a probabilistic approach is that it uses all the information available - - it is a full information method. While deterministic methods must discard most of the evidence about a variable when picking a single value to represent a range of values, probabilistic methods use the full range of information available in the form of a probability distribution. Another important advantage of probabilistic methods is that known or suggested correlations between variables, such as body weight and inhalation rate, may be used, which reduces the compounding of conservative assumptions that occurs with traditional point estimate methods. Overestimation of potential risks increases in point estimate methods as more parameters, represented by "protective" high confidence levels, are combined. A probabilistic approach was used to estimate risks for each of the study regions in order to provide a more complete and accurate quantification of potential risks than is afforded by the point estimate assessment.

3.2 Why Probabilistic Methods Are Useful

Traditional (deterministic) risk assessment methods select single point estimates (typically upper-bound values) for each parameter of exposure, such as inhalation rate, exposure frequency, body weight, etc. A major drawback of this approach is that it does not include natural variability in human populations or uncertainty in the data. Combining a set of point values - some conservative and some typical - yields a point estimate of risk that falls at an unknown percentile of the full distribution of risk. Consequently, the degree of health-protectiveness afforded by the point estimate of risk is unknown (i.e., what percentage of the population falls below the risk estimate).

Rather than select one point estimate from a range of valid estimates, a better approach is to use the entire range of possible values in the risk calculation. This can be accomplished using probabilistic techniques, such as Monte Carlo Analysis, which use distributions for input variables and generates distributions of outputs. The resulting distribution provides a full characterization of risks and corresponding percentiles, which is considerably more useful and informative than a single value. The use of a probabilistic approach, rather than the traditional point estimate approach, is still somewhat novel in the field of environmental risk assessment. However, because of its statistical and scientific strengths, probabilistic methods are generally recognized within the risk assessment community as more accurate and complete, and are increasingly being accepted by regulatory agencies (U.S. EPA, 1992c, 1994; MA DEP, 1993; ADEQ, 1995).

The probabilistic analysis uses the same mathematical equations used in the point estimate analysis to predict potential carcinogenic and noncarcinogenic risks. Probability distributions are substituted for the point estimate input variables in the equations used to calculate exposure dose and risk. The deterministic and probabilistic approaches to exposure assessment are compared in Figure 3-1. The probabilistic analysis in this research used probability distributions

Point Estimate:

$$\frac{\text{Inhalation Rate} \times \text{Exposure Frequency} \times \text{Exposure Duration} \times \text{Air Concentrations}}{\text{Body Weight} \times \text{Averaging Period}} = \text{Exposure Estimate}$$

Probabilistic Estimate:

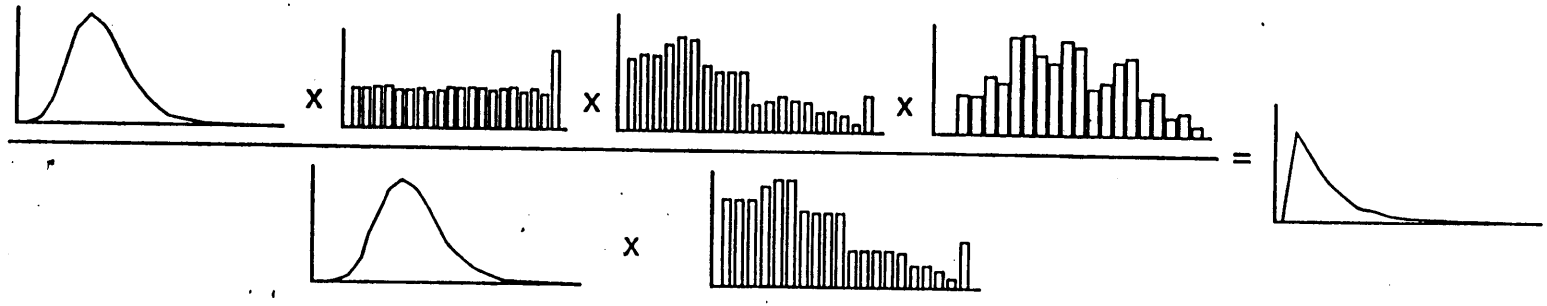


FIGURE 3-1
Comparison of Deterministic (Point) Versus a Probabilistic Estimate of Exposure

for all of the exposure variables, including exposure point concentrations and receptor-specific exposure assumptions (e.g., inhalation rate, body weight). The derivation of the exposure variable probability distributions used in this analysis is discussed in Section 3. While there is considerable uncertainty in the degree of conservatism afforded by the dose-response values, this uncertainty is harder to quantitate. Moreover, because of the length of the COI list and due to schedule constraints, this analysis used point estimate cancer slope factors (CSFs) and reference doses (RfDs) for the COI for each region.

3.2.1 Strengths of Probabilistic Methods

There are several reasons why probabilistic methods, such as Monte Carlo Analysis, are especially appropriate for assessing health risk. First, the standard definition of risk (see, e.g., Webster's Dictionary) starts: "risk is the chance [meaning, probability] of harm....." At its root, risk assessment is the assessment of probabilities of different adverse outcomes. However, the U.S. EPA routinely eliminates all notions of probability in the deterministic methods published in its standard Superfund guidance manuals (U.S. EPA, 1989a, 1991). At their core, probabilistic methods reintroduce the most basic notion of risk assessment -- probability -- into the practice of risk assessment.

Second, probabilistic methods quantify two very basic and important concepts -- variability (V) and uncertainty (U) -- at the same time as they estimate risks. In this report, we adopt the two most common definitions for the terms. Variability represents knowledge of heterogeneity in a well-characterized population, usually not reducible through further measurement or study. Uncertainty represents ignorance about a poorly-characterized phenomenon or model, sometimes reducible through further measurement or study. With probabilistic methods, risk assessors can quantify and understand the importance of V and U in a study.

A quick example helps illustrate the concept of variability. Here, variability represents knowledge of heterogeneity in a well-characterized population, usually not reducible through further measurement or study. According to various federal guidance documents, all adults weigh 70 kilograms (kg) -- even though few adults in the population weigh exactly 70 kg. It is more realistic to acknowledge that adults have different body weights, ranging from approximately 40 kg at the low end to 250 kg at the high end. It is even more realistic to acknowledge that not every body weight from 40 to 250 kg is equally likely; most adults have body weights in the 55 to 90 kg range, with fewer at the lower and higher values. Putting all these ideas together, we see that a probability distribution captures all the information about the range of possible values and the relative likelihood of the occurrence of particular values within the range. For a back-of-the-envelope calculation, one could model adult body weight with a normal distribution with an arithmetic mean of 70 kg and an arithmetic standard deviation of 10 kg. For more refined

calculations, one could model adult body weight with a lognormal distribution with two parameters selected from the literature (see, e.g., Brainard & Burmaster, 1992).

A related example helps illustrate the concept of uncertainty. Here, uncertainty represents ignorance about a poorly-characterized phenomenon or model, sometimes reducible through further measurement or study. As stated above, federal guidance documents give a single point value for adult body weight. In the previous paragraph, we developed the conceptual basis for using a probability distribution for adult body weights. The data used to fit the lognormal distribution come from a survey of adults in the United States. If a risk assessor were asked to do a probabilistic risk assessment in, say, Burma, she or he might not have data upon which to fit a distribution. In this situation, the risk assessor would have uncertainty about the variability of adult body weights in a different country. However, there are well accepted ways in statistics to start with the distribution for adult body weights in the U.S. to develop a distribution of adult body weights in Burma, as long as the risk assessor includes the uncertainty inherent in the extrapolation from country to country.

Third, probabilistic methods are "full information" methods. In a deterministic risk assessment, considerable time is spent destroying the diversity and heterogeneity inherent in information about a site and the behavior of persons using the site and instead picking a single "magic" or approved number to represent a fragment of the phenomenon. For example, in an old-style deterministic risk assessment, the analyst discards all the information known and measured about adult body weight and instead uses the patently false single value of 70 kilograms (kg) to represent adult body weights that may range from 40 kg to 250 kg. In a probabilistic risk assessment, the analyst does not destroy any information about a phenomenon -- all that is known (as V) and, to the extent possible, all that is unknown (as U) is included. Thus, a probabilistic risk assessor does not destroy information, but uses the full range of values in an appropriate statistical framework.

Fourth, probabilistic methods are inherently more understandable and acceptable to members of the public. The public can readily understand that all people do not weigh the same or drink the same amount of water every day. Furthermore, it makes common sense to the lay public to incorporate this variability into an analysis, rather than assume that all humans are identical. While lay people do not understand all the mathematics inherent in probabilistic methods, they quickly embrace the ideas inherent in variability and uncertainty.

3.2.2 Mechanics of Probabilistic Risk Assessment

In a standard or deterministic risk assessment, the risk assessor builds a spreadsheet to estimate the risk through numerical computation. To simplify the discussion, consider a formula of the general form:

$$\text{Risk} = f(X, Y, Z),$$

where Risk is a function of only three input variables, X, Y, and Z. In a deterministic risk assessment, the analyst picks a single number for X, a single number for Y, and a single number for Z. After the risk assessor inputs the function f(X, Y, Z), the spreadsheet calculates the single value for Risk in a single computation.

In a probabilistic risk assessment, the basic risk equation remains the same:

$$\underline{\text{Risk}} = f(\underline{X}, \underline{Y}, \underline{Z}).$$

However, each of the variables now has double-underscores to indicate that each is a random variable, i.e., a variable that can take different values within a range of values as described by a probability distribution. In a probabilistic risk assessment, the analyst picks not just a single number for each input but a whole distribution of values that represent the variability and/or the uncertainty in the input. After the risk assessor inputs the function f(X, Y, Z), the spreadsheet -- along with some auxiliary software -- computes a full distribution for Risk by completing thousands of iterations of the same basic calculation. In the first iteration, the computer picks a single random value from each of the three input distributions, X, Y, and Z, and computes a single value of Risk. In the second iteration, the computer picks a different random value from each of the three input distributions and computes a different single value of Risk. As the computer calculates additional iterations of this calculation, the software stores each of the values and displays them as a histogram of Risk. After many iterations, the histogram of Risk values converges to the distribution of Risk sought by the analyst.

3.2.3 Computation Methods

For this analysis, spreadsheets were constructed using the standard mathematical formulas for evaluating health risk from ingestion of and dermal contact with soil. The computational accuracy of the spreadsheets were verified by reproducing the point estimates of risk developed by ENSR in the deterministic baseline assessment. Developed in Lotus 1-2-3, Release 5.0, and Palisade's @Risk (version 3.1a) on IBM compatible computer, the spreadsheets accept a combination of point values and distributions as inputs for air concentrations and exposure

parameters. Using the spreadsheets and the probabilistic software, @Risk, output distributions of carcinogenic risk and noncarcinogenic hazard index were generated. Utilizing Latin Hypercube sampling, 5000 iterations were run for each spreadsheet, which is sufficient to provide solid numerical stability to the risk distributions.

3.3 Receptor-specific Exposure Parameter Distributions

Attachment F-2 presents the specific exposure parameter distributions used in this risk assessment. Each of the exposure parameters is discussed below. Representative distributions are shown in Figure 3-2.

3.3.1 Body Weight

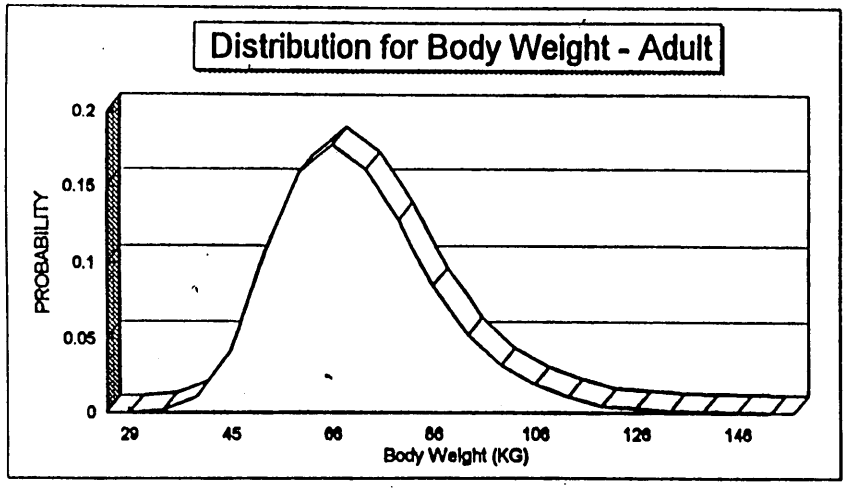
Data on body weight for each of the receptor age groups were obtained from the U.S. Public Health Service in the National Health and Nutrition Examination Survey (NHANES II). The data were used to define a lognormal distribution for each receptor age group. Because of the large amount of data on body weight for U.S. residents, the body weight distribution represents the variability, rather than the uncertainty, in this parameter.

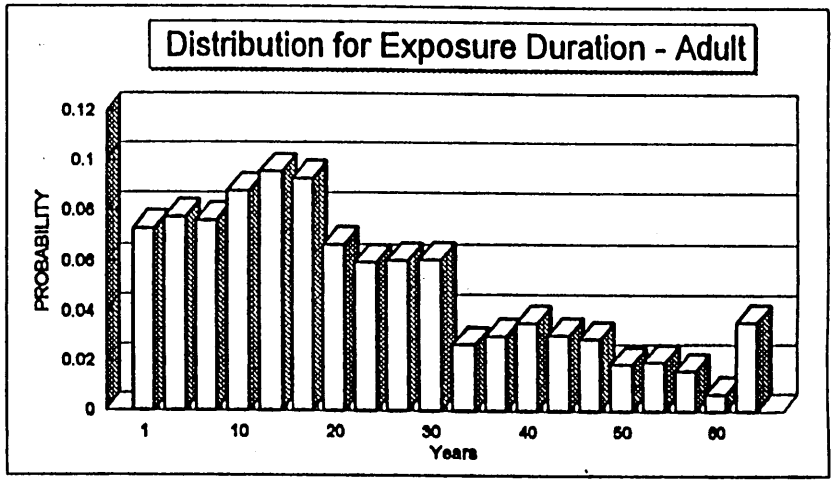
3.3.2 Inhalation Rate

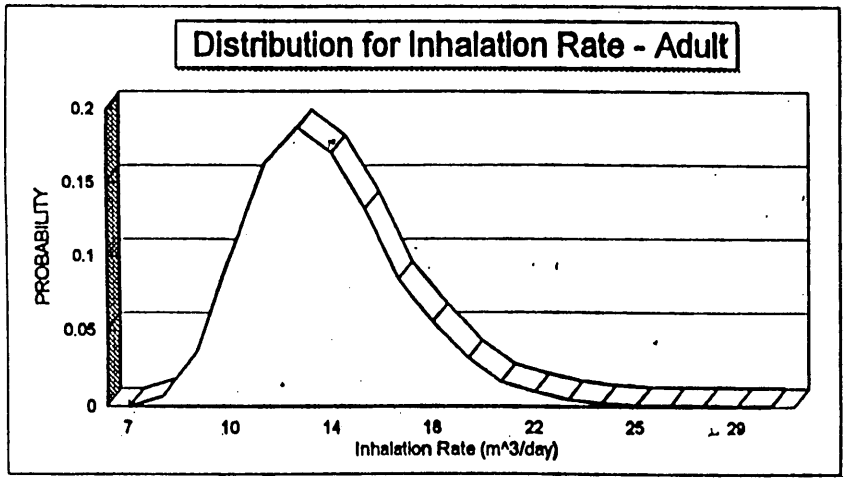
As stated in Volume I, inhalation rates in this risk assessment are based on basal metabolic rate rather than activity periods. As for the point estimates, the distributions for inhalation rate are based on the distributions presented by Finley et al. (1994), which are based on the basal metabolic rate models of Layton (1993). Basal metabolic rate as defined by Layton is a function of body weight, and this interdependence between inhalation rate and body weight has been incorporated into the distribution, as described in Attachment B. Because this parameter is based on a body weight distribution, the inhalation rate distribution represents variability rather than uncertainty in this parameter. However, there is still uncertainty about whether the correct relationship between body weight, basal metabolic rate and inhalation rate has been used, and this uncertainty cannot be quantified in this analysis.

3.3.3 Exposure Time

As with the point estimate approach, it is assumed that each receptor is exposed to COI in air 24 hours each day for every day spent in the study region. This is the best estimate for this parameter for both the deterministic and probabilistic approaches. Although there is likely to be little variability in this parameter on a population wide basis, the uncertainty in defining that







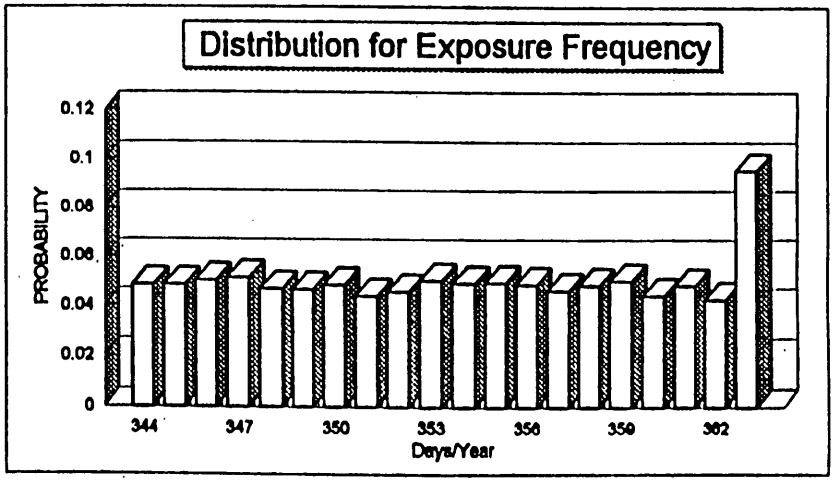


Figure 3-2. Representative Distributions for Adult Lifetime Resident Exposure Parameters

variability is large. However, the variability in this parameter is at least partially captured in the distribution of the next exposure parameter, exposure frequency.

3.3.4 Exposure Frequency

Because of a lack of data on daily movement of residents into and out of the study regions, the distribution for exposure frequency, or days exposed per year, is based on best professional judgement. It is assumed an individual exits the study regions in daily, not hourly increments. It was also assumed that a receptor is equally likely to be out of the study region between 1 and 21 days per year. Although it is understood that Arizona enjoys an increase in population during the winter months due to the immigration of generally elderly persons from colder climates, this type of a receptor was not specifically addressed in this risk assessment. Therefore, the use of this exposure distribution presents a source of uncertainty in the risk assessment.

3.3.5 Exposure Duration

Exposure duration distributions were defined as described in Volume I for the point estimates using a PC-based version of the "ROPSIM" model developed for the U.S. EPA (U.S.EPA, 1992). The point estimates for the RME and CTC cases were selected as the 95th and 50th percentiles of this distribution, respectively. For the probabilistic analysis, the entire distribution was used.

3.3.6 Averaging Period

As described in Volume I, the averaging period is defined differently for the evaluation of potentially carcinogenic and noncarcinogenic effects. In the derivation of the CSF values in the toxicity assessment, the assumption of a 70 year life-span is used. Therefore, for both the deterministic and the probabilistic analyses, a point-estimate of averaging period for the evaluation of potential carcinogenic effects of 70 years is used.

The averaging period for the evaluation of noncarcinogenic effects is equal to the exposure duration period. Therefore, the probabilistic assessment for noncarcinogenic effects has been set such that the same value selected for exposure duration is selected for averaging period for each model run.

3.4 Chemical-specific Exposure Parameters

Chemical-specific exposure parameters include the air concentration data and absorption adjustment factors (AAFs). Because of the long list of COI and schedule constraints, AAFs were assumed to have a point-value of 1.0 for each COI for both the deterministic and probabilistic analyses. This assumption may result in the over- or under-estimation of exposure and risk; however, for most of the COI, the assumption of 1.0 is appropriate.

3.4.1 Air Concentration

The purpose of this assessment is to evaluate the potential for the occurrence of adverse health effects in the population of each of the study regions. The results of the atmospheric simulation model have provided estimates of annual average air concentrations for each grid cell in each modeled study region. As demonstrated in Sections 4.0 through 6.0 of Volume 2 of the HAPs research program final report, there is quite a bit of variability in predicted air concentrations between grid cells. Moreover, there are differences in population between each grid cell. Although each person in the population resides within a defined grid cell, people are mobile to varying degrees. Some live in one area, shop in another area, and work or attend school in a third. Others, such as the elderly, may spend all of their time in one relatively small area, and yet others, such as sales or delivery workers, may travel extensively within a region. Because this analysis is intended to evaluate typical exposures within each region, and because it is assumed that people move around within a region, a distribution of air concentrations was developed for each modeled region (Phoenix, Tucson, and Casa Grande). As described in Section 7.0 of Volume 2, air monitoring data were used to evaluate potential exposure and risk in Payson and therefore, point estimates of air concentrations were used in the probabilistic analysis there.

Although for some chemicals, grid cells with higher air concentrations are also grid cells with higher populations, there is not a strict correlation. However, a randomly chosen individual in a population is more likely to be exposed to an air concentration associated with a highly populated grid cell than a very low populated grid cell, whether or not that air concentration is low or high. This association has served as the basis for the development of a probability distribution of air concentrations for each COI in each region.

The distribution of COI concentrations for each region was developed as follows, and was conducted separately for each COI in each modeled region. Based on the per grid results of the atmospheric simulation model, the minimum and maximum air concentrations of that COI were defined. The range of air concentrations between the minimum and maximum were divided up into discrete and sequential concentration "bins". The air concentration of each grid cell was

then assigned to its appropriate bin: if the population of that grid cell was one, the air concentration was entered into that bin once; if the population of that grid cell was 2000, the air concentration was entered into that bin 2000 times, etc. When all grid cells are so entered, the distribution was divided by the total population. Therefore, the probability of exposure to an air concentration encompassed by a bin is related to the population residing in grid cells within that concentration range.

For the deterministic analysis, the 95th and 50th percentile values were used as the point-estimate air concentrations for the RME and CTC scenarios, respectively. The point-estimate air concentrations for the RME and CTC scenarios are presented in Tables 3-2 through 3-4 for each of the modeled regions. Because only air monitoring data are used to evaluate potential exposures in Payson, the same air concentrations are used to evaluate both RME and CTC exposures (Table 3-5).

3.5 Multi-Pathway Evaluation

Phoenix was evaluated for whether or not a multi-pathway analysis of risks due to deposition onto soil was necessary. The air concentration results were used in conjunction with a screening level particle deposition model (U.S. EPA, 199_) to determine whether particulate HAPs were contributing significantly to regional soil concentrations. The particulate HAPs in the Phoenix study region consist of metals and PAH. The emissions profiles for each metal were evaluated to determine whether the emissions were of geologic or anthropogenic origin, e.g., whether barium in air was due to the resuspension of soil dust such as road or construction activity dust (geologic origin) or due to some commercial or industrial process (non-geologic origin). If there were both types of sources, that fraction of the air concentration due to anthropogenic sources was evaluated. The COI listed in Table 3-2 are those particulate HAPs in the Phoenix region that have all or some portion of their emissions due to non-geologic sources. Manganese was determined to be of geologic origin. The predicted RME soil concentration is based on the deposition that would occur from the annual average air concentration for 95% of the population over the course of 29.5 years or 10.5 years for the RME and CTC scenarios, respectively.

The deposition rate of each COI was estimated by multiplying the atmospheric concentration by a deposition velocity. This rate was then multiplied by the duration of deposition (29.5 or 10.5 years) to calculate total deposition, and it was assumed that the deposited amount was distributed uniformly throughout the top 5 cm of the soil. This approach overestimates the soil concentrations, because some of the deposited material would likely be leached out of this top soil layer over the assumed deposition periods. The deposition velocities were estimated based on an algorithm presented by Zannetti (1990), which utilizes a resistance approach that considers surface characteristics, atmospheric stability, wind velocity, particle diameter and article density.

TABLE 3-2
AMBIENT AIR CONCENTRATIONS - CTC AND RME SCENARIO
ARIZONA HAPS, PHOENIX, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	CTC Ambient Air Concentration * (mg/m^3)	RME Ambient Air Concentration* (mg/m^3)
ARSENIC	2.00E-06	2.00E-06
BARIUM	3.30E-05	3.30E-05
CADMIUM	4.00E-06	4.00E-06
CHROMIUM VI	1.58E-07	4.93E-07
MANGANESE	2.10E-05	2.10E-05
MERCURY	4.98E-06	1.55E-05
NICKEL	2.00E-06	2.00E-06
ACETALDEHYDE	4.78E-03	7.64E-03
ACROLEIN	2.52E-05	4.90E-05
ACRYLONITRILE	8.57E-06	2.12E-05
BENZENE	5.93E-03	1.14E-02
1,3-BUTADIENE	5.23E-04	9.79E-04
CARBON TETRACHLORIDE	7.50E-04	7.50E-04
CHLOROFORM	1.57E-04	4.47E-04
1,4-DICHLOROBENZENE	1.90E-04	6.16E-04
DICHLOROMETHANE	3.75E-03	6.86E-03
ETHYLENE DIBROMIDE	2.87E-06	1.13E-05
ETHYLENE OXIDE	4.65E-05	8.78E-05
FORMALDEHYDE	4.00E-03	7.73E-03
TETRACHLOROETHYLENE	8.28E-04	1.81E-03
TRICHLOROETHYLENE	1.12E-04	4.34E-04
TRIMETHYLBENZENE (TOTAL)	4.66E-04	8.53E-04
VINYL CHLORIDE	6.31E-06	1.15E-05
BENZO(A)PYRENE	2.44E-07	7.55E-07

Notes:
CTC - Central Tendency Case.
RME - Reasonable Maximum Exposure.
* - Concentration based on population-weighted values from the atmospheric simulation model.

Source: ENSR 1995
DATAPRT.WQ1
R.N.:6

TABLE 3-3
AMBIENT AIR CONCENTRATIONS - CTC AND RME SCENARIO
ARIZONA HAPS, TUCSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	CTC Ambient Air Concentration* (mg/m^3)	RME Ambient Air Concentration* (mg/m^3)
ARSENIC	1.40E-05	1.40E-05
BARIUM	4.00E-05	4.00E-05
CADMIUM	5.00E-06	5.00E-06
CHROMIUM (HEXAVALENT)	2.05E-08	2.18E-08
MANGANESE	7.00E-06	7.00E-06
NICKEL	1.00E-06	1.00E-06
ACETALDEHYDE	3.15E-03	4.18E-03
ACROLEIN	1.31E-05	2.07E-05
BENZENE	3.33E-03	5.29E-03
1,3-BUTADIENE	3.48E-04	5.64E-04
CARBON TETRACHLORIDE	7.50E-04	7.50E-04
CHLOROBENZENE	2.27E-05	2.61E-05
CHLOROFORM	1.72E-04	3.52E-04
1,4-DICHLOROBENZENE	3.59E-05	4.50E-05
DICHLOROMETHANE	5.97E-03	6.77E-03
ETHYLENE OXIDE	1.29E-05	2.28E-05
FORMALDEHYDE	8.13E-04	1.33E-03
N-HEXANE	4.19E-03	6.62E-03
TETRACHLOROETHYLENE	5.47E-04	9.28E-04
TOLUENE	6.17E-03	9.29E-03
TRICHLOROETHYLENE	2.33E-04	2.68E-04
TRIMETHYLBENZENE (TOTAL)	1.78E-05	2.38E-05
BENZO(A)PYRENE	3.24E-07	7.27E-07

Notes:
CTC - Central Tendency Case.
RME - Reasonable Maximum Exposure.
* - Concentration based on population-weighted values from the atmospheric simulation model.

Source: ENSR 1995
DATAPRT.WQ1
R.N.:5

TABLE 3-4
AMBIENT AIR CONCENTRATIONS - CTC AND RME SCENARIO
ARIZONA HAPS, CASA GRANDE, AIR
HUMAN HEALTH RISK ASSESSMENT

Compound	CTC Ambient Air Concentration* (mg/m^3)	RME Ambient Air Concentration* (mg/m^3)
ARSENIC	1.97E-07	3.07E-07
BARIUM	2.01E-05	3.81E-05
CADMIUM	4.26E-07	8.10E-07
CHROMIUM (HEXAVALENT)	3.68E-07	7.00E-07
MANGANESE	1.60E-05	2.99E-05
NICKEL	1.57E-06	2.90E-06
ACETALDEHYDE	2.07E-03	2.61E-03
ACROLEIN	1.37E-05	2.75E-05
AMMONIA	6.38E-04	5.56E-03
BENZENE	1.32E-03	2.15E-03
1,3-BUTADIENE	1.51E-04	2.72E-04
CARBON TETRACHLORIDE	7.50E-04	7.50E-04
CHLOROFORM	1.15E-04	2.08E-04
1,4-DICHLOROBENZENE	4.01E-05	6.80E-05
DICHLOROMETHANE	2.96E-03	3.24E-03
FORMALDEHYDE	6.63E-04	1.20E-03
TOLUENE	3.48E-03	9.52E-03
TRICHLOROETHYLENE	4.78E-04	8.20E-04
TRIMETHYLBENZENE (TOTAL)	2.90E-06	4.59E-06
BENZO(A)PYRENE	8.45E-07	2.49E-07
CHLORPYRIFOS	3.74E-05	5.69E-05
DISULFOTON	1.80E-06	6.20E-06
METHYL PARATHION	2.10E-05	7.70E-05
PENTACHLORONITROBENZENE	2.05E-06	6.01E-06
TRIFLURALIN	4.76E-06	1.76E-05

Notes:
CTC - Central Tendency Case.
RME - Reasonable Maximum Exposure.
* - Concentration based on population-weighted values from the atmospheric simulation model.

Source: ENSR 1995
DATAPRT.WQ1
R.N.:6

TABLE 4-25
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, PAYSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	7.33E-07	8.83E-07	1.94E-06	3.55E-06	2.73E-06	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	6.99E-03	8.41E-03	1.85E-02	3.39E-02	6.87E-02	2.60E-02
CADMIUM	7.24E-07	8.72E-07	1.92E-06	3.51E-06	2.70E-06	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	6.62E-02	7.97E-02	1.75E-01	3.21E-01	6.51E-01	2.46E-01
NICKEL	3.22E-08	3.88E-08	8.51E-08	1.56E-07	1.20E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	1.15E-06	1.39E-06	3.05E-06	5.60E-06	4.30E-06	6.92E-02	8.33E-02	1.83E-01	3.36E-01	6.80E-01	2.58E-01
ACROLEIN	NA	NA	NA	NA	NA	2.70E-01	3.25E-01	7.14E-01	1.31E+00	2.66E+00	1.01E+00
BENZENE	4.24E-06	5.11E-06	1.12E-05	2.06E-05	1.58E-05	1.02E-01	1.22E-01	2.69E-01	4.92E-01	9.98E-01	3.78E-01
1,3-BUTADIENE	1.27E-05	1.53E-05	3.37E-05	6.18E-05	4.74E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROFORM	5.23E-07	6.3E-07	1.38E-06	2.54E-06	1.95E-06	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1.13E-07	1.36E-07	2.98E-07	5.46E-07	4.19E-07	1.45E-05	1.75E-05	3.84E-05	7.05E-05	1.43E-04	5.41E-05
DICHLOROMETHANE	3.98E-07	4.79E-07	1.05E-06	1.93E-06	1.48E-06	3.43E-04	4.13E-04	9.07E-04	1.66E-03	3.37E-03	1.28E-03
FORMALDEHYDE	2.34E-06	2.82E-06	6.19E-06	1.13E-05	8.71E-06	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	2.82E-03	3.39E-03	7.45E-03	1.37E-02	2.77E-02	1.05E-02
TETRACHLOROETHYLENE	6.99E-08	8.42E-08	1.85E-07	3.39E-07	2.60E-07	3.64E-04	4.38E-04	9.62E-04	1.76E-03	3.58E-03	1.35E-03
TOLUENE	NA	NA	NA	NA	NA	4.47E-03	5.39E-03	1.18E-02	2.17E-02	4.40E-02	1.66E-02
TRICHLOROETHYLENE	2E-07	2.41E-07	5.3E-07	9.71E-07	7.45E-07	NA	NA	NA	NA	NA	NA
BENZO(A)ANTHRACENE	2.78E-08	3.35E-08	7.37E-08	1.35E-07	1.04E-07	7.43E-05	8.95E-05	1.97E-04	3.60E-04	7.31E-04	2.77E-04
BENZO(A)PYRENE	2.38E-07	2.86E-07	6.29E-07	1.15E-06	8.85E-07	6.35E-05	7.64E-05	1.68E-04	3.08E-04	6.24E-04	2.36E-04
BENZO(B+J+K)FLUORANTHENE	3.56E-08	4.29E-08	9.43E-08	1.73E-07	1.33E-07	9.51E-05	1.15E-04	2.52E-04	4.61E-04	9.35E-04	3.54E-04
CHRYSENE	3.07E-10	3.7E-10	8.12E-10	1.49E-09	1.14E-09	8.20E-05	9.87E-05	2.17E-04	3.97E-04	8.06E-04	3.05E-04
DIBENZ(A,H+A,C)ANTHRACENE	2.77E-08	3.33E-08	7.32E-08	1.34E-07	1.03E-07	7.41E-06	8.92E-06	1.96E-05	3.59E-05	7.28E-05	2.76E-05
INDENO(1,2,3-CD)PYRENE	1.29E-08	1.56E-08	3.42E-08	6.28E-08	4.82E-08	3.45E-05	4.16E-05	9.14E-05	1.68E-04	3.40E-04	1.29E-04
ACENAPHTHENE	NA	NA	NA	NA	NA	7.39E-04	8.90E-04	1.95E-03	3.58E-03	7.26E-03	2.75E-03
ACENAPHTHYLENE	NA	NA	NA	NA	NA	8.92E-04	1.07E-03	2.36E-03	4.33E-03	8.77E-03	3.32E-03
ANTHRACENE	NA	NA	NA	NA	NA	1.87E-04	2.25E-04	4.93E-04	9.05E-04	1.83E-03	6.94E-04
BENZO(B)CHRYSENE	NA	NA	NA	NA	NA	7.94E-06	9.56E-06	2.10E-05	3.85E-05	7.81E-05	2.96E-05
BENZO(G,H,I)PERYLENE	NA	NA	NA	NA	NA	5.94E-05	7.15E-05	1.57E-04	2.88E-04	5.84E-04	2.21E-04
BIPHENYL	NA	NA	NA	NA	NA	9.18E-04	1.11E-03	2.43E-03	4.45E-03	9.03E-03	3.42E-03
1,2-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	8.54E-04	1.03E-03	2.26E-03	4.14E-03	8.39E-03	3.18E-03
1,3+1,6+1,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	3.35E-03	4.03E-03	8.85E-03	1.62E-02	3.29E-02	1.25E-02
1,4+1,5+2,3-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.00E-03	1.21E-03	2.66E-03	4.87E-03	9.88E-03	3.74E-03
1,8-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	2.19E-05	2.63E-05	5.78E-05	1.06E-04	2.15E-04	8.14E-05
2,6+2,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.53E-03	1.84E-03	4.05E-03	7.42E-03	1.50E-02	5.69E-03
FLUORANTHENE	NA	NA	NA	NA	NA	3.44E-04	4.15E-04	9.11E-04	1.67E-03	3.39E-03	1.28E-03
FLUORENE	NA	NA	NA	NA	NA	7.74E-04	9.32E-04	2.05E-03	3.75E-03	7.61E-03	2.88E-03
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	7.74E-03	9.32E-03	2.05E-02	3.75E-02	7.61E-02	2.88E-02
2-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.66E-02	2.00E-02	4.40E-02	8.07E-02	1.64E-01	6.19E-02
NAPHTHALENE	NA	NA	NA	NA	NA	2.82E-02	3.40E-02	7.46E-02	1.37E-01	2.77E-01	1.05E-01
PHENANTHRENE	NA	NA	NA	NA	NA	1.20E-03	1.45E-03	3.18E-03	5.83E-03	1.18E-02	4.48E-03
PYRENE	NA	NA	NA	NA	NA	2.78E-04	3.35E-04	7.35E-04	1.35E-03	2.73E-03	1.03E-03
TOTAL	2.59E-05	3.12E-05	6.84E-05	1.25E-04	9.63E-05	6.76E-01	8.14E-01	1.79E+00	3.28E+00	6.65E+00	2.52E+00

Notes:

Source: ENSR 1995

TOTAL.WQ1

R.N.: 3

80% of the current risk estimates, and future HI are approximately 95% of the current values for both the RME and CTC scenarios. Therefore, total risks and hazards due to HAPs are expected to decrease in the future in the Casa Grande study region.

4.4 Payson Study Region

Table 4-25 presents the ELCR and HI results for the RME scenario for the Payson study region based on estimates of current emissions. Risk and hazard estimates are presented for each COI for the total lifetime resident (and for each age group) and the adult resident. In addition, hazard estimates are presented for the young child resident. An "NA" in the table indicates that a dose-response value with which to evaluate either carcinogenic or noncarcinogenic health effects was unavailable.

A comparison of the RME ELCR values for the two receptors evaluated in Table 4-25 indicates that the ELCR value is higher for the lifetime resident, i.e., the resident aged 0-59 years, than for the adult resident, i.e., the resident assumed to be an adult for the entire 59 year exposure period. Therefore, the following discussion will focus on the most sensitive receptor, the lifetime resident.

The risk estimate for the RME scenario is 1.25E-04, which is equivalent to 125 excess cancer cases (i.e., in excess of cancer cases expected to naturally occur) in a population of one million over a lifetime of 70 years. Based on the current population estimate for Payson of 6,667 people, that is equivalent to 0.8 excess cancer cases over a 70 year period, or approximately 0.01 excess cancer cases per year. For comparison, the current U.S. "background" cancer rate due to all causes is approximately 1 in 4, or 250,000 in a population of one million.

Table 4-26 presents the same information for the CTC scenario and shows that the ELCR for the CTC scenario for the Casa Grande study region is approximately one-third of that for the RME scenario.

A comparison of the RME HI values for the three receptor scenarios evaluated for noncarcinogenic effects, as shown in Table 4-25, indicates that the HI value is higher for the young child resident, i.e., the resident aged 0-6 years, than for the lifetime or adult residents. Therefore, the following discussion of noncarcinogenic effects will focus on the most sensitive receptor, the young child resident.

The HI for the RME young child resident of 6.65 is above the target value of 1.0, indicating that a toxicity endpoint analysis should be conducted. The HI for the CTC scenario is approximately two-thirds that of the RME scenario. The toxicity endpoint analyses for noncarcinogenic effects

for the young child receptor for the RME and the CTC scenarios are presented in Tables 4-27 and 4-28, respectively. For both the RME and CTC cases, only the HI for respiratory effects is above 1. An HI value above one only indicates that the predicted threshold value has been exceeded; it does not indicate any probability that adverse health effects will occur, or to what extent they may occur.

The IEUBK results for the RME case for lead for the Payson study region are presented in Table 4-29. As shown, none of the population are expected to exhibit a blood lead concentration in excess of 10 μg/dL, the regulatory target value, as a result of exposure to ambient concentrations of lead in air.

Table 4-30 presents a comparison of the probabilistic and deterministic risk results based on the current emissions estimates. The comparison is presented graphically in Figures 4-7 and 4-8 for the ELCR and HI, respectively. The results indicate that the point estimates are fairly good predictors of the range of estimates provided by the probabilistic analysis. For comparison, the 95th percentile risk estimate is 1.20E-04, while the RME estimate is 1.25E-04. The close agreement for the Payson study region is due in part to the fact that only point estimates of air concentrations rather than distributions were available for the Payson study region.

Turning to the future, Tables 4-31 and 4-32 present the ELCR and HI results for the RME and CTC scenarios, respectively, based on estimates of future emissions. Risk and hazard estimates are presented for each COI for each receptor. For the future scenario, risks are approximately 73% of the current risk estimates, and future HI are approximately 80% of the current values for both the RME and CTC scenarios. Therefore, total risks and hazards due to HAPs are expected to decrease in the future in the Payson study region.

4.5 Summary

Tables 4-33 and 4-34 summarize the point and probabilistic estimates of risk and hazard, respectively. Despite the geographic and economic differences between the study regions, there is only a three to four fold difference in the risk estimates between the highest and lowest risk regions.

TABLE 4-26
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, PAYSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	4.99E-07	5.79E-07	1.12E-07	1.19E-06	7.14E-07	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	1.34E-02	1.55E-02	3.00E-03	3.19E-02	4.68E-02	1.91E-02
CADMIUM	4.93E-07	5.73E-07	1.11E-07	1.18E-06	7.06E-07	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.27E-01	1.47E-01	2.84E-02	3.02E-01	4.43E-01	1.81E-01
NICKEL	2.19E-08	2.54E-08	4.91E-09	5.23E-08	3.14E-08	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	7.85E-07	9.12E-07	1.76E-07	1.87E-06	1.12E-06	1.32E-01	1.54E-01	2.97E-02	3.16E-01	4.63E-01	1.89E-01
ACROLEIN	NA	NA	NA	NA	NA	5.16E-01	6.00E-01	1.16E-01	1.23E+00	1.81E+00	7.39E-01
BENZENE	2.89E-06	3.35E-06	6.48E-07	6.89E-06	4.13E-06	1.94E-01	2.25E-01	4.35E-02	4.63E-01	6.79E-01	2.78E-01
1,3-BUTADIENE	8.67E-06	1.01E-05	1.94E-06	2.07E-05	1.24E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROFORM	3.56E-07	4.13E-07	7.98E-08	8.49E-07	5.09E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	7.67E-08	8.91E-08	1.72E-08	1.83E-07	1.10E-07	2.78E-05	3.23E-05	6.23E-06	6.63E-05	9.72E-05	3.98E-05
DICHLOROMETHANE	2.71E-07	3.14E-07	6.07E-08	6.46E-07	3.87E-07	6.55E-04	7.61E-04	1.47E-04	1.56E-03	2.29E-03	9.39E-04
FORMALDEHYDE	1.59E-06	1.85E-06	3.57E-07	3.80E-06	2.28E-06	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	5.38E-03	6.25E-03	1.21E-03	1.28E-02	1.88E-02	7.71E-03
TETRACHLOROETHYLENE	4.76E-08	5.53E-08	1.07E-08	1.14E-07	6.81E-08	6.96E-04	8.08E-04	1.56E-04	1.66E-03	2.43E-03	9.96E-04
TOLUENE	NA	NA	NA	NA	NA	8.55E-03	9.93E-03	1.92E-03	2.04E-02	2.99E-02	1.22E-02
TRICHLOROETHYLENE	1.36E-07	1.58E-07	3.05E-08	3.25E-07	1.95E-07	NA	NA	NA	NA	NA	NA
BENZO(A)ANTHRACENE	1.89E-08	2.2E-08	4.25E-09	4.52E-08	2.71E-08	1.42E-04	1.65E-04	3.19E-05	3.39E-04	4.97E-04	2.03E-04
BENZO(A)PYRENE	1.62E-07	1.88E-07	3.63E-08	3.86E-07	2.32E-07	1.21E-04	1.41E-04	2.72E-05	2.90E-04	4.25E-04	1.74E-04
BENZO(B+J+K)FLUORANTHENE	2.43E-08	2.82E-08	5.44E-09	5.79E-08	3.47E-08	1.82E-04	2.11E-04	4.08E-05	4.34E-04	6.36E-04	2.60E-04
CHRYSENE	2.09E-10	2.43E-10	4.69E-11	4.99E-10	2.99E-10	1.57E-04	1.82E-04	3.51E-05	3.74E-04	5.48E-04	2.24E-04
DIBENZ(A,H+A,C)ANTHRACENE	1.88E-08	2.19E-08	4.22E-09	4.50E-08	2.70E-08	1.42E-05	1.65E-05	3.18E-06	3.38E-05	4.96E-05	2.03E-05
INDENO(1,2,3-CD)PYRENE	8.81E-09	1.02E-08	1.98E-09	2.10E-08	1.26E-08	6.61E-05	7.67E-05	1.48E-05	1.58E-04	2.31E-04	9.46E-05
ACENAPHTHENE	NA	NA	NA	NA	NA	1.41E-03	1.64E-03	3.17E-04	3.37E-03	4.94E-03	2.02E-03
ACENAPHTHYLENE	NA	NA	NA	NA	NA	1.71E-03	1.98E-03	3.82E-04	4.07E-03	5.97E-03	2.44E-03
ANTHRACENE	NA	NA	NA	NA	NA	3.57E-04	4.14E-04	8.00E-05	8.51E-04	1.25E-03	5.11E-04
BENZO(B)CHRYSENE	NA	NA	NA	NA	NA	1.52E-05	1.76E-05	3.40E-06	3.62E-05	5.31E-05	2.17E-05
BENZO(G,H,I)PERYLENE	NA	NA	NA	NA	NA	1.14E-04	1.32E-04	2.55E-05	2.71E-04	3.97E-04	1.63E-04
BIPHENYL	NA	NA	NA	NA	NA	1.76E-03	2.04E-03	3.94E-04	4.19E-03	6.15E-03	2.51E-03
1,2-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.63E-03	1.90E-03	3.66E-04	3.89E-03	5.71E-03	2.34E-03
1,3+1,6+1,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	6.40E-03	7.43E-03	1.44E-03	1.53E-02	2.24E-02	9.16E-03
1,4+1,5+2,3-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.92E-03	2.23E-03	4.31E-04	4.58E-03	6.72E-03	2.75E-03
1,8-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	4.18E-05	4.86E-05	9.37E-06	9.97E-05	1.46E-04	5.99E-05
2,6+2,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	2.92E-03	3.40E-03	6.56E-04	6.98E-03	1.02E-02	4.19E-03
FLUORANTHENE	NA	NA	NA	NA	NA	6.58E-04	7.65E-04	1.48E-04	1.57E-03	2.30E-03	9.43E-04
FLUORENE	NA	NA	NA	NA	NA	1.48E-03	1.72E-03	3.32E-04	3.53E-03	5.18E-03	2.12E-03
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.48E-02	1.72E-02	3.32E-03	3.53E-02	5.18E-02	2.12E-02
2-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	3.18E-02	3.69E-02	7.13E-03	7.59E-02	1.11E-01	4.55E-02
NAPHTHALENE	NA	NA	NA	NA	NA	5.39E-02	6.26E-02	1.21E-02	1.29E-01	1.89E-01	7.72E-02
PHENANTHRENE	NA	NA	NA	NA	NA	2.30E-03	2.67E-03	5.16E-04	5.49E-03	8.05E-03	3.29E-03
PYRENE	NA	NA	NA	NA	NA	5.31E-04	6.17E-04	1.19E-04	1.27E-03	1.86E-03	7.60E-04
TOTAL	1.76E-05	2.05E-05	3.95E-06	4.20E-05	2.52E-05	1.29E+00	1.50E+00	2.90E-01	3.08E+00	4.52E+00	1.85E+00

Notes:

Source: ENSR 1995 TOTAL.WQ1 R.N.: 3

TABLE 4-16
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, TUCSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	8.14E-06	9.45E-06	1.82E-06	1.94E-05	1.17E-05	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	4.74E-02	5.50E-02	1.06E-02	1.13E-01	1.66E-01	6.78E-02
CADMIUM	1.55E-06	1.80E-06	3.48E-07	3.70E-06	2.22E-06	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	8.45E-02	9.81E-02	1.89E-02	2.01E-01	2.96E-01	1.21E-01
NICKEL	4.32E-08	5.02E-08	9.70E-09	1.03E-07	6.19E-08	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	8.69E-07	1.01E-06	1.95E-07	2.07E-06	1.24E-06	1.46E-01	1.70E-01	3.28E-02	3.49E-01	5.12E-01	2.10E-01
ACROLEIN	NA	NA	NA	NA	NA	2.94E-01	3.42E-01	6.60E-02	7.02E-01	1.03E+00	4.21E-01
BENZENE	3.01E-06	3.50E-06	6.76E-07	7.19E-06	4.31E-06	2.03E-01	2.35E-01	4.54E-02	4.83E-01	7.09E-01	2.90E-01
1,3-BUTADIENE	9.84E-06	1.14E-05	2.21E-06	2.35E-05	1.41E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROBENZENE	NA	NA	NA	NA	NA	7.10E-04	8.25E-04	1.59E-04	1.69E-03	2.48E-03	1.02E-03
CHLOROFORM	6.54E-07	7.60E-07	1.47E-07	1.56E-06	9.36E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	6.89E-08	8.01E-08	1.55E-08	1.64E-07	9.87E-08	2.50E-05	2.90E-05	5.60E-06	5.96E-05	8.74E-05	3.58E-05
DICHLOROMETHANE	4.16E-07	4.83E-07	9.33E-08	9.93E-07	5.96E-07	1.01E-03	1.17E-03	2.26E-04	2.40E-03	3.53E-03	1.44E-03
ETHYLENE OXIDE	2.27E-07	2.64E-07	5.10E-08	5.43E-07	3.26E-07	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	1.37E-06	1.59E-06	3.08E-07	3.27E-06	1.97E-06	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	8.91E-03	1.04E-02	2.00E-03	2.13E-02	3.12E-02	1.28E-02
TETRACHLOROETHYLENE	2.89E-08	3.36E-08	6.48E-09	6.90E-08	4.14E-08	4.23E-04	4.91E-04	9.48E-05	1.01E-03	1.48E-03	6.05E-04
TOLUENE	NA	NA	NA	NA	NA	7.09E-03	8.23E-03	1.59E-03	1.69E-02	2.48E-02	1.01E-02
TRICHLOROETHYLENE	3.03E-08	3.53E-08	6.81E-09	7.24E-08	4.35E-08	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	1.66E-03	1.93E-03	3.72E-04	3.96E-03	5.81E-03	2.38E-03
BENZO(A)PYRENE	3.06E-08	3.56E-08	6.86E-09	7.30E-08	4.38E-08	2.30E-05	2.67E-05	5.15E-06	5.48E-05	8.03E-05	3.29E-05
TOTAL	2.78E-05	3.23E-05	6.24E-06	6.64E-05	3.98E-05	9.64E-01	1.12E+00	2.16E-01	2.30E+00	3.38E+00	1.38E+00

Notes:
NA - Not Applicable

Source: ENSR 1995 RRISK.WQ1 R.N.: 3

TABLE 4-17
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, CASA GRANDE, AIR
HUMAN HEALTH RISK ASSESSMENT

	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	2.62E-07	3.16E-07	6.94E-07	1.27E-06	9.76E-07	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	1.80E-02	2.17E-02	4.76E-02	8.73E-02	1.77E-01	6.70E-02
CADMIUM	2.91E-07	3.5E-07	7.69E-07	1.41E-06	1.08E-06	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	1.63E-06	1.97E-06	4.32E-06	7.92E-06	6.08E-06	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.41E-01	1.70E-01	3.74E-01	6.85E-01	1.39E+00	5.26E-01
NICKEL	1.39E-07	1.67E-07	3.67E-07	6.73E-07	5.16E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	1.14E-06	1.38E-06	3.03E-06	5.55E-06	4.26E-06	6.86E-02	8.26E-02	1.81E-01	3.33E-01	6.75E-01	2.55E-01
ACROLEIN	NA	NA	NA	NA	NA	3.25E-01	3.92E-01	8.61E-01	1.58E+00	3.20E+00	1.21E+00
AMMONIA	NA	NA	NA	NA	NA	1.31E-02	1.58E-02	3.47E-02	6.37E-02	1.29E-01	4.89E-02
BENZENE	3.55E-06	4.28E-06	9.39E-06	1.72E-05	1.32E-05	8.49E-02	1.02E-01	2.25E-01	4.12E-01	8.35E-01	3.16E-01
1,3-BUTADIENE	1.52E-05	1.83E-05	4.01E-05	7.36E-05	5.65E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROFORM	9.59E-07	1.16E-06	2.54E-06	4.65E-06	3.57E-06	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1.55E-07	1.87E-07	4.1E-07	7.51E-07	5.76E-07	2.00E-05	2.41E-05	5.28E-05	9.69E-05	1.96E-04	7.43E-05
DICHLOROMETHANE	2.95E-07	3.55E-07	7.81E-07	1.43E-06	1.10E-06	2.55E-04	3.07E-04	6.73E-04	1.23E-03	2.50E-03	9.47E-04
FORMALDEHYDE	3.07E-06	3.7E-06	8.13E-06	1.49E-05	1.14E-05	NA	NA	NA	NA	NA	NA
TOLUENE	NA	NA	NA	NA	NA	5.64E-03	6.79E-03	1.49E-02	2.74E-02	5.55E-02	2.10E-02
TRICHLOROETHYLENE	2.8E-07	3.37E-07	7.41E-07	1.36E-06	1.04E-06	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	1.81E-04	2.18E-04	4.80E-04	8.79E-04	1.78E-03	6.75E-04
BENZO(A)PYRENE	5.53E-08	6.66E-08	1.46E-07	2.68E-07	2.06E-07	1.48E-05	1.78E-05	3.90E-05	7.16E-05	1.45E-04	5.49E-05
CHLORPYRIFOS	NA	NA	NA	NA	NA	1.28E-03	1.54E-03	3.39E-03	6.21E-03	1.26E-02	4.77E-03
DISULFOTON	NA	NA	NA	NA	NA	1.05E-02	1.26E-02	2.77E-02	5.08E-02	1.03E-01	3.90E-02
METHYL PARATHION	NA	NA	NA	NA	NA	2.08E-02	2.51E-02	5.50E-02	1.01E-01	2.05E-01	7.74E-02
PENTACHLORONITROBENZENE	8.9E-08	1.07E-07	2.35E-07	4.31E-07	3.31E-07	1.35E-04	1.63E-04	3.58E-04	6.56E-04	1.33E-03	5.04E-04
TRIFLURALIN	7.72E-09	9.29E-09	2.04E-08	3.74E-08	2.87E-08	1.59E-04	1.91E-04	4.19E-04	7.69E-04	1.56E-03	5.90E-04
TOTAL	2.94E-05	3.54E-05	7.77E-05	1.42E-04	1.09E-04	7.79E-01	9.38E-01	2.06E+00	3.78E+00	7.66E+00	2.90E+00

Notes:

Source: ENSR 1995 TOTAL.WQ1 R.N.: 3

Table 4-18 presents the same information for the CTC scenario which shows that the ELCR for the CTC scenario for the Casa Grande study region is approximately 5-fold less than for the RME scenario.

A comparison of the RME HI values for the three receptor scenarios evaluated for noncarcinogenic effects, shown in Table 4-17, indicates that the HI value is higher for the young child resident, i.e., the resident aged 0-6 years, than for the lifetime or adult residents. Therefore, the following discussion of noncarcinogenic effects will focus on the most sensitive receptor, the young child resident.

The HI for the RME young child resident of 7.66 is above the target value of 1.0, indicating that a toxicity endpoint analysis should be conducted. The HI for the CTC scenario is approximately 2.5-fold less than for the RME scenario. The toxicity endpoint analyses for noncarcinogenic effects for the young child receptor for the RME and the CTC scenarios are presented in Tables 4-19 and 4-20, respectively. For the RME case, the HI for respiratory effects, neurological effects and blood effects are each above 1. For the CTC scenario, only the HI for respiratory effects is above 1. An HI value above one only indicates that the predicted threshold value has been exceeded - it does not indicate any probability that adverse health effects will occur, or to what extent they may occur.

The IEUBK results for the RME case for lead for the Casa Grande study region are presented in Table 4-21. As shown, none of the population are expected to exhibit a blood lead concentration in excess of 10 μg/dL, the regulatory target value, as a result of exposure to ambient concentrations of lead in air.

Table 4-22 presents a comparison of the probabilistic and deterministic risk results based on the current emissions estimates. The comparison is presented graphically in Figures 4-5 and 4-6 for the ELCR and HI, respectively. The results indicate that the point estimates are fairly good predictors of the range of estimates provided by the probabilistic analysis, although the RME risk results are approximately 1.5 times as high as the 95th percentile risk estimate from the distribution. For comparison, the 95th percentile risk estimate is 9.06E-05, which is equivalent to 91 excess cancer cases (i.e., in excess of cancer cases expected to naturally occur) in a population of one million over a lifetime of 70 years. Based on the current population estimate for Casa Grande that represents 4 excess cancer cases over a 70 year period, or approximately 0.06 excess cancer cases per year.

Turning to the future, Tables 4-23 and 4-24 present the ELCR and HI results for the RME and CTC scenarios, respectively, based on estimates of future emissions. Risk and hazard estimates are presented for each COI for each receptor. For the future scenario, risks are approximately

TABLE 4-18
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, CASA GRANDE, AIR
HUMAN HEALTH RISK ASSESSMENT

	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	1.14E-07	1.33E-07	2.57E-08	2.73E-07	1.64E-07	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	1.82E-02	2.11E-02	4.07E-03	4.33E-02	6.35E-02	2.60E-02
CADMIUM	1.04E-07	1.21E-07	2.33E-08	2.48E-07	1.49E-07	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	5.85E-07	6.79E-07	1.31E-07	1.39E-06	8.37E-07	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.45E-01	1.68E-01	3.24E-02	3.45E-01	5.06E-01	2.07E-01
NICKEL	5.11E-08	5.94E-08	1.15E-08	1.22E-07	7.32E-08	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	6.18E-07	7.17E-07	1.38E-07	1.47E-06	8.84E-07	1.04E-01	1.21E-01	2.33E-02	2.48E-01	3.64E-01	1.49E-01
ACROLEIN	NA	NA	NA	NA	NA	3.10E-01	3.60E-01	6.95E-02	7.39E-01	1.08E+00	4.44E-01
AMMONIA	NA	NA	NA	NA	NA	2.88E-03	3.35E-03	6.46E-04	6.87E-03	1.01E-02	4.13E-03
BENZENE	1.48E-06	1.72E-06	3.33E-07	3.54E-06	2.12E-06	9.97E-02	1.16E-01	2.24E-02	2.38E-01	3.49E-01	1.43E-01
1,3-BUTADIENE	5.73E-06	6.66E-06	1.29E-06	1.37E-05	8.21E-06	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROFORM	3.61E-07	4.19E-07	8.09E-08	8.61E-07	5.17E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	6.22E-08	7.22E-08	1.39E-08	1.48E-07	8.90E-08	2.25E-05	2.62E-05	5.05E-06	5.37E-05	7.88E-05	3.22E-05
DICHLOROMETHANE	1.84E-07	2.13E-07	4.12E-08	4.38E-07	2.63E-07	4.45E-04	5.16E-04	9.97E-05	1.06E-03	1.56E-03	6.37E-04
FORMALDEHYDE	1.16E-06	1.34E-06	2.59E-07	2.76E-06	1.66E-06	NA	NA	NA	NA	NA	NA
TOLUENE	NA	NA	NA	NA	NA	3.94E-03	4.58E-03	8.84E-04	9.41E-03	1.38E-02	5.65E-03
TRICHLOROETHYLENE	1.11E-07	1.29E-07	2.49E-08	2.65E-07	1.59E-07	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	2.19E-04	2.54E-04	4.91E-05	5.23E-04	7.67E-04	3.14E-04
BENZO(A)PYRENE	1.28E-07	1.48E-07	2.86E-08	3.05E-07	1.83E-07	9.57E-05	1.11E-04	2.15E-05	2.28E-04	3.35E-04	1.37E-04
CHLORPYRIFOS	NA	NA	NA	NA	NA	1.61E-03	1.87E-03	3.61E-04	3.84E-03	5.64E-03	2.31E-03
DISULFOTON	NA	NA	NA	NA	NA	5.81E-03	6.75E-03	1.30E-03	1.39E-02	2.03E-02	8.32E-03
METHYL PARATHION	NA	NA	NA	NA	NA	1.08E-02	1.26E-02	2.43E-03	2.59E-02	3.80E-02	1.55E-02
PENTACHLORONITROBENZENE	2.07E-08	2.4E-08	4.63E-09	4.93E-08	2.96E-08	8.83E-05	1.03E-04	1.98E-05	2.11E-04	3.09E-04	1.26E-04
TRIFLURALIN	1.42E-09	1.65E-09	3.18E-10	3.39E-09	2.03E-09	8.20E-05	9.52E-05	1.84E-05	1.96E-04	2.87E-04	1.17E-04
TOTAL	1.23E-05	1.42E-05	2.75E-06	2.92E-05	1.75E-05	8.72E-01	1.01E+00	1.96E-01	2.08E+00	3.05E+00	1.25E+00

Notes:

Source: ENSR 1995 TOTAL.WQ1 R.N.: 3

TABLE 4-8
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL FUTURE NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, PHOENIX, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	1.53E-06	1.78E-06	3.44E-07	3.66E-06	2.20E-06	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	4.04E-02	4.69E-02	9.06E-03	9.64E-02	1.41E-01	5.79E-02
CADMIUM	1.27E-06	1.48E-06	2.85E-07	3.03E-06	1.82E-06	NA	NA	NA	NA	NA	NA
CHROMIUM VI	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	2.61E-01	3.03E-01	5.84E-02	6.22E-01	9.12E-01	3.73E-01
MERCURY	NA	NA	NA	NA	NA	1.09E-02	1.27E-02	2.45E-03	2.61E-02	3.83E-02	1.57E-02
NICKEL	8.90E-08	1.03E-07	2.00E-08	2.12E-07	1.28E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	1.07E-06	1.24E-06	2.39E-07	2.54E-06	1.52E-06	1.79E-01	2.08E-01	4.02E-02	4.28E-01	6.28E-01	2.57E-01
ACROLEIN	NA	NA	NA	NA	NA	4.74E-01	5.51E-01	1.06E-01	1.13E+00	1.66E+00	6.79E-01
ACRYLONITRILE	9.60E-08	1.12E-07	2.15E-08	2.29E-07	1.37E-07	2.34E-03	2.71E-03	5.24E-04	5.57E-03	8.17E-03	3.34E-03
BENZENE	4.08E-06	4.74E-06	9.15E-07	9.73E-06	5.84E-06	2.74E-01	3.18E-01	6.15E-02	6.54E-01	9.60E-01	3.93E-01
1,3-BUTADIENE	1.19E-05	1.38E-05	2.67E-06	2.84E-05	1.70E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROFORM	6.49E-07	7.54E-07	1.46E-07	1.55E-06	9.30E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	3.74E-07	4.35E-07	8.39E-08	8.93E-07	5.36E-07	1.36E-04	1.57E-04	3.04E-05	3.23E-04	4.74E-04	1.94E-04
DICHLOROMETHANE	2.30E-07	2.67E-07	5.15E-08	5.48E-07	3.29E-07	5.58E-04	6.48E-04	1.25E-04	1.33E-03	1.95E-03	7.99E-04
ETHYLENE DIBROMIDE	8.56E-08	9.95E-08	1.92E-08	2.04E-07	1.23E-07	6.49E-03	7.54E-03	1.46E-03	1.55E-02	2.27E-02	9.30E-03
ETHYLENE OXIDE	7.89E-07	9.16E-07	1.77E-07	1.88E-06	1.13E-06	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	6.03E-06	7.00E-06	1.35E-06	1.44E-05	8.63E-06	NA	NA	NA	NA	NA	NA
TETRACHLOROETHYLENE	4.76E-08	5.53E-08	1.07E-08	1.14E-07	6.81E-08	6.96E-04	8.08E-04	1.56E-04	1.66E-03	2.43E-03	9.96E-04
TRICHLOROETHYLENE	1.47E-08	1.71E-08	3.30E-09	3.52E-08	2.11E-08	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	4.08E-02	4.74E-02	9.14E-03	9.73E-02	1.43E-01	5.84E-02
VINYL CHLORIDE	9.96E-08	1.16E-07	2.23E-08	2.38E-07	1.43E-07	NA	NA	NA	NA	NA	NA
BENZO(A)PYRENE	3.20E-08	3.71E-08	7.17E-09	7.63E-08	4.58E-08	2.40E-05	2.78E-05	5.38E-06	5.72E-05	8.39E-05	3.43E-05
TOTAL	2.99E-05	3.48E-05	6.71E-06	7.14E-05	4.29E-05	1.46E+00	1.70E+00	3.27E-01	3.48E+00	5.11E+00	2.09E+00

Notes:

Source: ENSR 1996 RRISK.WQ1 R.N.: 4

TABLE 4-9
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, TUCSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	1.20E-05	1.44E-05	3.16E-05	5.80E-05	4.45E-05	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	1.89E-02	2.28E-02	5.00E-02	9.16E-02	1.86E-01	7.03E-02
CADMIUM	1.79E-06	2.16E-06	4.74E-06	8.70E-06	6.68E-06	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	5.09E-08	6.13E-08	1.35E-07	2.47E-07	1.89E-07	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	3.31E-02	3.98E-02	8.75E-02	1.60E-01	3.25E-01	1.23E-01
NICKEL	4.78E-08	5.76E-08	1.27E-07	2.32E-07	1.78E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	1.83E-06	2.21E-06	4.85E-06	8.89E-06	6.82E-06	1.10E-01	1.32E-01	2.91E-01	5.33E-01	1.08E+00	4.09E-01
ACROLEIN	NA	NA	NA	NA	NA	2.45E-01	2.95E-01	6.48E-01	1.19E+00	2.41E+00	9.11E-01
BENZENE	8.73E-06	1.05E-05	2.31E-05	4.24E-05	3.25E-05	2.09E-01	2.52E-01	5.53E-01	1.01E+00	2.06E+00	7.78E-01
1,3-BUTADIENE	3.15E-05	3.79E-05	8.32E-05	1.53E-04	1.17E-04	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROBENZENE	NA	NA	NA	NA	NA	3.09E-04	3.73E-04	8.18E-04	1.50E-03	3.04E-03	1.15E-03
CHLOROFORM	1.62E-06	1.96E-06	4.29E-06	7.87E-06	6.04E-06	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1.02E-07	1.23E-07	2.71E-07	4.97E-07	3.81E-07	1.32E-05	1.59E-05	3.50E-05	6.41E-05	1.30E-04	4.92E-05
DICHLOROMETHANE	6.17E-07	7.43E-07	1.63E-06	2.99E-06	2.30E-06	5.32E-04	6.40E-04	1.41E-03	2.58E-03	5.23E-03	1.98E-03
ETHYLENE OXIDE	4.54E-07	5.47E-07	1.20E-06	2.20E-06	1.69E-06	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	3.41E-06	4.10E-06	9.01E-06	1.65E-05	1.27E-05	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	7.83E-03	9.43E-03	2.07E-02	3.80E-02	7.70E-02	2.91E-02
TETRACHLOROETHYLENE	1.06E-07	1.27E-07	2.80E-07	5.12E-07	3.93E-07	5.50E-04	6.62E-04	1.45E-03	2.67E-03	5.41E-03	2.05E-03
TOLUENE	NA	NA	NA	NA	NA	5.51E-03	6.63E-03	1.46E-02	2.67E-02	5.41E-02	2.05E-02
TRICHLOROETHYLENE	9.16E-08	1.10E-07	2.42E-07	4.44E-07	3.41E-07	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	9.40E-04	1.13E-03	2.49E-03	4.56E-03	9.25E-03	3.50E-03
BENZO(A)PYRENE	1.61E-07	1.94E-07	4.27E-07	7.83E-07	6.01E-07	4.31E-05	5.19E-05	1.14E-04	2.09E-04	4.24E-04	1.60E-04
TOTAL	6.47E-05	7.79E-05	1.71E-04	3.14E-04	2.41E-04	7.20E-01	8.67E-01	1.90E+00	3.49E+00	7.08E+00	2.68E+00

Notes:

Source: ENSR 1995 TOTAL.WQ1 R.N.: 3

A comparison of the RME ELCR values for the two receptors evaluated in Table 4-9 indicates that the ELCR value is higher for the lifetime resident, i.e., the resident aged 0-59 years, than for the adult resident, i.e., the resident assumed to be an adult for the entire 59 year exposure period. Therefore, the following discussion will focus on most sensitive receptor, the lifetime resident.

The risk estimate for the RME scenario is 3.14E-04, which is equivalent to 314 excess cancer cases (i.e., in excess of cancer cases expected to naturally occur) in a population of one million over a lifetime of 70 years. Based on the current population estimate for Tucson of 599,299 people, that represents 188 excess cancer cases over a 70 year period, or approximately 3 excess cancer cases per year. For comparison the current U.S. "background" cancer rate due to all causes is approximately 1 in 4, or 250,000 in a population of one million.

Table 4-10 presents the same information for the CTC scenario and shows that the ELCR for the CTC scenario for the Tucson study region is approximately 4-fold less than for the RME scenario.

A comparison of the RME HI values for the three receptor scenarios evaluated for noncarcinogenic effects, shown in Table 4-9, indicates that the HI value is higher for the young child resident, i.e., the resident aged 0-6 years, than for the lifetime or adult residents. Therefore, the following discussion of noncarcinogenic effects will focus on the most sensitive receptor, young child resident.

The HI for the RME young child resident of 7.1 is above the target value of 1.0, indicating that a toxicity endpoint analysis should be conducted. The HI for the CTC scenario is approximately 2-fold less than for the RME scenario. The toxicity endpoint analyses for noncarcinogenic effects for the young child receptor for the RME and the CTC scenarios are presented in Tables 4-11 and 4-12, respectively. For the RME case, the HI for respiratory effects and blood effects are each above 1. For the CTC scenario, only the HI for respiratory effects is above 1. An HI value above one only indicates that the predicted threshold value has been exceeded; it does not indicate any probability that adverse health effects will occur, or to what extent they may occur.

The IEUBK results for the RME case for lead for the Tucson study region are presented in Table 4-13. As shown, none of the population are expected to exhibit a blood lead concentration in excess of 10 μg/dL, the regulatory target value, as a result of exposure to ambient concentrations of lead in air.

TABLE 4-10
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, TUCSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	8.14E-06	9.45E-06	1.82E-06	1.94E-05	1.17E-05	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	3.61E-02	4.20E-02	8.10E-03	8.62E-02	1.26E-01	5.17E-02
CADMIUM	1.22E-06	1.42E-06	2.74E-07	2.91E-06	1.75E-06	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	3.26E-08	3.78E-08	7.3E-09	7.77E-08	4.66E-08	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	6.32E-02	7.34E-02	1.42E-02	1.51E-01	2.21E-01	9.05E-02
NICKEL	3.25E-08	3.78E-08	7.3E-09	7.77E-08	4.66E-08	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	9.4E-07	1.09E-06	2.11E-07	2.24E-06	1.35E-06	1.58E-01	1.84E-01	3.55E-02	3.78E-01	5.54E-01	2.27E-01
ACROLEIN	NA	NA	NA	NA	NA	2.96E-01	3.44E-01	6.64E-02	7.07E-01	1.04E+00	4.24E-01
BENZENE	3.74E-06	4.35E-06	8.39E-07	8.93E-06	5.36E-06	2.52E-01	2.92E-01	5.64E-02	6.00E-01	8.80E-01	3.60E-01
1,3-BUTADIENE	1.32E-05	1.54E-05	2.96E-06	3.15E-05	1.89E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROBENZENE	NA	NA	NA	NA	NA	5.14E-04	5.97E-04	1.15E-04	1.23E-03	1.80E-03	7.36E-04
CHLOROFORM	5.4E-07	6.27E-07	1.21E-07	1.29E-06	7.73E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	5.56E-08	6.46E-08	1.25E-08	1.33E-07	7.97E-08	2.02E-05	2.34E-05	4.52E-06	4.81E-05	7.06E-05	2.89E-05
DICHLOROMETHANE	3.7E-07	4.3E-07	8.3E-08	8.83E-07	5.30E-07	8.97E-04	1.04E-03	2.01E-04	2.14E-03	3.14E-03	1.28E-03
ETHYLENE OXIDE	1.75E-07	2.03E-07	3.92E-08	4.17E-07	2.50E-07	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	1.42E-06	1.65E-06	3.18E-07	3.38E-06	2.03E-06	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	9.48E-03	1.10E-02	2.13E-03	2.26E-02	3.32E-02	1.36E-02
TETRACHLOROETHYLENE	4.24E-08	4.92E-08	9.51E-09	1.01E-07	6.07E-08	6.20E-04	7.20E-04	1.39E-04	1.48E-03	2.17E-03	8.87E-04
TOLUENE	NA	NA	NA	NA	NA	6.99E-03	8.12E-03	1.57E-03	1.67E-02	2.45E-02	1.00E-02
TRICHLOROETHYLENE	5.42E-08	6.29E-08	1.21E-08	1.29E-07	7.76E-08	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	1.34E-03	1.56E-03	3.01E-04	3.21E-03	4.71E-03	1.93E-03
BENZO(A)PYRENE	4.9E-08	5.69E-08	1.1E-08	1.17E-07	7.01E-08	3.67E-05	4.26E-05	8.23E-06	8.76E-05	1.28E-04	5.26E-05
TOTAL	3.16E-05	3.67E-05	7.08E-06	7.53E-05	4.52E-05	9.95E-01	1.16E+00	2.23E-01	2.37E+00	3.48E+00	1.42E+00

Notes:

Source: ENSR 1995 TOTALWQ1 R.N.: 3

TABLE 4-1
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, PHOENIX, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	1.71E-06	2.06E-06	4.52E-06	8.28E-06	6.36E-06	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	1.56E-02	1.88E-02	4.12E-02	7.56E-02	1.53E-01	5.80E-02
CADMIUM	1.43E-06	1.73E-06	3.80E-06	6.96E-06	5.34E-06	NA	NA	NA	NA	NA	NA
CHROMIUM VI	1.15E-06	1.39E-06	3.04E-06	5.58E-06	4.28E-06	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	9.92E-02	1.19E-01	2.62E-01	4.81E-01	9.76E-01	3.69E-01
MERCURY	NA	NA	NA	NA	NA	1.22E-02	1.47E-02	3.23E-02	5.93E-02	1.20E-01	4.55E-02
NICKEL	9.57E-08	1.15E-07	2.53E-07	4.64E-07	3.56E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	3.35E-06	4.03E-06	8.86E-06	1.62E-05	1.25E-05	2.01E-01	2.42E-01	5.31E-01	9.74E-01	1.97E+00	7.47E-01
ACROLEIN	NA	NA	NA	NA	NA	5.80E-01	6.98E-01	1.53E+00	2.81E+00	5.70E+00	2.16E+00
ACRYLONITRILE	2.90E-07	3.49E-07	7.66E-07	1.40E-06	1.08E-06	2.51E-03	3.02E-03	6.63E-03	1.22E-02	2.47E-02	9.34E-03
BENZENE	1.88E-05	2.27E-05	4.98E-05	9.13E-05	7.01E-05	4.50E-01	5.42E-01	1.19E+00	2.18E+00	4.43E+00	1.68E+00
1,3-BUTADIENE	5.46E-05	6.58E-05	1.45E-04	2.65E-04	2.03E-04	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROFORM	2.06E-06	2.48E-06	5.45E-06	1.00E-05	7.67E-06	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1.40E-06	1.69E-06	3.71E-06	6.80E-06	5.22E-06	1.81E-04	2.18E-04	4.79E-04	8.77E-04	1.78E-03	6.73E-04
DICHLOROMETHANE	6.25E-07	7.53E-07	1.65E-06	3.03E-06	2.33E-06	5.41E-04	6.51E-04	1.43E-03	2.62E-03	5.32E-03	2.01E-03
ETHYLENE DIBROMIDE	4.95E-07	5.97E-07	1.31E-06	2.40E-06	1.84E-06	1.34E-02	1.61E-02	3.54E-02	6.48E-02	1.31E-01	4.98E-02
ETHYLENE OXIDE	1.75E-06	2.11E-06	4.63E-06	8.49E-06	6.51E-06	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	1.98E-05	2.39E-05	5.24E-05	9.60E-05	7.37E-05	NA	NA	NA	NA	NA	NA
TETRACHLOROETHYLENE	2.06E-07	2.48E-07	5.45E-07	1.00E-06	7.67E-07	1.07E-03	1.29E-03	2.84E-03	5.20E-03	1.05E-02	3.99E-03
TRICHLOROETHYLENE	1.48E-07	1.79E-07	3.92E-07	7.19E-07	5.52E-07	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	3.37E-02	4.06E-02	8.91E-02	1.63E-01	3.31E-01	1.25E-01
VINYL CHLORIDE	1.96E-07	2.37E-07	5.20E-07	9.53E-07	7.31E-07	NA	NA	NA	NA	NA	NA
BENZO(A)PYRENE	1.68E-07	2.02E-07	4.43E-07	8.13E-07	6.24E-07	4.47E-05	5.39E-05	1.18E-04	2.17E-04	4.40E-04	1.67E-04
TOTAL	1.11E-04	1.33E-04	2.93E-04	5.36E-04	4.12E-04	1.50E+00	1.80E+00	3.96E+00	7.26E+00	1.47E+01	5.58E+00

Notes:

Source: ENSR 1995

TOTALWQ1

R.N.: 5

TABLE 4-2
SUMMARY OF TOTAL ESTIMATED INCREMENTAL CARCINOGENIC RISK
AND TOTAL ESTIMATED NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, PHOENIX, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

	Total Estimated Incremental Carcinogenic Risk					Total Estimated Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	1.16E-06	1.35E-06	2.61E-07	2.77E-06	1.66E-06	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	2.98E-02	3.46E-02	6.68E-03	7.11E-02	1.04E-01	4.27E-02
CADMIUM	9.76E-07	1.13E-06	2.19E-07	2.33E-06	1.40E-06	NA	NA	NA	NA	NA	NA
CHROMIUM VI	2.51E-07	2.92E-07	5.63E-08	5.99E-07	3.59E-07	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.90E-01	2.20E-01	4.25E-02	4.53E-01	6.64E-01	2.72E-01
MERCURY	NA	NA	NA	NA	NA	7.51E-03	8.72E-03	1.68E-03	1.79E-02	2.63E-02	1.07E-02
NICKEL	6.51E-08	7.56E-08	1.46E-08	1.55E-07	9.32E-08	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	1.43E-06	1.66E-06	3.20E-07	3.40E-06	2.04E-06	2.40E-01	2.79E-01	5.39E-02	5.73E-01	8.41E-01	3.44E-01
ACROLEIN	NA	NA	NA	NA	NA	5.70E-01	6.62E-01	1.28E-01	1.36E+00	2.00E+00	8.16E-01
ACRYLONITRILE	7.97E-08	9.26E-08	1.79E-08	1.90E-07	1.14E-07	1.94E-03	2.25E-03	4.35E-04	4.63E-03	6.78E-03	2.78E-03
BENZENE	6.66E-06	7.74E-06	1.49E-06	1.59E-05	9.54E-06	4.48E-01	5.20E-01	1.00E-01	1.07E+00	1.57E+00	6.41E-01
1,3-BUTADIENE	1.99E-05	2.31E-05	4.45E-06	4.74E-05	2.84E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROFORM	4.93E-07	5.72E-07	1.10E-07	1.18E-06	7.06E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	2.94E-07	3.42E-07	6.60E-08	7.03E-07	4.22E-07	1.07E-04	1.24E-04	2.39E-05	2.55E-04	3.73E-04	1.53E-04
DICHLOROMETHANE	2.32E-07	2.70E-07	5.21E-08	5.55E-07	3.33E-07	5.65E-04	6.57E-04	1.27E-04	1.35E-03	1.98E-03	8.09E-04
ETHYLENE DIBROMIDE	8.56E-08	9.95E-08	1.92E-08	2.04E-07	1.23E-07	6.49E-03	7.54E-03	1.46E-03	1.55E-02	2.27E-02	9.30E-03
ETHYLENE OXIDE	6.31E-07	7.33E-07	1.41E-07	1.50E-06	9.03E-07	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	6.97E-06	8.10E-06	1.56E-06	1.66E-05	9.99E-06	NA	NA	NA	NA	NA	NA
TETRACHLOROETHYLENE	6.42E-08	7.45E-08	1.44E-08	1.53E-07	9.19E-08	9.38E-04	1.09E-03	2.10E-04	2.24E-03	3.28E-03	1.34E-03
TRICHLOROETHYLENE	2.60E-08	3.02E-08	5.84E-09	6.21E-08	3.73E-08	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	3.52E-02	4.09E-02	7.89E-03	8.40E-02	1.23E-01	5.04E-02
VINYL CHLORIDE	7.33E-08	8.52E-08	1.64E-08	1.75E-07	1.05E-07	NA	NA	NA	NA	NA	NA
BENZO(A)PYRENE	3.69E-08	4.28E-08	8.27E-09	8.80E-08	5.28E-08	2.76E-05	3.21E-05	6.20E-06	6.60E-05	9.68E-05	3.96E-05
TOTAL	4.09E-05	4.76E-05	9.18E-06	9.77E-05	5.86E-05	1.70E+00	1.97E+00	3.81E-01	4.06E+00	5.95E+00	2.43E+00

Notes:

Source: ENSR 1995 . TOTAL.WQ1 R.N.: 5

TABLE 3-5
ANNUAL AVERAGE AIR CONCENTRATIONS
ARIZONA HAPS, PAYSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Annual Average Air Concentration * (mg/m^3)
ARSENIC	8.58E-07
BARIUM	1.48E-05
CADMIUM	2.02E-06
MANGANESE	1.40E-05
NICKEL	6.73E-07
ACETALDEHYDE	2.63E-03
ACROLEIN	2.28E-05
BENZENE	2.57E-03
1,3-BUTADIENE	2.28E-04
CARBON TETRACHLORIDE	7.50E-04
CHLOROFORM	1.13E-04
1,4-DICHLOROBENZENE	4.95E-05
DICHLOROMETHANE	4.36E-03
FORMALDEHYDE	9.13E-04
N-HEXANE	2.38E-03
TETRACHLOROETHYLENE	6.14E-04
TOLUENE	7.55E-03
TRICHLOROETHYLENE	5.86E-04
BENZO(A)ANTHRACENE	1.25E-06
BENZO(A)PYRENE	1.07E-06
BENZO(B+J+K)FLUORANTHENE	1.61E-06
CHRYSENE	1.38E-06
DIBENZ(A,H+A,C)ANTHRACENE	1.25E-07
INDENO(1,2,3-CD)PYRENE	5.83E-07
ACENAPHTHENE	1.25E-05
ACENAPHTHYLENE	1.51E-05
ANTHRACENE	3.15E-06
BENZO(B)CHRYSENE	1.34E-07
BENZO(G,H,I)PERYLENE	1.00E-06
BIPHENYL	1.55E-05
1,2-DIMETHYLNAPHTHALENE	1.44E-05
1,3+1,6+1,7-DIMETHYLNAPHTHALENE	5.65E-05
1,4+1,5+2,3-DIMETHYLNAPHTHALENE	1.69E-05
1,8-DIMETHYLNAPHTHALENE	3.69E-07
2,6+2,7-DIMETHYLNAPHTHALENE	2.58E-05
FLUORANTHENE	5.81E-06
FLUORENE	1.31E-05
1-METHYLNAPHTHALENE	1.31E-04
2-METHYLNAPHTHALENE	2.81E-04
NAPHTHALENE	4.76E-04
PHENANTHRENE	2.03E-05
PYRENE	4.69E-06

Notes:
* - Based on air monitoring data.

Source: ENSR 1995
DATA.WQ1
R.N.: 7

TABLE 3-6
EVALUATION OF MULTIPATHWAY EXPOSURES TO PARTICULATE HAPS
ARIZONA HAPS, PHOENIX
HUMAN HEALTH RISK ASSESSMENT

Particulate HAPS	Predicted RME Soil Concentration (a) (mg/kg)	Predicted CTC Soil Concentration (b) (mg/kg)	Arizona Background Concentration (c) (mg/kg)	Arizona Residential HBGL (d) (mg/kg)	Evaluate for Multipathway Exposure?	Reason
ARSENIC	0.02977	0.010596	9.4	0.91	NO	<Background, << HBGL
BARIUM	1.694656	0.603183	161.3	8200	NO	<Background, << HBGL
CADMIUM	0.047632	0.016954	0.4	58	NO	<Background, << HBGL
CHROMIUM VI	0.017685	0.006295	17.5	580	NO	<Background, << HBGL
LEAD	0.2977	0.105961	7.7	400	NO	<Background, << HBGL
MERCURY	0.161064	0.057328	0.05	35	NO	<<HBGL
BENZO(A)PYRENE	0.161064	0.057328	ND	0.19	NO	< HBGL

Notes:
(a) - Assuming a 59 year exposure period.
(b) - Assuming a 21 year exposure period.
(c) - "Evaluation of Background Metals Concentrations in Arizona Soils" prepared for ADEQ by The Earth Technology Corporation, June, 1991.
(d) - Proposed Interim Rule. Title 18. Environmental Quality. Chapter 7. Remedial Action. Article 2. Interim Soil Remediation Standards. July, 1995.

MULTIEXP.WQ1 RN: 3

the resulting deposition velocities that were used ranged from about 0.3 to 0.6 cm/s, which corresponded to deposition of particles smaller than about 8 μm aerodynamic diameter to open scrub area.

The results of the analysis are presented in Table 3-6. The predicted soil concentrations are compared to the background concentrations of these species in Arizona soils (ADEQ, 1991), and the Arizona Residential Health-Based Guidance Levels (HBGLs) (ADEQ, 1995). In all cases the predicted soil concentrations are below the HBGL values, and below the background values, with the exception of benzo(a)pyrene for which there is no background data. Therefore, a multi-pathway evaluation of risks due to potential exposures to HAPs was considered to be unnecessary for the Phoenix region.

4.0 RISK CHARACTERIZATION

The risk characterization combines the results of the exposure assessment and the toxicity assessment for each COI in order to estimate the potential for carcinogenic and noncarcinogenic human health risks from potential exposure to COI.

As noted in Volume I of this report, the characterization of the potential health impact of carcinogenic and non-carcinogenic COI is approached in very different ways. The difference in approaches derives from the health-protective assumption that compounds with possible carcinogenic effects act by a no-threshold mechanism, whereas compounds that exhibit noncarcinogenic effects may have a threshold, a dose below which few if any individuals would be expected to respond. Thus under the no-threshold assumption, it is necessary to calculate a risk, termed the excess lifetime cancer risk (ELCR), but for compounds with a threshold, it is possible to simply characterize an exposure as above or below a reference dose, using the ratio termed the hazard quotient (HQ). The sum of the HQ for a particular receptor is termed the hazard index (HI).

This section presents the risk characterization for each of the four study regions. For each study region the following results are discussed:

- Total ELCR and HI based on estimates of current emissions for the RME scenario,
- Total ELCR and HI based on estimates of current emissions for the CTC scenario,
- Toxicity endpoint analyses for the RME and CTC scenarios,
- IEUBK model results for lead,
- Total ELCR and HI based on estimates of future emissions for the RME scenario,
- Total ELCR and HI based on estimates of future emissions for the CTC scenario, and
- Presentation of the probabilistic results based on estimates of current emissions and comparison with the point estimate results.

The results for the Phoenix study region are presented in Section 4.1. The results for the Tucson study region are presented in Section 4.2. The results for the Casa Grande study region are presented in Section 4.3, and the results for the Payson study region are presented in Section 4.4. All results are summarized in Section 4.5.

4.1 Phoenix Study Region

Table 4-1 presents the ELCR and HI results for the RME scenario based on estimates of current emissions. Risk and hazard estimates are presented for each COI for the total lifetime resident (and for each age group) and the adult resident. In addition, hazard estimates are presented for the young child resident. An "NA" in the table indicates that a dose-response value with which to evaluate either carcinogenic or noncarcinogenic health effects was unavailable.

A comparison of the RME ELCR values for the two receptors evaluated indicates that the ELCR value is higher for the lifetime resident, i.e., the resident aged 0-59 years, than for the adult resident, i.e., the resident assumed to be an adult for the entire 59 year exposure period. In risk assessment terminology, the lifetime resident is termed the most sensitive receptor because it has the highest risk. Therefore, the following discussion will focus on the lifetime resident.

The risk estimate for the RME scenario is 5.36E-04, which is equivalent to 536 excess cancer cases (i.e., in excess of cancer cases expected to naturally occur) in a population of one million over a lifetime of 70 years. Based on the current population estimate for Phoenix of 2,119,549 people, that represents 1,136 excess cancer cases over a 70 year period, or approximately 16 excess cancer cases per year. For comparison, the current U.S. "background" cancer rate due to all causes is approximately 1 in 4, or 250,000 in a population of one million. Table 4-2 presents the same information for the CTC scenario and shows that the ELCR for the CTC scenario for the Phoenix study region is approximately 5-fold less than for the RME scenario .

A comparison of the RME HI values for the three receptor scenarios evaluated for noncarcinogenic effects, shown in Table 4-1, indicates that the HI value is higher for the young child resident, i.e., the resident aged 0-6 years, than for the lifetime or adult residents. This young child resident is the most sensitive receptor because it has the highest predicted HI. This is generally due to the combination of a low body weight and high inhalation rate for young children, that results in a higher exposure dose, and therefore higher hazard estimates. Therefore, the following discussion of noncarcinogenic effects will focus on the young child resident.

The RME HI for the young child resident of 37.5 is above the target value of 1.0, indicating that a toxicity endpoint analysis should be conducted. The HI for the CTC scenario is approximately half of that for the RME scenario. Toxicity endpoint analyses for noncarcinogenic effects for the young child receptor for the RME and the CTC scenarios are presented in Tables 4-3 and 4-4, respectively. For the RME case, the HI for respiratory effects, neurological effects and blood effects are each above 1. For the CTC scenario, only the HI for respiratory effects and blood effects are above 1. An HI value above one only indicates that the predicted threshold value has

TABLE 4-3
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - RME SCENARIO
ARIZONA HAPS, PHOENIX, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Developmental Effects	HQ Respiratory Effects	HQ Neurological Effects	HQ Blood Effects	HQ Liver Effects	HQ Kidney Effects	HQ Reproductive Effects
ARSENIC							
BARIUM	1.53E-01						
CADMIUM							
CHROMIUM VI							
MANGANESE		9.76E-01	9.76E-01				
MERCURY			1.20E-01				
NICKEL							
ACETALDEHYDE		1.97E+00					
ACROLEIN		5.70E+00					
ACRYLONITRILE		2.47E-02					
BENZENE				4.43E+00			
1,3-BUTADIENE							
CARBON TETRACHLORIDE					8.73E-01		
CHLOROFORM							
1,4-DICHLOROBENZENE					1.78E-03		
DICHLOROMETHANE					5.32E-03		
ETHYLENE DIBROMIDE							1.31E-01
ETHYLENE OXIDE							
FORMALDEHYDE							
TETRACHLOROETHYLENE						1.05E-02	
TRICHLOROETHYLENE							
TRIMETHYLBENZENE (TOTAL)		3.31E-01	3.31E-01	3.31E-01			
VINYL CHLORIDE							
BENZO(A)PYRENE		4.40E-04					
Total HI =	1.53E-01	9.01E+00	1.43E+00	4.76E+00	8.80E-01	1.05E-02	1.31E-01

Notes:
HI - Hazard Index
HQ - Hazard Quotient

Source: ENSR 1995 TXEPRCR.WQ1 RN: 5

TABLE 4-4
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - CTC SCENARIO
ARIZONA HAPS, PHOENIX, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Developmental Effects	HQ Respiratory Effects	HQ Neurological Effects	HQ Blood Effects	HQ Liver Effects	HQ Kidney Effects	HQ Reproductive Effects
ARSENIC							
BARIUM	1.04E-01						
CADMIUM							
CHROMIUM VI							
MANGANESE		6.64E-01	6.64E-01				
MERCURY			2.63E-02				
NICKEL							
ACETALDEHYDE		8.41E-01					
ACROLEIN		2.00E+00					
ACRYLONITRILE		6.78E-03					
BENZENE				1.57E+00			
1,3-BUTADIENE							
CARBON TETRACHLORIDE					5.94E-01		
CHLOROFORM							
1,4-DICHLOROBENZENE					3.73E-04		
DICHLOROMETHANE					1.98E-03		
ETHYLENE DIBROMIDE							2.27E-02
ETHYLENE OXIDE							
FORMALDEHYDE							
TETRACHLOROETHYLENE						3.28E-03	
TRICHLOROETHYLENE							
TRIMETHYLBENZENE (TOTAL)		1.23E-01	1.23E-01	1.23E-01			
VINYL CHLORIDE							
BENZO(A)PYRENE		9.68E-05					
Total HI =	1.04E-01	3.63E+00	8.13E-01	1.69E+00	5.96E-01	3.28E-03	2.27E-02

Notes:
HI - Hazard Index
HQ - Hazard Quotient

Source: ENSR 1995

TXEPCCR.WQ1 RN: 5

been exceeded; it does not indicate any probability that adverse health effects will occur, or to what extent they may occur. This is a drawback in the current method for evaluating noncarcinogenic effects. Although alternative methods are currently being investigated, none have yet achieved regulatory approval.

The IEUBK model results for the RME case for lead are presented in Table 4-5. As shown, none of the population are expected to exhibit a blood lead concentration in excess of 10 μg/dL, the regulatory target value, as a result of exposure to ambient concentrations of lead in air.

Table 4-6 presents a comparison of the probabilistic and deterministic risk results based on the current emissions estimates. The comparison is presented graphically in Figures 4-1 and 4-2 for the ELCR and HI, respectively. The results indicate that the point estimates are fairly good predictors of the range of estimates provided by the probabilistic analysis, although the RME risk results are approximately one and a half times as high as the 95th percentile risk estimate from the distribution. The 95th percentile risk estimate is 3.28E-04, which is equivalent to 328 excess cancer cases (i.e., in excess of the cancer cases expected to occur in the absence of these HAPs) in a population of one million over a lifetime of 70 years. Based on the current population estimate for Phoenix that represents 695 excess cancer cases over a 70 year period, or approximately 10 excess cancer cases per year. The fairly close agreement between the deterministic and probabilistic results in this analysis is in part due to the fact that the point-estimate values for the exposure parameters were selected as best estimates from the literature rather than using standard agency default exposure assumptions.

Turning to the future, Tables 4-7 and 4-8 present the ELCR and HI results for the RME and CTC scenarios, respectively, based on estimates of future emissions. Risk and hazard estimates are presented for each COI for each receptor. Future cancer risks are predicted to be approximately 70% of the current risk estimates, and future HI are estimated at approximately 85% of the current values for both the RME and CTC scenarios. Therefore, total risks and hazards due to HAPs are expected to decrease in the future in the Phoenix study region.

4.2 Tucson Study Region

Table 4-9 presents the ELCR and HI results for the RME scenario for the Tucson study region based on estimates of current emissions. Risk and hazard estimates are presented for each COI for the total lifetime resident (and for each age group) and the adult resident. In addition, hazard estimates are presented for the young child resident. An "NA" in the table indicates that a dose-response value with which to evaluate either carcinogenic or noncarcinogenic health effects was unavailable.

TABLE 4-5

ARIZONA HAPS, PHOENIX, AIR ASSESSMENT
INTEGRATED EXPOSURE UPTAKE BIOKINETIC (IEUBK) MODEL FOR LEAD IN CHILDREN, U.S. EPA, 1994

Background Soil Pb Concentration =	7.7	mg/kg
Background Indoor Dust Pb Concentration	7.7	mg/kg
Average Outdoor Air Concentration =	2E-05	mg/m^3

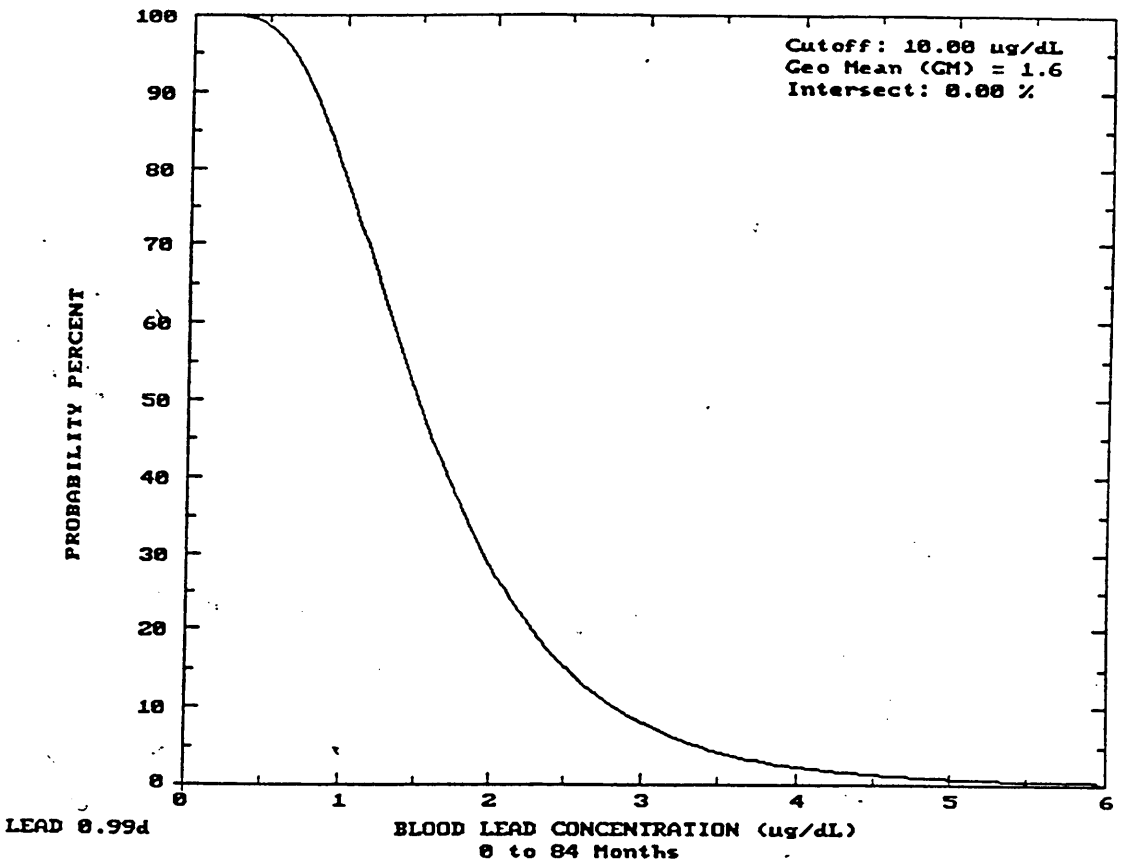


TABLE 4-6
COMPARISON OF DETERMINISTIC AND PROBABILISTIC RESULTS FOR ESTIMATED CARCINOGENIC RISK AND NONCARCINOGENIC HAZARD INDEX
ARIZONA HAPS, PHOENIX STUDY REGION, AIR
HUMAN HEALTH RISK ASSESSMENT

Resident	Distribution of Excess Lifetime Cancer Risk				RME Total Excess Lifetime Cancer Risk	CTC Total Excess Lifetime Cancer Risk
	Mean	50%	95%	Max		
Lifetime	1.70E-04	1.59E-04	3.28E-04	7.93E-04	5.36E-04	9.77E-05
Resident	**Distribution of Noncarcinogenic Hazard Index**				**RME Total Hazard Index**	**CTC Total Hazard Index**
	Mean	**50%**	**95%**	**Max**		
Young Child	7.00E+00	7.00E+00	1.00E+01	1.80E+01	1.50E+01	6.00E+00

Notes:
RME - Reasonable Maximum Exposure
CTC - Central Tendency Case

Source: ENSR 1995 TBL48.WQ1 RN: 0

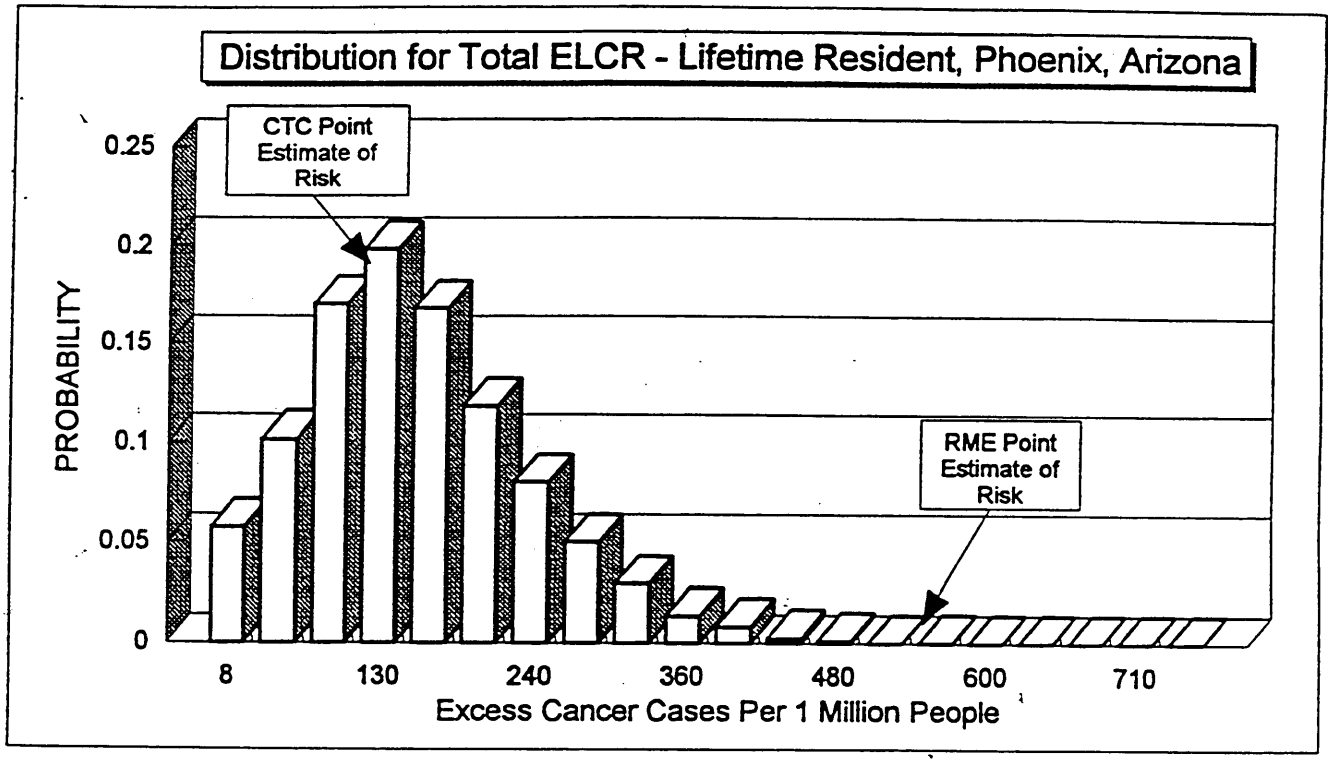

Distribution for Total ELCR - Lifetime Resident, Phoenix, Arizona
CTC Point Estimate of Risk
RME Point Estimate of Risk
PROBABILITY
0.25
0.2
0.15
0.1
0.05
0
8
130
240
360
480
600
710
Excess Cancer Cases Per 1 Million People

Figure 4-1

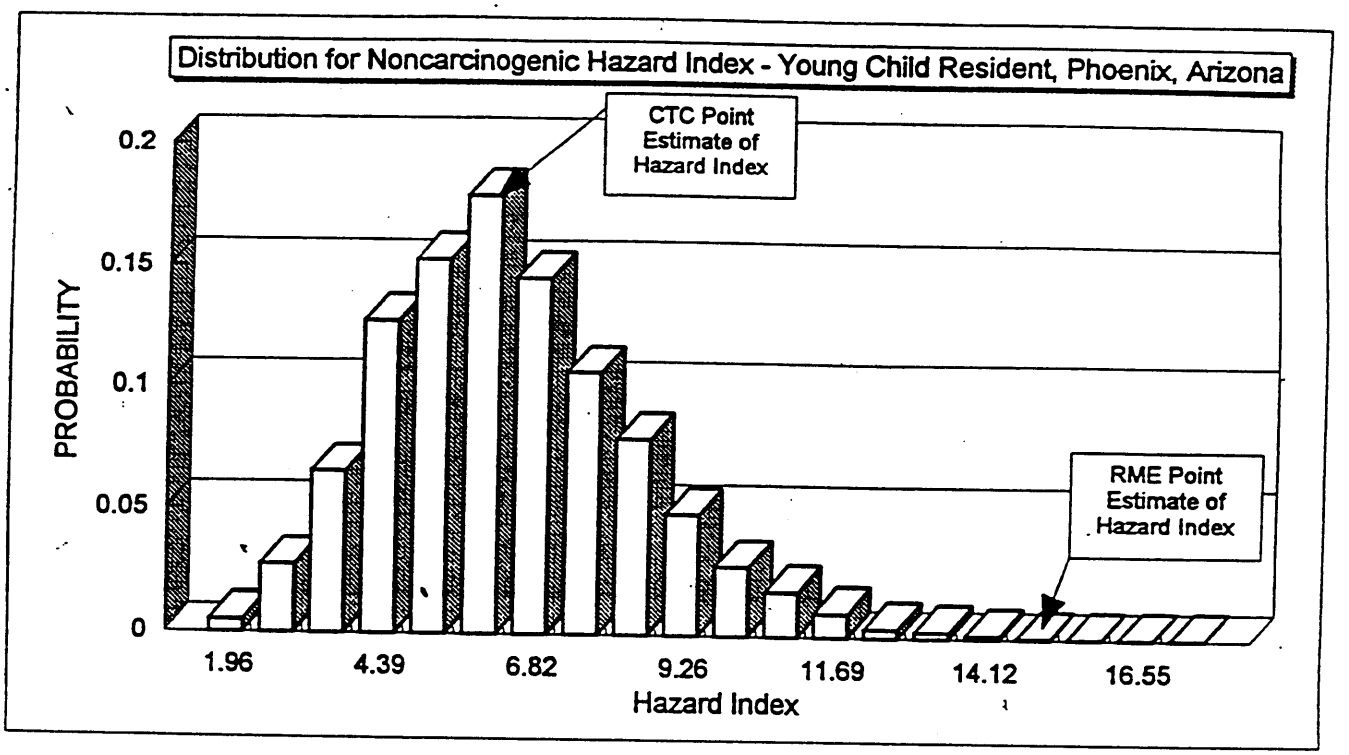

Distribution for Noncarcinogenic Hazard Index - Young Child Resident, Phoenix, Arizona
CTC Point Estimate of Hazard Index
RME Point Estimate of Hazard Index
PROBABILITY
0.2
0.15
0.1
0.05
0
1.96
4.39
6.82
9.26
11.69
14.12
16.55
Hazard Index

Figure 4-2

TABLE 4-7
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL FUTURE NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, PHOENIX, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	2.26E-06	2.72E-06	5.97E-06	1.09E-05	8.39E-06	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	2.11E-02	2.55E-02	5.59E-02	1.02E-01	2.08E-01	7.87E-02
CADMIUM	1.87E-06	2.25E-06	4.94E-06	9.05E-06	6.95E-06	NA	NA	NA	NA	NA	NA
CHROMIUM VI	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.36E-01	1.64E-01	3.61E-01	6.61E-01	1.34E+00	5.07E-01
MERCURY	NA	NA	NA	NA	NA	1.78E-02	2.14E-02	4.71E-02	8.64E-02	1.75E-01	6.63E-02
NICKEL	1.31E-07	1.58E-07	3.46E-07	6.35E-07	4.87E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	2.50E-06	3.01E-06	6.62E-06	1.21E-05	9.31E-06	1.50E-01	1.81E-01	3.97E-01	7.27E-01	1.47E+00	5.58E-01
ACROLEIN	NA	NA	NA	NA	NA	4.82E-01	5.81E-01	1.28E+00	2.34E+00	4.74E+00	1.80E+00
ACRYLONITRILE	3.49E-07	4.20E-07	9.23E-07	1.69E-06	1.30E-06	3.02E-03	3.64E-03	7.99E-03	1.47E-02	2.97E-02	1.12E-02
BENZENE	1.15E-05	1.39E-05	3.05E-05	5.59E-05	4.29E-05	2.76E-01	3.32E-01	7.29E-01	1.34E+00	2.71E+00	1.03E+00
1,3-BUTADIENE	3.27E-05	3.94E-05	8.66E-05	1.59E-04	1.22E-04	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROFORM	2.72E-06	3.27E-06	7.19E-06	1.32E-05	1.01E-05	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1.78E-06	2.15E-06	4.72E-06	8.64E-06	6.63E-06	2.30E-04	2.77E-04	6.08E-04	1.11E-03	2.26E-03	8.56E-04
DICHLOROMETHANE	6.17E-07	7.43E-07	1.63E-06	2.99E-06	2.30E-06	5.34E-04	6.43E-04	1.41E-03	2.59E-03	5.25E-03	1.99E-03
ETHYLENE DIBROMIDE	4.95E-07	5.97E-07	1.31E-06	2.40E-06	1.84E-06	1.34E-02	1.61E-02	3.54E-02	6.48E-02	1.31E-01	4.98E-02
ETHYLENE OXIDE	2.19E-06	2.64E-06	5.79E-06	1.06E-05	8.15E-06	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	1.71E-05	2.06E-05	4.53E-05	8.30E-05	6.37E-05	NA	NA	NA	NA	NA	NA
TETRACHLOROETHYLENE	1.53E-07	1.84E-07	4.04E-07	7.41E-07	5.69E-07	7.95E-04	9.58E-04	2.10E-03	3.86E-03	7.82E-03	2.96E-03
TRICHLOROETHYLENE	8.39E-08	1.01E-07	2.22E-07	4.07E-07	3.12E-07	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	3.90E-02	4.70E-02	1.03E-01	1.89E-01	3.84E-01	1.45E-01
VINYL CHLORIDE	2.67E-07	3.21E-07	7.06E-07	1.29E-06	9.93E-07	NA	NA	NA	NA	NA	NA
BENZO(A)PYRENE	1.45E-07	1.75E-07	3.85E-07	7.05E-07	5.41E-07	3.88E-05	4.67E-05	1.03E-04	1.88E-04	3.82E-04	1.44E-04
TOTAL	7.92E-05	9.54E-05	2.10E-04	3.84E-04	2.95E-04	1.23E+00	1.48E+00	3.25E+00	5.96E+00	1.21E+01	4.57E+00

Notes:

Source: ENSR 1995 RRISK.WQ1 R.N.: 4

TABLE 4-11
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - RME SCENARIO
ARIZONA HAPS, TUCSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Respiratory Effects	HQ Neurological Effects	HQ Blood Effects	HQ Liver Effects	HQ Kidney Effects	HQ Developmental Effects
ARSENIC						
BARIUM						
CADMIUM						1.86E-01
CHROMIUM (HEXAVALENT)						
MANGANESE	3.25E-01	3.25E-01				
NICKEL						
ACETALDEHYDE	1.08E+00					
ACROLEIN	2.41E+00					
BENZENE			2.06E+00			
1,3-BUTADIENE						
CARBON TETRACHLORIDE				8.73E-01		
CHLOROBENZENE				3.04E-03	3.04E-03	
CHLOROFORM						
1,4-DICHLOROBENZENE				1.30E-04		
DICHLOROMETHANE				5.23E-03		
ETHYLENE OXIDE						
FORMALDEHYDE						
N-HEXANE		7.70E-02				
TETRACHLOROETHYLENE					5.41E-03	
TOLUENE		5.41E-02				
TRICHLOROETHYLENE						
TRIMETHYLBENZENE (TOTAL)	9.25E-03	9.25E-03	9.25E-03			
BENZO(A)PYRENE	4.24E-04					
Total HI =	3.82E+00	4.66E-01	2.06E+00	8.81E-01	8.45E-03	1.86E-01

Notes:
HQ - Hazard Quotient
HI - Hazard Index
Source: ENSR 1995 TXEPRCR.WQ RN: 1

TABLE 4-12
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - CTC SCENARIO
ARIZONA HAPS, TUCSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Respiratory Effects	HQ Neurological Effects	HQ Blood Effects	HQ Liver Effects	HQ Kidney Effects	HQ Developmental Effects
ARSENIC						
BARIUM						1.26E-01
CADMIUM						
CHROMIUM (HEXAVALENT)						
MANGANESE	2.21E-01	2.21E-01				
NICKEL						
ACETALDEHYDE	5.54E-01					
ACROLEIN	1.04E+00					
BENZENE			8.80E-01			
1,3-BUTADIENE						
CARBON TETRACHLORIDE				5.94E-01		
CHLOROBENZENE				1.80E-03	1.80E-03	
CHLOROFORM						
1,4-DICHLOROBENZENE				7.06E-05		
DICHLOROMETHANE				3.14E-03		
ETHYLENE OXIDE						
FORMALDEHYDE						
N-HEXANE		3.32E-02				
TETRACHLOROETHYLENE					2.17E-03	
TOLUENE		2.45E-02				
TRICHLOROETHYLENE						
TRIMETHYLBENZENE (TOTAL)	4.71E-03	4.71E-03	4.71E-03			
BENZO(A)PYRENE	1.28E-04					
Total HI =	1.82E+00	2.84E-01	8.85E-01	5.99E-01	3.97E-03	1.26E-01

Notes:
HQ - Hazard Quotient
HI - Hazard Index
Source: ENSR 1995 TXEPCCR.WQ RN: 1

TABLE 4-13

ARIZONA HAPS, TUCSON, AIR ASSESSMENT

INTEGRATED EXPOSURE UPTAKE BIOKINETIC (IEUBK) MODEL FOR LEAD IN CHILDREN, U.S. EPA, 1994

Background Soil Pb Concentration =	7.7	mg/kg
Background Indoor Dust Pb Concentration	7.7	mg/kg
Average Outdoor Air Concentration =	3E-05	mg/m^3

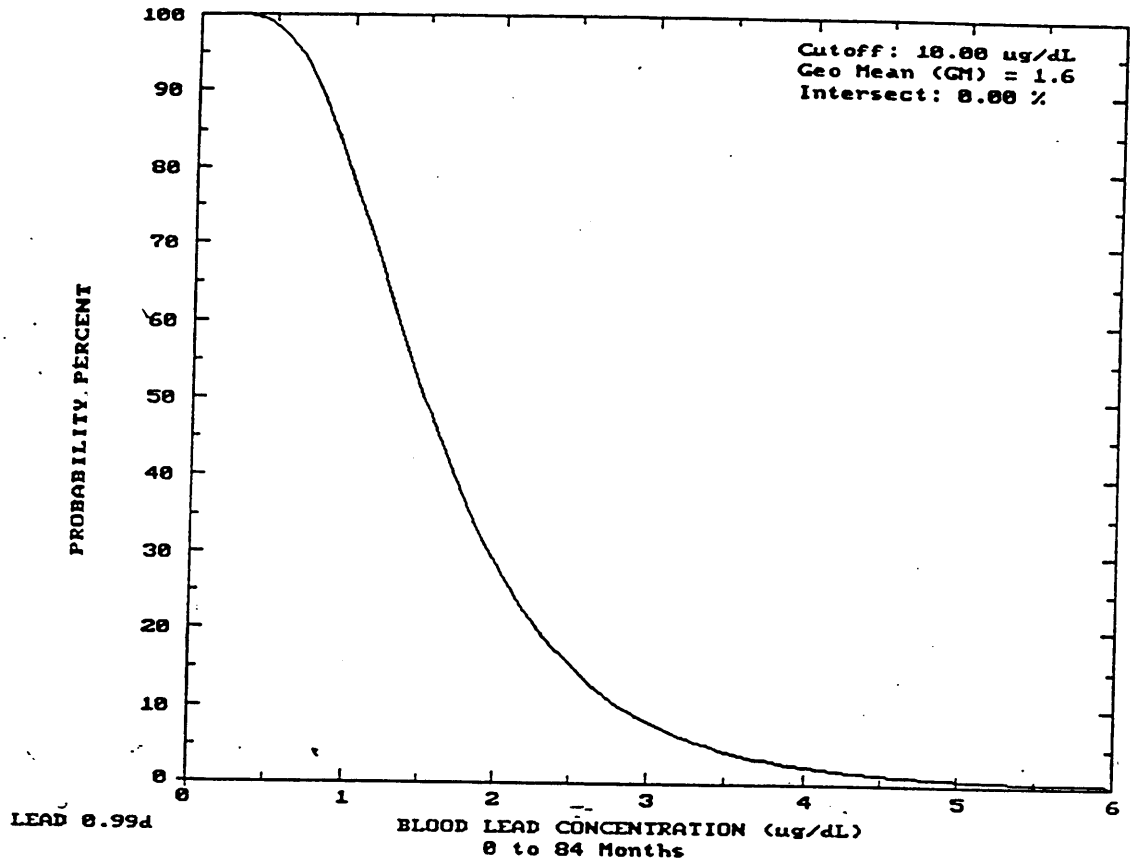


Table 4-14 presents a comparison of the probabilistic and deterministic risk results based on the current emissions estimates. The comparison is presented graphically in Figures 4-3 and 4-4 for the ELCR and HI, respectively. The results indicate that the point estimates are fairly good predictors of the range of estimates provided by the probabilistic analysis, although the RME risk is approximately three times as high as the 95th percentile risk estimate from the distribution. For comparison, the 95th percentile risk estimate is 2.28E-04, which is equivalent to 228 excess cancer cases (i.e., in excess of cancer cases expected to naturally occur) in a population of one million over a lifetime of 70 years. Based on the current population estimate for Tucson that represents 137 excess cancer cases over a 70 year period, or approximately two excess cancer cases per year.

Tables 4-15 and 4-16 present the ELCR and HI results for the RME and CTC scenarios, respectively, based on estimates of future emissions. Future cancer risks are presented to be approximately 85% of the current risk estimates, and future HI are approximately 95% of the current values for both the RME and CTC scenarios. Therefore, total risks and hazards due to HAPs are expected to decrease in the future in the Tucson study region.

4.3 Casa Grande Study Region

Table 4-17 presents the ELCR and HI results for the RME scenario for the Casa Grande study region based on estimates of current emissions. Risk and hazard estimates are presented for each COI for the total lifetime resident (and for each age group) and the adult resident. In addition, hazard estimates are presented for the young child resident. An "NA" in the table indicates that a dose-response value with which to evaluate either carcinogenic or noncarcinogenic health effects was unavailable.

A comparison of the RME ELCR values for the two receptors evaluated in Table 4-17 indicates that the ELCR value is higher for the lifetime resident, i.e., the resident aged 0-59 years, than for the adult resident, i.e., the resident assumed to be an adult for the entire 59 year exposure period. Therefore, the following discussion will focus on the most sensitive receptor, the lifetime resident.

The risk estimate for the RME scenario is 1.42E-04, which is equivalent to 142 excess cancer cases (i.e., in excess of cancer cases expected to naturally occur) in a population of one million over a lifetime of 70 years. Based on the current population estimate for Casa Grande of 46,631 people, that represents 7 excess cancer cases over a 70 year period, or approximately 1 excess cancer case per year. For comparison, the current U.S. "background" cancer rate due to all causes is approximately 1 in 4, or 250,000 in a population of one million.

TABLE 4-14
COMPARISON OF DETERMINISTIC AND PROBABILISTIC RESULTS FOR ESTIMATED CARCINOGENIC RISK AND NONCARCINOGENIC HAZARD INDEX
ARIZONA HAPS, TUCSON STUDY REGION, AIR
HUMAN HEALTH RISK ASSESSMENT

Resident	Distribution of Excess Lifetime Cancer Risk				RME Total Excess Lifetime Cancer Risk	CTC Total Excess Lifetime Cancer Risk
	Mean	50%	95%	Max		
Lifetime	1.26E-04	1.21E-04	2.28E-04	3.87E-04	3.14E-04	7.53E-05
Resident	**Distribution of Noncarcinogenic Hazard Index**				**RME Total Hazard Index**	**CTC Total Hazard Index**
	Mean	**50%**	**95%**	**Max**		
Young Child	3.88E+00	3.78E+00	5.57E+00	8.25E+00	7.08E+00	3.48E+00

Notes:
RME - Reasonable Maximum Exposure
CTC - Central Tendency Case

Source: ENSR 1995 TBL416.WQ1 RN: 0

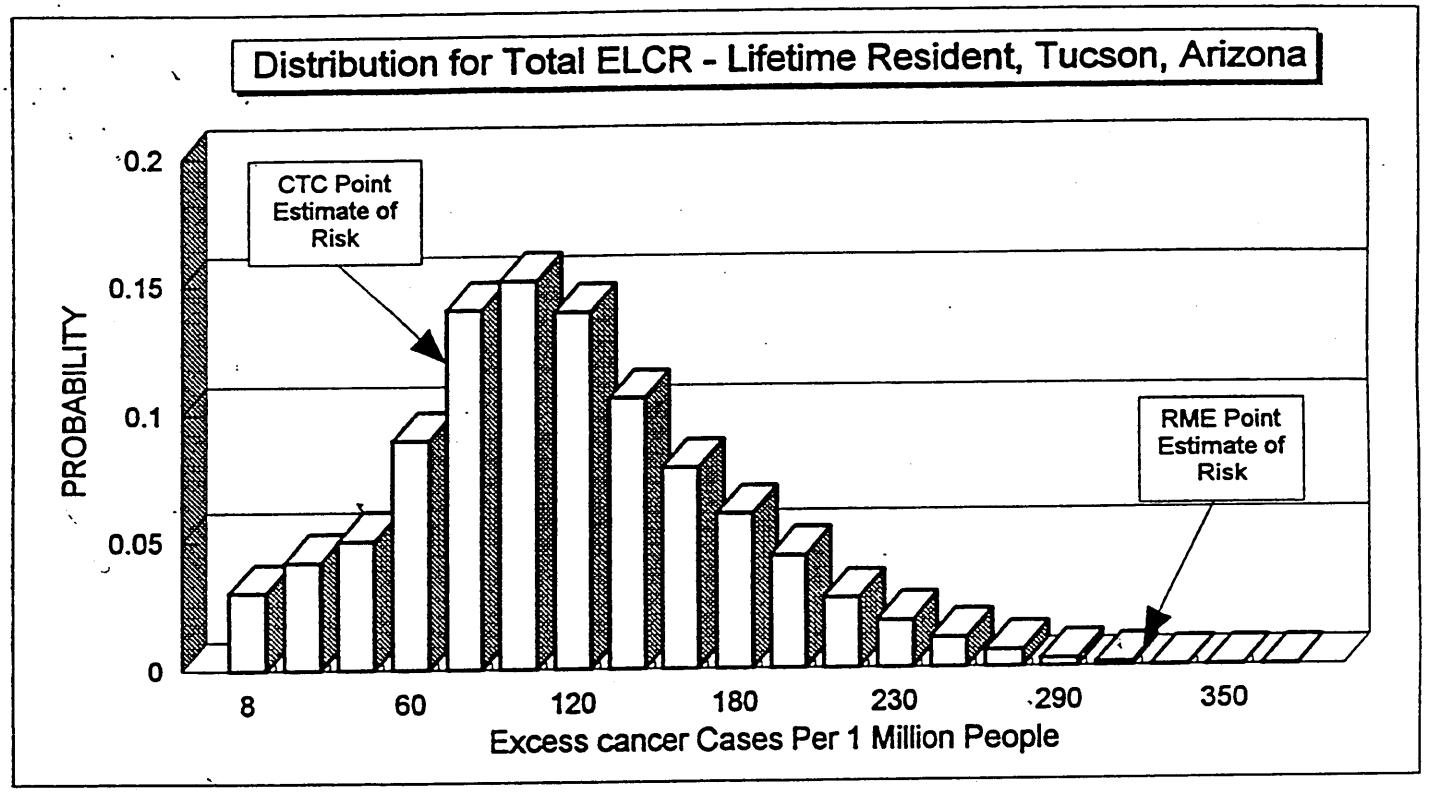

Distribution for Total ELCR - Lifetime Resident, Tucson, Arizona
CTC Point Estimate of Risk
RME Point Estimate of Risk
PROBABILITY
0.2
0.15
0.1
0.05
0
8
60
120
180
230
290
350
Excess cancer Cases Per 1 Million People

Figure 4-3

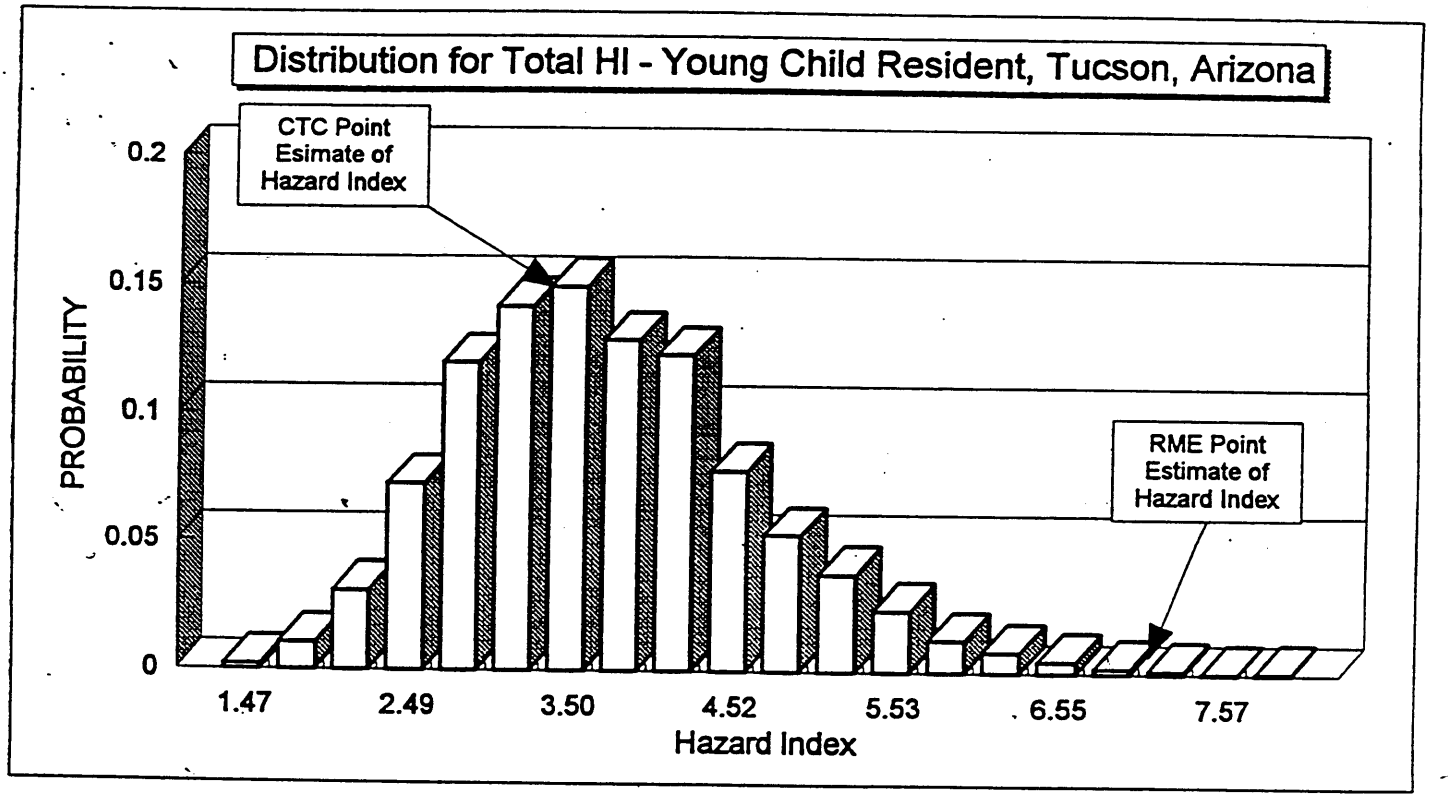

Distribution for Total HI - Young Child Resident, Tucson, Arizona
CTC Point Esimate of Hazard Index
RME Point Estimate of Hazard Index
PROBABILITY
0.2
0.15
0.1
0.05
0
1.47
2.49
3.50
4.52
5.53
6.55
7.57
Hazard Index

Figure 4-4

TABLE 4-15
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL FUTURE NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, TUCSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	1.20E-05	1.44E-05	3.16E-05	5.80E-05	4.45E-05	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	2.48E-02	2.98E-02	6.55E-02	1.20E-01	2.44E-01	9.22E-02
CADMIUM	2.28E-06	2.74E-06	6.03E-06	1.11E-05	8.48E-06	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	4.42E-02	5.32E-02	1.17E-01	2.14E-01	4.34E-01	1.64E-01
NICKEL	6.35E-08	7.65E-08	1.68E-07	3.08E-07	2.36E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	1.69E-06	2.04E-06	4.48E-06	8.22E-06	6.31E-06	1.02E-01	1.22E-01	2.69E-01	4.93E-01	9.99E-01	3.78E-01
ACROLEIN	NA	NA	NA	NA	NA	2.43E-01	2.93E-01	6.43E-01	1.18E+00	2.39E+00	9.05E-01
BENZENE	7.03E-06	8.47E-06	1.86E-05	3.41E-05	2.62E-05	1.68E-01	2.03E-01	4.45E-01	8.16E-01	1.65E+00	6.26E-01
1,3-BUTADIENE	2.34E-05	2.82E-05	6.20E-05	1.14E-04	8.72E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROBENZENE	NA	NA	NA	NA	NA	4.27E-04	5.14E-04	1.13E-03	2.07E-03	4.20E-03	1.59E-03
CHLOROFORM	1.97E-06	2.37E-06	5.20E-06	9.54E-06	7.32E-06	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1.27E-07	1.53E-07	3.36E-07	6.16E-07	4.73E-07	1.64E-05	1.97E-05	4.33E-05	7.94E-05	1.61E-04	6.09E-05
DICHLOROMETHANE	6.93E-07	8.35E-07	1.83E-06	3.36E-06	2.58E-06	5.98E-04	7.20E-04	1.58E-03	2.90E-03	5.88E-03	2.23E-03
ETHYLENE OXIDE	5.91E-07	7.11E-07	1.56E-06	2.86E-06	2.20E-06	NA	NA	NA	NA	NA	NA
FORMALDEHYDE	3.30E-06	3.97E-06	8.73E-06	1.60E-05	1.23E-05	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	7.37E-03	8.87E-03	1.95E-02	3.57E-02	7.24E-02	2.74E-02
TETRACHLOROETHYLENE	7.21E-08	8.68E-08	1.91E-07	3.49E-07	2.68E-07	3.75E-04	4.52E-04	9.92E-04	1.82E-03	3.69E-03	1.40E-03
TOLUENE	NA	NA	NA	NA	NA	5.58E-03	6.72E-03	1.48E-02	2.71E-02	5.49E-02	2.08E-02
TRICHLOROETHYLENE	5.13E-08	6.18E-08	1.36E-07	2.49E-07	1.91E-07	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	1.16E-03	1.40E-03	3.07E-03	5.63E-03	1.14E-02	4.32E-03
BENZO(A)PYRENE	1.01E-07	1.22E-07	2.67E-07	4.89E-07	3.76E-07	2.69E-05	3.24E-05	7.12E-05	1.31E-04	2.65E-04	1.00E-04
TOTAL	5.56E-05	6.70E-05	1.47E-04	2.70E-04	2.07E-04	6.86E-01	8.26E-01	1.82E+00	3.33E+00	6.75E+00	2.55E+00

Notes:
NA - Not Applicable

Source: ENSR 1995 RRISK.WQ1 R.N.: 3

TABLE 4-19
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - RME SCENARIO
ARIZONA HAPS, CASA GRANDE, AIR
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Respiratory Effects	HQ Neurological Effects	HQ Blood Effects	HQ Liver Effects	HQ Developmental Effects	No Observed Effects
ARSENIC						
BARIUM						
CADMIUM					1.77E-01	
CHROMIUM (HEXAVALENT)						
MANGANESE	1.39E+00	1.39E+00				
NICKEL						
ACETALDEHYDE	6.75E-01					
ACROLEIN	3.20E+00					
AMMONIA						
BENZENE						1.29E-01
1,3-BUTADIENE			8.35E-01			
CARBON TETRACHLORIDE						
CHLOROFORM				8.73E-01		
1,4-DICHLOROBENZENE						
DICHLOROMETHANE				1.96E-04		
FORMALDEHYDE				2.50E-03		
TOLUENE		5.55E-02				
TRICHLOROETHYLENE						
TRIMETHYLBENZENE (TOTAL)	1.78E-03	1.78E-03	1.78E-03			
BENZO(A)PYRENE	1.45E-04					
CHLORPYRIFOS			1.26E-02			
DISULFOTON		1.03E-01	1.03E-01			
METHYL PARATHION			2.05E-01			
PENTACHLORONITROBENZENE				1.33E-03		
TRIFLURALIN			1.56E-03	1.56E-03		
Total HI =	5.26E+00	1.55E+00	1.16E+00	8.75E-01	1.77E-01	

Notes:
HQ - Hazard Quotient
HI - Hazard Index
Source: ENSR 1995 TXEPRCR.WQ RN: 0

TABLE 4-20
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - CTC SCENARIO
ARIZONA HAPS, CASA GRANDE, AIR
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Respiratory Effects	HQ Neurological Effects	HQ Blood Effects	HQ Liver Effects	HQ Developmental Effects	No Observed Effects
ARSENIC						
BARIUM					6.35E-02	
CADMIUM						
CHROMIUM (HEXAVALENT)						
MANGANESE	5.06E-01	5.06E-01				
NICKEL						
ACETALDEHYDE	3.64E-01					
ACROLEIN	1.08E+00					
AMMONIA						1.01E-02
BENZENE			3.49E-01			
1,3-BUTADIENE						
CARBON TETRACHLORIDE				5.94E-01		
CHLOROFORM						
1,4-DICHLOROBENZENE				7.88E-05		
DICHLOROMETHANE				1.56E-03		
FORMALDEHYDE						
TOLUENE		1.38E-02				
TRICHLOROETHYLENE						
TRIMETHYLBENZENE (TOTAL)	7.67E-04	7.67E-04	7.67E-04			
BENZO(A)PYRENE	3.35E-04					
CHLORPYRIFOS			5.64E-03			
DISULFOTON		2.03E-02	2.03E-02			
METHYL PARATHION			3.80E-02			
PENTACHLORONITROBENZENE				3.09E-04		
TRIFLURALIN			2.87E-04	2.87E-04		
Total HI =	1.96E+00	5.41E-01	4.14E-01	5.95E-01	6.35E-02	

Notes:
HQ - Hazard Quotient
HI - Hazard Index

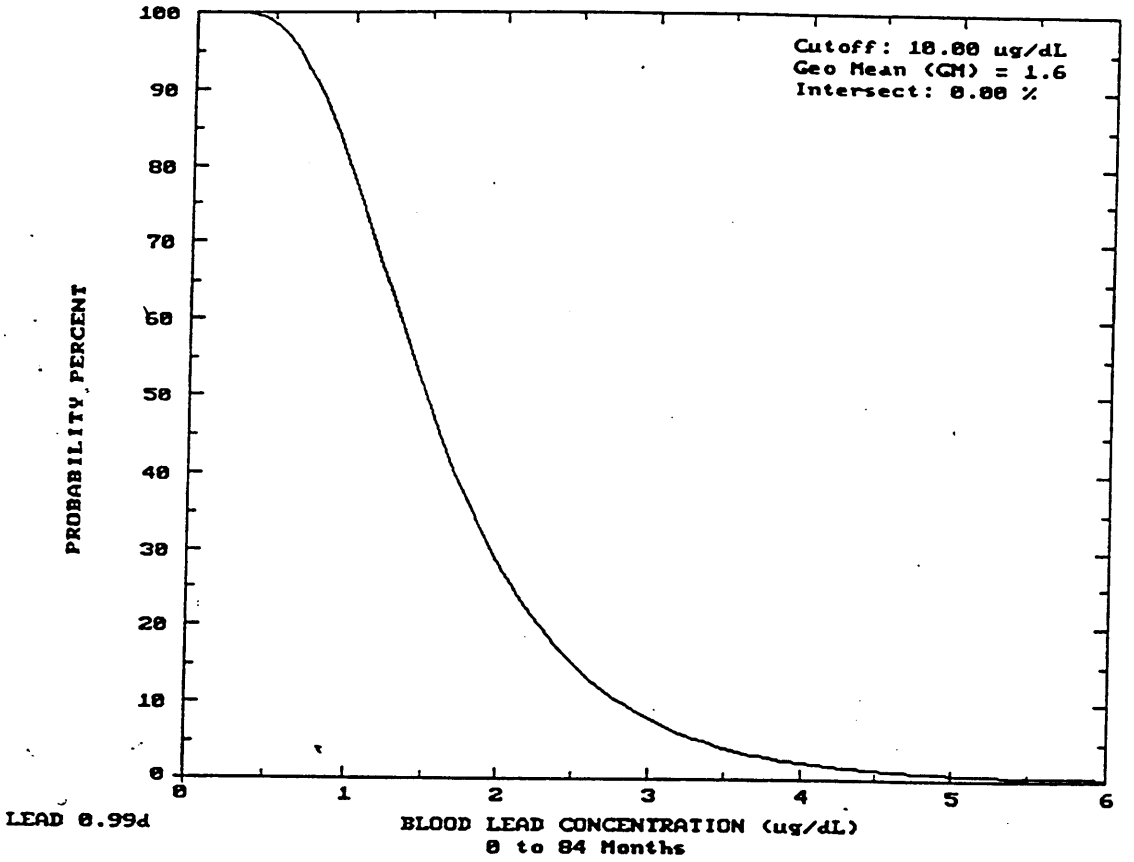
Source: ENSR 1995 TXEPCCR.WQ RN: 0

TABLE 4-21

ARIZONA HAPS, CASA GRANDE, AIR ASSESSMENT

INTEGRATED EXPOSURE UPTAKE BIOKINETIC (IEUBK) MODEL FOR LEAD IN CHILDREN, U.S. EPA, 1994

Background Soil Pb Concentration =	7.7	mg/kg
Background Indoor Dust Pb Concentration	7.7	mg/kg
Average Outdoor Air Concentration =	2.2E-06	mg/m^3



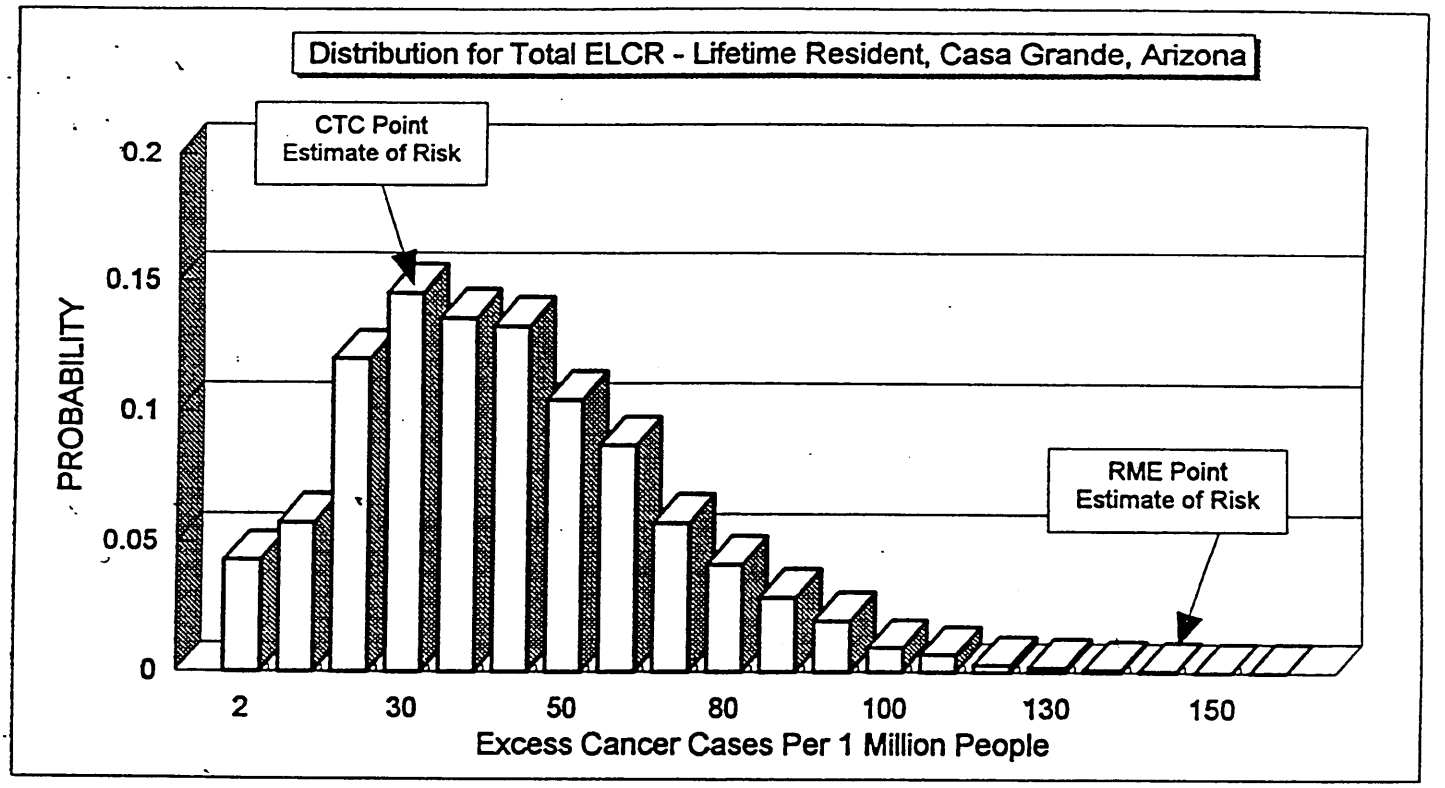

Distribution for Total ELCR - Lifetime Resident, Casa Grande, Arizona
CTC Point Estimate of Risk
RME Point Estimate of Risk
PROBABILITY
0.2
0.15
0.1
0.05
0
2
30
50
80
100
130
150
Excess Cancer Cases Per 1 Million People

Figure 4-5

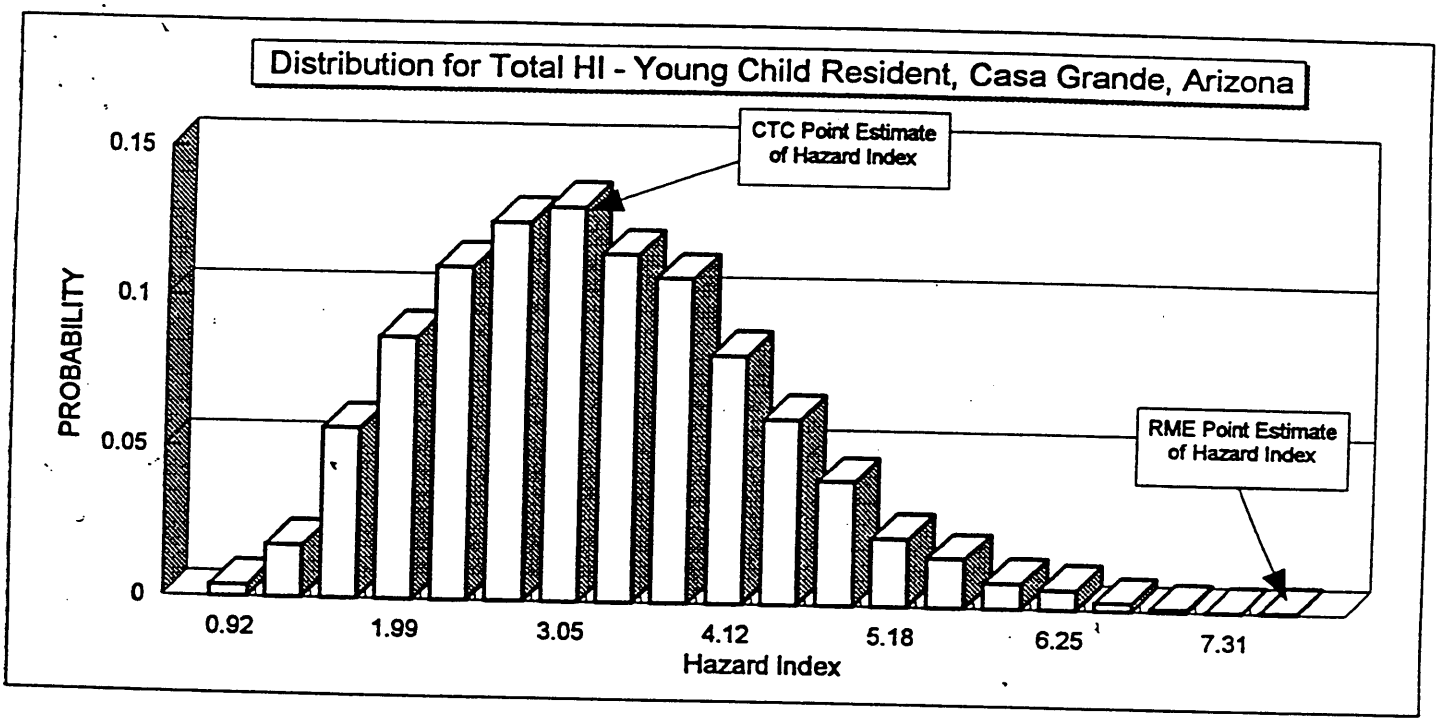

Distribution for Total HI - Young Child Resident, Casa Grande, Arizona
CTC Point Estimate of Hazard Index
RME Point Estimate of Hazard Index
PROBABILITY
0.15
0.1
0.05
0
0.92
1.99
3.05
4.12
5.18
6.25
7.31
Hazard Index

Figure 4-6

TABLE 4-22
COMPARISON OF DETERMINISTIC AND PROBABILISTIC RESULTS FOR ESTIMATED CARCINOGENIC RISK AND NONCARCINOGENIC HAZARD INDEX
ARIZONA HAPS, CASA GRANDE STUDY REGION, AIR
HUMAN HEALTH RISK ASSESSMENT

Resident	Distribution of Excess Lifetime Cancer Risk				RME Total Excess Lifetime Cancer Risk	CTC Total Excess Lifetime Cancer Risk
	Mean	50%	95%	Max		
Lifetime	4.63E-05	4.33E-05	9.06E-05	1.67E-04	1.42E-04	2.92E-05
Resident	**Distribution of Noncarcinogenic Hazard Index**				**RME Total Hazard Index**	**CTC Total Hazard Index**
	Mean	**50%**	**95%**	**Max**		
Young Child	3.41E+00	3.30E+00	5.29E+00	8.02E+00	7.66E+00	3.05E+00

Notes:
RME - Reasonable Maximum Exposure
CTC - Central Tendency Case

Source: ENSR 1995 TBL424.WQ1 RN: 0

TABLE 4-23
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL FUTURE NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, CASA GRANDE, AIR
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	3.41E-07	4.11E-07	9.03E-07	1.66E-06	1.27E-06	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	2.41E-02	2.91E-02	6.38E-02	1.17E-01	2.37E-01	8.98E-02
CADMIUM	3.68E-07	4.43E-07	9.74E-07	1.79E-06	1.37E-06	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	2.09E-07	2.52E-07	5.53E-07	1.01E-06	7.79E-07	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.88E-01	2.27E-01	4.98E-01	9.13E-01	1.85E+00	7.01E-01
NICKEL	1.88E-07	2.26E-07	4.97E-07	9.11E-07	6.99E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	9.24E-07	1.11E-06	2.44E-06	4.48E-06	3.44E-06	5.54E-02	6.67E-02	1.46E-01	2.69E-01	5.45E-01	2.06E-01
ACROLEIN	NA	NA	NA	NA	NA	2.64E-01	3.18E-01	6.98E-01	1.28E+00	2.59E+00	9.82E-01
AMMONIA	NA	NA	NA	NA	NA	1.75E-02	2.10E-02	4.62E-02	8.47E-02	1.72E-01	6.50E-02
BENZENE	2.77E-06	3.34E-06	7.33E-06	1.34E-05	1.03E-05	6.63E-02	7.99E-02	1.75E-01	3.22E-01	6.52E-01	2.47E-01
1,3-BUTADIENE	1.10E-05	1.33E-05	2.92E-05	5.35E-05	4.11E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROFORM	1.14E-06	1.38E-06	3.02E-06	5.54E-06	4.25E-06	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	2.06E-07	2.48E-07	5.44E-07	9.97E-07	7.65E-07	2.65E-05	3.19E-05	7.01E-05	1.29E-04	2.61E-04	9.87E-05
DICHLOROMETHANE	3.22E-07	3.87E-07	8.51E-07	1.56E-06	1.20E-06	2.77E-04	3.34E-04	7.33E-04	1.34E-03	2.73E-03	1.03E-03
FORMALDEHYDE	2.78E-06	3.35E-06	7.36E-06	1.35E-05	1.04E-05	NA	NA	NA	NA	NA	NA
TOLUENE	NA	NA	NA	NA	NA	5.80E-03	6.98E-03	1.53E-02	2.81E-02	5.70E-02	2.16E-02
TRICHLOROETHYLENE	1.52E-07	1.83E-07	4.03E-07	7.39E-07	5.67E-07	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	2.23E-04	2.68E-04	5.89E-04	1.08E-03	2.19E-03	8.28E-04
BENZO(A)PYRENE	7.67E-08	9.23E-08	2.03E-07	3.72E-07	2.85E-07	2.05E-05	2.46E-05	5.41E-05	9.92E-05	2.01E-04	7.61E-05
CHLORPYRIFOS	NA	NA	NA	NA	NA	1.54E-03	1.85E-03	4.07E-03	7.46E-03	1.51E-02	5.72E-03
DISULFOTON	NA	NA	NA	NA	NA	1.26E-02	1.51E-02	3.32E-02	6.09E-02	1.24E-01	4.68E-02
METHYL PARATHION	NA	NA	NA	NA	NA	2.50E-02	3.01E-02	6.60E-02	1.21E-01	2.46E-01	9.29E-02
PENTACHLORONITROBENZENE	1.07E-07	1.29E-07	2.82E-07	5.18E-07	3.97E-07	1.62E-04	1.96E-04	4.30E-04	7.88E-04	1.60E-03	6.04E-04
TRIFLURALIN	9.26E-09	1.12E-08	2.45E-08	4.49E-08	3.45E-08	1.90E-04	2.29E-04	5.03E-04	9.23E-04	1.87E-03	7.08E-04
TOTAL	2.29E-05	2.76E-05	6.06E-05	1.11E-04	8.52E-05	7.50E-01	9.03E-01	1.98E+00	3.64E+00	7.37E+00	2.79E+00

Notes:

Source: ENSR 1995

RRISKWQ1

R.N.: 3

TABLE 4-24
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL FUTURE NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, CASA GRANDE, AIR
HUMAN HEALTH RISK ASSESSMENT

	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
Compound	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	1.49E-07	1.73E-07	3.34E-08	3.56E-07	2.13E-07	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	2.43E-02	2.83E-02	5.46E-03	5.81E-02	8.52E-02	3.49E-02
CADMIUM	1.32E-07	1.53E-07	2.95E-08	3.14E-07	1.89E-07	NA	NA	NA	NA	NA	NA
CHROMIUM (HEXAVALENT)	7.48E-08	8.69E-08	1.68E-08	1.79E-07	1.07E-07	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.93E-01	2.24E-01	4.32E-02	4.60E-01	6.74E-01	2.76E-01
NICKEL	6.92E-08	8.04E-08	1.55E-08	1.65E-07	9.91E-08	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	4.99E-07	5.79E-07	1.12E-07	1.19E-06	7.14E-07	8.40E-02	9.75E-02	1.88E-02	2.00E-01	2.94E-01	1.20E-01
ACROLEIN	NA	NA	NA	NA	NA	2.51E-01	2.92E-01	5.63E-02	5.99E-01	8.79E-01	3.60E-01
AMMONIA	NA	NA	NA	NA	NA	3.83E-03	4.45E-03	8.59E-04	9.14E-03	1.34E-02	5.49E-03
BENZENE	1.16E-06	1.35E-06	2.60E-07	2.76E-06	1.66E-06	7.78E-02	9.04E-02	1.75E-02	1.86E-01	2.72E-01	1.11E-01
1,3-BUTADIENE	4.17E-06	4.84E-06	9.35E-07	9.94E-06	5.97E-06	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROFORM	4.30E-07	5.00E-07	9.64E-08	1.03E-06	6.16E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	8.25E-08	9.59E-08	1.85E-08	1.97E-07	1.18E-07	2.99E-05	3.47E-05	6.70E-06	7.13E-05	1.05E-04	4.28E-05
DICHLOROMETHANE	2.00E-07	2.32E-07	4.48E-08	4.77E-07	2.86E-07	4.84E-04	5.63E-04	1.09E-04	1.16E-03	1.70E-03	6.93E-04
FORMALDEHYDE	1.05E-06	1.22E-06	2.35E-07	2.50E-06	1.50E-06	NA	NA	NA	NA	NA	NA
TOLUENE	NA	NA	NA	NA	NA	4.05E-03	4.71E-03	9.09E-04	9.67E-03	1.42E-02	5.80E-03
TRICHLOROETHYLENE	6.04E-08	7.02E-08	1.35E-08	1.44E-07	8.65E-08	NA	NA	NA	NA	NA	NA
TRIMETHYLBENZENE (TOTAL)	NA	NA	NA	NA	NA	2.69E-04	3.12E-04	6.03E-05	6.41E-04	9.41E-04	3.85E-04
BENZO(A)PYRENE	1.77E-07	2.06E-07	3.97E-08	4.22E-07	2.54E-07	1.33E-04	1.54E-04	2.98E-05	3.17E-04	4.65E-04	1.90E-04
CHLORPYRIFOS	NA	NA	NA	NA	NA	1.93E-03	2.24E-03	4.33E-04	4.61E-03	6.76E-03	2.77E-03
DISULFOTON	NA	NA	NA	NA	NA	6.97E-03	8.10E-03	1.56E-03	1.66E-02	2.44E-02	9.99E-03
METHYL PARATHION	NA	NA	NA	NA	NA	1.30E-02	1.51E-02	2.92E-03	3.11E-02	4.56E-02	1.86E-02
PENTACHLORONITROBENZENE	2.48E-08	2.88E-08	5.56E-09	5.91E-08	3.55E-08	1.06E-04	1.23E-04	2.37E-05	2.53E-04	3.71E-04	1.52E-04
TRIFLURALIN	1.70E-09	1.98E-09	3.82E-10	4.07E-09	2.44E-09	9.84E-05	1.14E-04	2.21E-05	2.35E-04	3.44E-04	1.41E-04
TOTAL	9.81E-06	1.14E-05	2.20E-06	2.34E-05	1.40E-05	8.31E-01	9.65E-01	1.86E-01	1.98E+00	2.91E+00	1.19E+00

Notes:

Source: ENSR 1995 RRISK.WQ1 R.K.: 3

TABLE 4-27
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - RME SCENARIO
ARIZONA HAPS, PAYSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Developmental Effects	HQ Respiratory Effects	HQ Neurologoical Effects	HQ Blood Effects	HQ Liver Effects	HQ Kidney Effects
ARSENIC						
BARIUM	6.87E-02					
CADMIUM						
MANGANESE		6.51E-01	6.51E-01			
NICKEL						
ACETALDEHYDE		6.80E-01				
ACROLEIN		2.66E+00				
BENZENE				9.98E-01		
1,3-BUTADIENE						
CARBON TETRACHLORIDE					8.73E-01	
CHLOROFORM						
1,4-DICHLOROBENZENE					1.43E-04	
DICHLOROMETHANE					3.37E-03	
FORMALDEHYDE						
N-HEXANE			2.77E-02			
TETRACHLOROETHYLENE						3.58E-03
TOLUENE			4.40E-02			
TRICHLOROETHYLENE						
BENZO(A)ANTHRACENE		7.31E-04				
BENZO(A)PYRENE		6.24E-04				
BENZO(B+J+K)FLUORANTHENE		9.35E-04				
CHRYSENE		8.06E-04				
DIBENZ(A,H+A,C)ANTHRACENE		7.28E-05				
INDENO(1,2,3-CD)PYRENE		3.40E-04				
ACENAPHTHENE		7.26E-03				
ACENAPHTHYLENE		8.77E-03				
ANTHRACENE		1.83E-03				
BENZO(B)CHRYSENE		7.81E-05				
BENZO(G,H,I)PERYLENE		5.84E-04				
BIPHENYL		9.03E-03				
1,2-DIMETHYLNAPHTHALENE		8.39E-03				
1,3+1,6+1,7-DIMETHYLNAPHTHALENE		3.29E-02				
1,4+1,5+2,3-DIMETHYLNAPHTHALENE		9.88E-03				
1,8-DIMETHYLNAPHTHALENE		2.15E-04				
2,6+2,7-DIMETHYLNAPHTHALENE		1.50E-02				
FLUORANTHENE		3.39E-03				
FLUORENE		7.61E-03				
1-METHYLNAPHTHALENE		7.61E-02				
2-METHYLNAPHTHALENE		1.64E-01				
NAPHTHALENE		2.77E-01				
PHENANTHRENE		1.18E-02				
PYRENE		2.73E-03				
Total HI =	6.87E-02	4.63E+00	7.22E-01	9.98E-01	8.76E-01	3.58E-03

Notes:
HQ - Hazard Quotient
HI - Hazard Index
Source: ENSR 1995

TXEPRCR.WQ RN: 3

TABLE 4-28
TOXICITY ENDPOINTS ANALYSIS FOR NONCARCINOGENIC EFFECTS OF COI DUE TO INHALATION
YOUNG CHILD RESIDENT - CTC SCENARIO
ARIZONA HAPS, PAYSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	HQ Developmental Effects	HQ Respiratory Effects	HQ Neurologoical Effects	HQ Blood Effects	HQ Liver Effects	HQ Kidney Effects
ARSENIC						
BARIUM	4.68E-02					
CADMIUM						
MANGANESE		4.43E-01	4.43E-01			
NICKEL						
ACETALDEHYDE		4.63E-01				
ACROLEIN		1.81E+00				
BENZENE				6.79E-01		
1,3-BUTADIENE						
CARBON TETRACHLORIDE					5.94E-01	
CHLOROFORM						
1,4-DICHLOROBENZENE					9.72E-05	
DICHLOROMETHANE					2.29E-03	
FORMALDEHYDE						
N-HEXANE			1.88E-02			
TETRACHLOROETHYLENE						2.43E-03
TOLUENE			2.99E-02			
TRICHLOROETHYLENE						
BENZO(A)ANTHRACENE		4.97E-04				
BENZO(A)PYRENE		4.25E-04				
BENZO(B+J+K)FLUORANTHENE		6.36E-04				
CHRYSENE		5.48E-04				
DIBENZ(A,H+A,C)ANTHRACENE		4.96E-05				
INDENO(1,2,3-CD)PYRENE		2.31E-04				
ACENAPHTHENE		4.94E-03				
ACENAPHTHYLENE		5.97E-03				
ANTHRACENE		1.25E-03				
BENZO(B)CHRYSENE		5.31E-05				
BENZO(G,H,I)PERYLENE		3.97E-04				
BIPHENYL		6.15E-03				
1,2-DIMETHYLNAPHTHALENE		5.71E-03				
1,3+1,6+1,7-DIMETHYLNAPHTHALENE		2.24E-02				
1,4+1,5+2,3-DIMETHYLNAPHTHALENE		6.72E-03				
1,8-DIMETHYLNAPHTHALENE		1.46E-04				
2,6+2,7-DIMETHYLNAPHTHALENE		1.02E-02				
FLUORANTHENE		2.30E-03				
FLUORENE		5.18E-03				
1-METHYLNAPHTHALENE		5.18E-02				
2-METHYLNAPHTHALENE		1.11E-01				
NAPHTHALENE		1.89E-01				
PHENANTHRENE		8.05E-03				
PYRENE		1.86E-03				
Total HI =	4.68E-02	3.15E+00	4.92E-01	6.79E-01	5.96E-01	2.43E-03

Notes:
HQ - Hazard Quotient
HI - Hazard Index

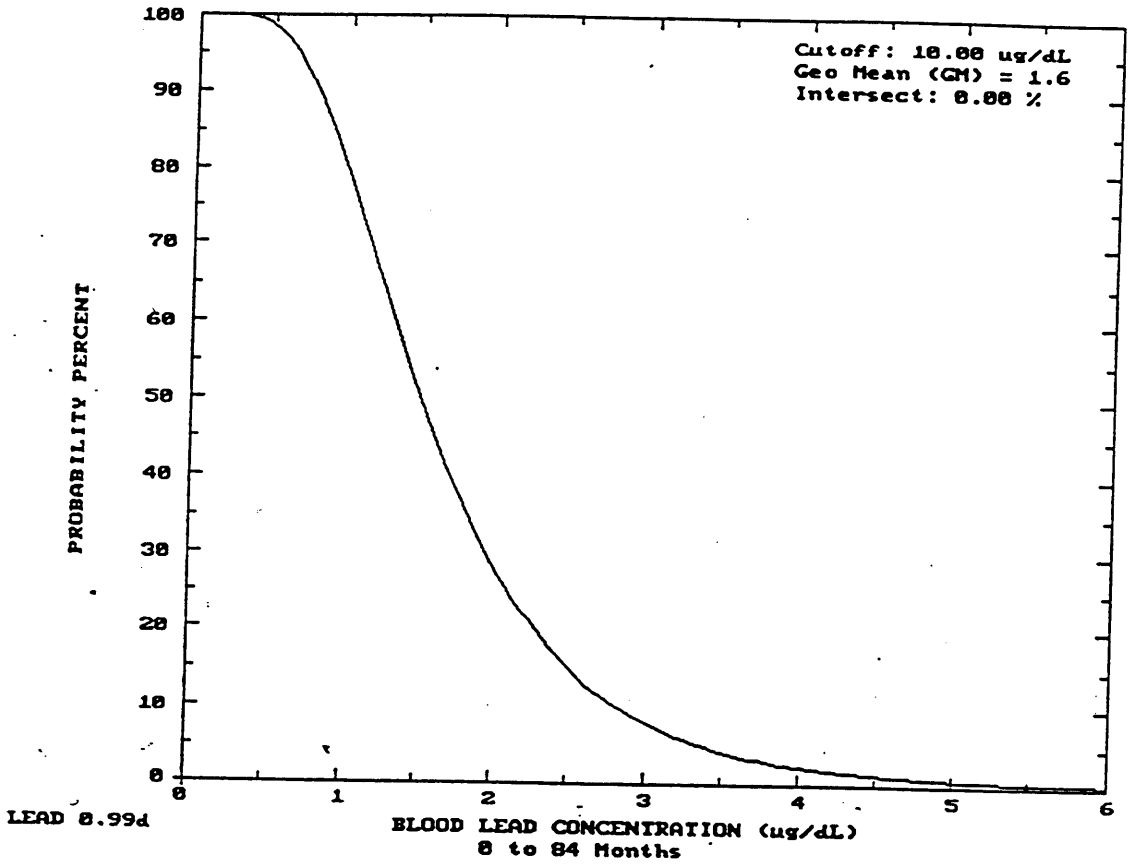
Source: ENSR 1995 TXEPCCR.WQ RN: 3

TABLE 4-29

ARIZONA HAPS, PAYSON, AIR ASSESSMENT

INTEGRATED EXPOSURE UPTAKE BIOKINETIC (IEUBK) MODEL FOR LEAD IN CHILDREN, U.S. EPA, 1994

Background Soil Pb Concentration =	7.7	mg/kg
Background Indoor Dust Pb Concentration	7.7	mg/kg
Average Outdoor Air Concentration =	6.3E-06	mg/m^3



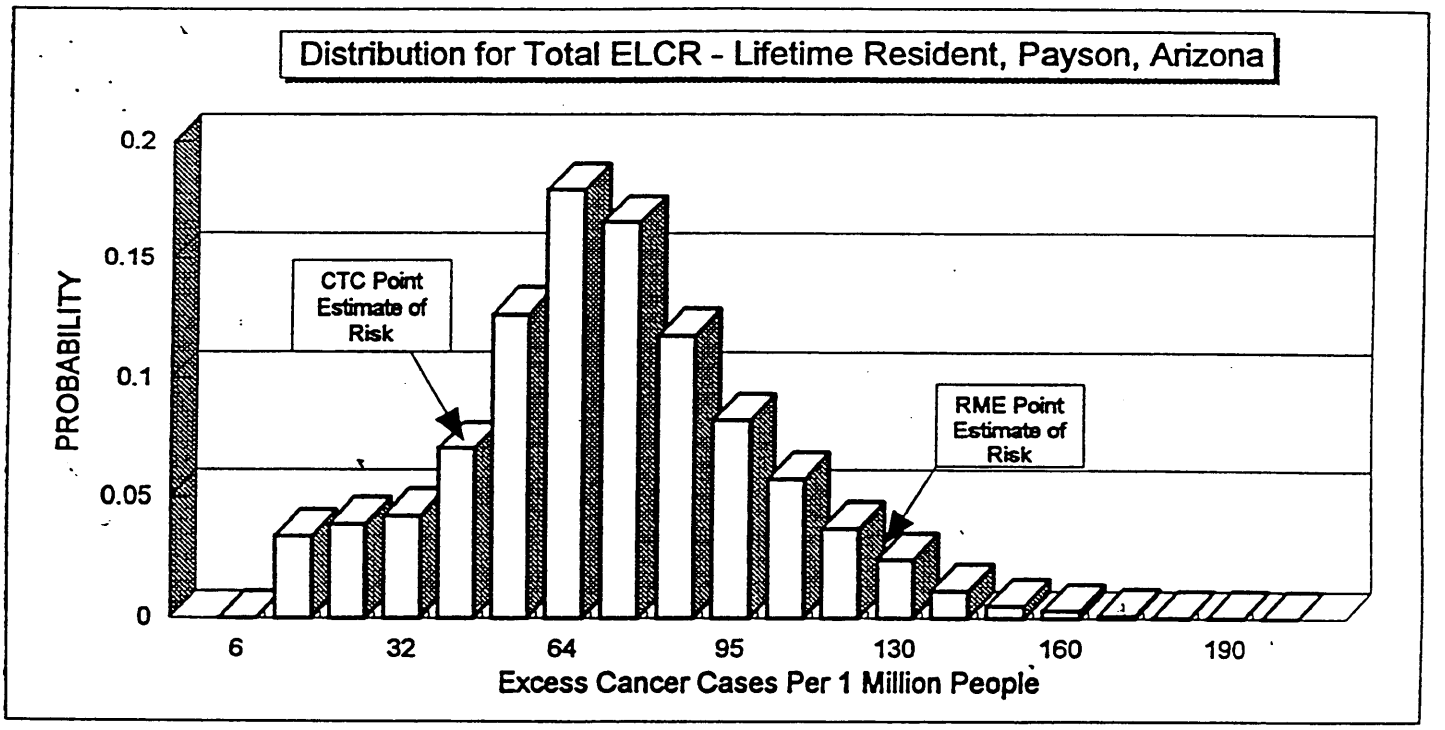

Distribution for Total ELCR - Lifetime Resident, Payson, Arizona
PROBABILITY
0.2
0.15
0.1
0.05
0
CTC Point Estimate of Risk
RME Point Estimate of Risk
6
32
64
95
130
160
190
Excess Cancer Cases Per 1 Million People

Figure 4-7

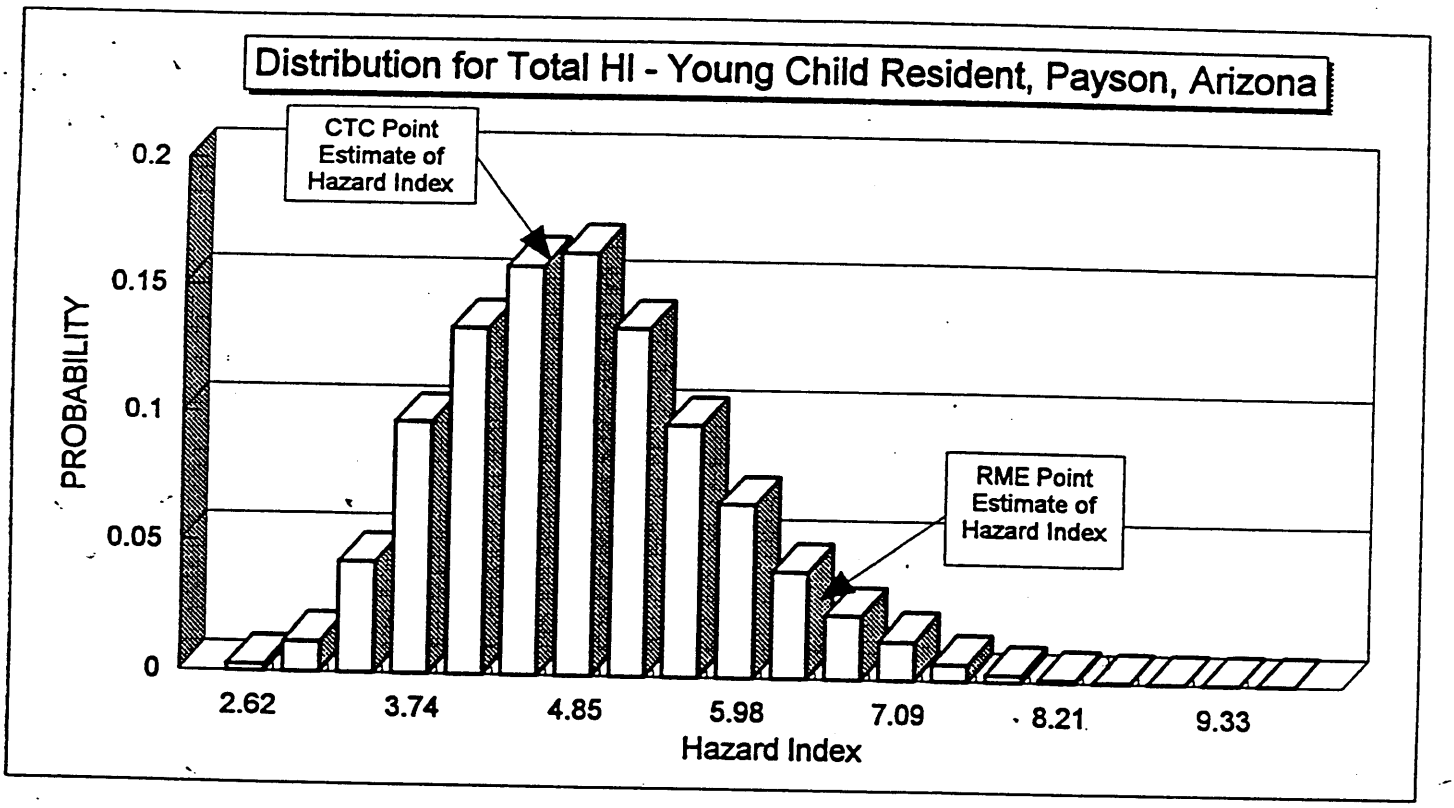

Distribution for Total HI - Young Child Resident, Payson, Arizona
CTC Point Estimate of Hazard Index
RME Point Estimate of Hazard Index
PROBABILITY
0.2
0.15
0.1
0.05
0
2.62
3.74
4.85
5.98
7.09
8.21
9.33
Hazard Index

Figure 4-8

TABLE 4-30
COMPARISON OF DETERMINISTIC AND PROBABILISTIC RESULTS FOR ESTIMATED CARCINOGENIC RISK AND NONCARCINOGENIC HAZARD INDEX
ARIZONA HAPS, PAYSON STUDY REGION, AIR
HUMAN HEALTH RISK ASSESSMENT

Resident	Distribution of Excess Lifetime Cancer Risk				RME Total Excess Lifetime Cancer Risk	CTC Total Excess Lifetime Cancer Risk
	Mean	50%	95%	Max		
Lifetime	7.05E-05	6.94E-05	1.20E-04	2.17E-04	1.25E-04	4.20E-05
Resident	**Distribution of Noncarcinogenic Hazard Index**				**RME Total Hazard Index**	**CTC Total Hazard Index**
	Mean	**50%**	**95%**	**Max**		
Young Child	5.05E+00	4.97E+00	6.74E+00	1.01E+01	6.65E+00	4.52E+00

Notes:
RME - Reasonable Maximum Exposure
CTC - Central Tendency Case

Source: ENSR 1995 TBL432.WQ1 RN: 1

TABLE 4-31
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL FUTURE NONCARCINOGENIC HAZARD INDEX
FOR THE RME SCENARIO
ARIZONA HAPS, PAYSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	9.77E-07	1.18E-06	2.58E-06	4.74E-06	3.64E-06	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	9.64E-03	1.16E-02	2.55E-02	4.68E-02	9.48E-02	3.59E-02
CADMIUM	8.80E-07	1.06E-06	2.33E-06	4.27E-06	3.27E-06	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	9.19E-02	1.11E-01	2.43E-01	4.46E-01	9.04E-01	3.42E-01
NICKEL	4.50E-08	5.42E-08	1.19E-07	2.18E-07	1.67E-07	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	7.97E-07	9.60E-07	2.11E-06	3.87E-06	2.97E-06	4.78E-02	5.76E-02	1.26E-01	2.32E-01	4.70E-01	1.78E-01
ACROLEIN	NA	NA	NA	NA	NA	1.93E-01	2.33E-01	5.11E-01	9.37E-01	1.90E+00	7.19E-01
BENZENE	2.69E-06	3.24E-06	7.12E-06	1.30E-05	1.00E-05	6.44E-02	7.75E-02	1.70E-01	3.12E-01	6.33E-01	2.40E-01
1,3-BUTADIENE	8.14E-06	9.80E-06	2.15E-05	3.95E-05	3.03E-05	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	2.26E-06	2.73E-06	5.99E-06	1.10E-05	8.42E-06	8.87E-02	1.07E-01	2.35E-01	4.30E-01	8.73E-01	3.30E-01
CHLOROFORM	5.75E-07	6.93E-07	1.52E-06	2.79E-06	2.14E-06	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1.24E-07	1.49E-07	3.28E-07	6.01E-07	4.61E-07	1.60E-05	1.93E-05	4.23E-05	7.75E-05	1.57E-04	5.95E-05
DICHLOROMETHANE	4.35E-07	5.24E-07	1.15E-06	2.11E-06	1.62E-06	3.75E-04	4.52E-04	9.93E-04	1.82E-03	3.69E-03	1.40E-03
FORMALDEHYDE	1.59E-06	1.92E-06	4.21E-06	7.72E-06	5.93E-06	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	2.15E-03	2.59E-03	5.68E-03	1.04E-02	2.11E-02	8.00E-03
TETRACHLOROETHYLENE	4.16E-08	5.00E-08	1.10E-07	2.02E-07	1.55E-07	2.16E-04	2.60E-04	5.72E-04	1.05E-03	2.13E-03	8.05E-04
TOLUENE	NA	NA	NA	NA	NA	3.36E-03	4.04E-03	8.88E-03	1.63E-02	3.30E-02	1.25E-02
TRICHLOROETHYLENE	1.06E-07	1.27E-07	2.79E-07	5.12E-07	3.93E-07	NA	NA	NA	NA	NA	NA
BENZO(A)ANTHRACENE	1.60E-08	1.93E-08	4.23E-08	7.76E-08	5.95E-08	4.27E-05	5.14E-05	1.13E-04	2.07E-04	4.20E-04	1.59E-04
BENZO(A)PYRENE	1.35E-07	1.63E-07	3.57E-07	6.55E-07	5.03E-07	3.61E-05	4.34E-05	9.54E-05	1.75E-04	3.55E-04	1.34E-04
BENZO(B+J+K)FLUORANTHENE	2.05E-08	2.46E-08	5.41E-08	9.93E-08	7.62E-08	5.46E-05	6.58E-05	1.44E-04	2.65E-04	5.37E-04	2.03E-04
CHRYSENE	1.72E-10	2.07E-10	4.56E-10	8.35E-10	6.41E-10	4.60E-05	5.54E-05	1.22E-04	2.23E-04	4.52E-04	1.71E-04
DIBENZ(A,H+A,C)ANTHRACENE	1.59E-08	1.91E-08	4.21E-08	7.71E-08	5.92E-08	4.25E-06	5.12E-06	1.13E-05	2.06E-05	4.18E-05	1.58E-05
INDENO(1,2,3-CD)PYRENE	7.43E-09	8.95E-09	1.97E-08	3.61E-08	2.77E-08	1.98E-05	2.39E-05	5.25E-05	9.62E-05	1.95E-04	7.38E-05
ACENAPHTHENE	NA	NA	NA	NA	NA	4.24E-04	5.11E-04	1.12E-03	2.06E-03	4.17E-03	1.58E-03
ACENAPHTHYLENE	NA	NA	NA	NA	NA	5.12E-04	6.17E-04	1.36E-03	2.48E-03	5.04E-03	1.91E-03
ANTHRACENE	NA	NA	NA	NA	NA	1.07E-04	1.29E-04	2.83E-04	5.20E-04	1.05E-03	3.99E-04
BENZO(B)CHRYSENE	NA	NA	NA	NA	NA	4.56E-06	5.49E-06	1.21E-05	2.21E-05	4.48E-05	1.70E-05
BENZO(G,H,I)PERYLENE	NA	NA	NA	NA	NA	3.41E-05	4.11E-05	9.02E-05	1.65E-04	3.35E-04	1.27E-04
BIPHENYL	NA	NA	NA	NA	NA	5.27E-04	6.35E-04	1.40E-03	2.56E-03	5.19E-03	1.96E-03
1,2-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	4.90E-04	5.90E-04	1.30E-03	2.38E-03	4.82E-03	1.82E-03
1,3+1,6+1,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.92E-03	2.31E-03	5.08E-03	9.32E-03	1.89E-02	7.15E-03
1,4+1,5+2,3-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	5.77E-04	6.95E-04	1.53E-03	2.80E-03	5.67E-03	2.15E-03
1,8-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.26E-05	1.51E-05	3.32E-05	6.09E-05	1.23E-04	4.67E-05
2,6+2,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	8.79E-04	1.06E-03	2.32E-03	4.26E-03	8.64E-03	3.27E-03
FLUORANTHENE	NA	NA	NA	NA	NA	1.98E-04	2.38E-04	5.23E-04	9.59E-04	1.94E-03	7.36E-04
FLUORENE	NA	NA	NA	NA	NA	4.44E-04	5.35E-04	1.18E-03	2.16E-03	4.37E-03	1.65E-03
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	4.44E-03	5.35E-03	1.18E-02	2.15E-02	4.37E-02	1.65E-02
2-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	9.55E-03	1.15E-02	2.53E-02	4.63E-02	9.39E-02	3.55E-02
NAPHTHALENE	NA	NA	NA	NA	NA	1.58E-02	1.91E-02	4.19E-02	7.68E-02	1.56E-01	5.90E-02
PHENANTHRENE	NA	NA	NA	NA	NA	6.91E-04	8.32E-04	1.83E-03	3.35E-03	6.79E-03	2.57E-03
PYRENE	NA	NA	NA	NA	NA	1.60E-04	1.92E-04	4.22E-04	7.74E-04	1.57E-03	5.94E-04
TOTAL	1.89E-05	2.27E-05	4.99E-05	9.15E-05	7.02E-05	5.39E-01	6.49E-01	1.43E+00	2.61E+00	5.30E+00	2.01E+00

Notes:

Source: EKSR 1996

RRISK.WO1

R.N.: 3

TABLE 4-32
SUMMARY OF TOTAL FUTURE INCREMENTAL CARCINOGENIC RISK
AND TOTAL FUTURE NONCARCINOGENIC HAZARD INDEX
FOR THE CTC SCENARIO
ARIZONA HAPS, PAYSON, AIR PATHWAY
HUMAN HEALTH RISK ASSESSMENT

Compound	Total Future Incremental Carcinogenic Risk					Total Future Noncarcinogenic Hazard Index					
	Lifetime Resident					Lifetime Resident					
	Young Child	Older Child	Adult	Total Lifetime Resident	Adult Resident	Young Child	Older Child	Adult	Total Lifetime Resident	Young Child Resident	Adult Resident
ARSENIC	6.65E-07	7.72E-07	1.49E-07	1.59E-06	9.52E-07	NA	NA	NA	NA	NA	NA
BARIUM	NA	NA	NA	NA	NA	1.84E-02	2.14E-02	4.13E-03	4.40E-02	6.45E-02	2.64E-02
CADMIUM	5.99E-07	6.95E-07	1.34E-07	1.43E-06	8.57E-07	NA	NA	NA	NA	NA	NA
MANGANESE	NA	NA	NA	NA	NA	1.76E-01	2.04E-01	3.94E-02	4.19E-01	6.15E-01	2.52E-01
NICKEL	3.06E-08	3.56E-08	6.87E-09	7.31E-08	4.38E-08	NA	NA	NA	NA	NA	NA
ACETALDEHYDE	5.43E-07	6.30E-07	1.22E-07	1.29E-06	7.77E-07	9.14E-02	1.06E-01	2.05E-02	2.18E-01	3.20E-01	1.31E-01
ACROLEIN	NA	NA	NA	NA	NA	3.69E-01	4.29E-01	8.28E-02	8.81E-01	1.29E+00	5.29E-01
BENZENE	1.83E-06	2.13E-06	4.11E-07	4.37E-06	2.62E-06	1.23E-01	1.43E-01	2.76E-02	2.94E-01	4.31E-01	1.76E-01
1,3-BUTADIENE	5.54E-06	6.43E-06	1.24E-06	1.32E-05	7.93E-06	NA	NA	NA	NA	NA	NA
CARBON TETRACHLORIDE	1.54E-06	1.79E-06	3.45E-07	3.67E-06	2.21E-06	1.70E-01	1.97E-01	3.80E-02	4.05E-01	5.94E-01	2.43E-01
CHLOROFORM	3.91E-07	4.55E-07	8.78E-08	9.34E-07	5.60E-07	NA	NA	NA	NA	NA	NA
1,4-DICHLOROBENZENE	8.44E-08	9.80E-08	1.89E-08	2.01E-07	1.21E-07	3.06E-05	3.55E-05	6.85E-06	7.29E-05	1.07E-04	4.38E-05
DICHLOROMETHANE	2.96E-07	3.44E-07	6.64E-08	7.07E-07	4.24E-07	7.18E-04	8.34E-04	1.61E-04	1.71E-03	2.51E-03	1.03E-03
FORMALDEHYDE	1.08E-06	1.26E-06	2.43E-07	2.59E-06	1.55E-06	NA	NA	NA	NA	NA	NA
N-HEXANE	NA	NA	NA	NA	NA	4.11E-03	4.77E-03	9.21E-04	9.80E-03	1.44E-02	5.88E-03
TETRACHLOROETHYLENE	2.83E-08	3.28E-08	6.34E-09	6.75E-08	4.05E-08	4.13E-04	4.80E-04	9.27E-05	9.86E-04	1.45E-03	5.92E-04
TOLUENE	NA	NA	NA	NA	NA	6.42E-03	7.46E-03	1.44E-03	1.53E-02	2.25E-02	9.19E-03
TRICHLOROETHYLENE	7.19E-08	8.35E-08	1.61E-08	1.71E-07	1.03E-07	NA	NA	NA	NA	NA	NA
BENZO(A)ANTHRACENE	1.09E-08	1.26E-08	2.44E-09	2.60E-08	1.56E-08	8.16E-05	9.48E-05	1.83E-05	1.95E-04	2.86E-04	1.17E-04
BENZO(A)PYRENE	9.19E-08	1.07E-07	2.06E-08	2.19E-07	1.32E-07	6.89E-05	8.01E-05	1.55E-05	1.64E-04	2.41E-04	9.87E-05
BENZO(B+J+K)FLUORANTHENE	1.39E-08	1.62E-08	3.12E-09	3.32E-08	1.99E-08	1.04E-04	1.21E-04	2.34E-05	2.49E-04	3.65E-04	1.50E-04
CHRYSENE	1.17E-10	1.36E-10	2.63E-11	2.80E-10	1.68E-10	8.79E-05	1.02E-04	1.97E-05	2.10E-04	3.08E-04	1.26E-04
DIBENZ(A,H+A,C)ANTHRACENE	1.08E-08	1.26E-08	2.43E-09	2.58E-08	1.55E-08	8.13E-06	9.45E-06	1.82E-06	1.94E-05	2.85E-05	1.16E-05
INDENO(1,2,3-CD)PYRENE	5.06E-09	5.88E-09	1.13E-09	1.21E-08	7.24E-09	3.79E-05	4.41E-05	8.51E-06	9.05E-05	1.33E-04	5.43E-05
ACENAPHTHENE	NA	NA	NA	NA	NA	8.11E-04	9.42E-04	1.82E-04	1.94E-03	2.84E-03	1.16E-03
ACENAPHTHYLENE	NA	NA	NA	NA	NA	9.79E-04	1.14E-03	2.20E-04	2.34E-03	3.43E-03	1.40E-03
ANTHRACENE	NA	NA	NA	NA	NA	2.05E-04	2.38E-04	4.59E-05	4.89E-04	7.17E-04	2.93E-04
BENZO(B)CHRYSENE	NA	NA	NA	NA	NA	8.72E-06	1.01E-05	1.96E-06	2.08E-05	3.05E-05	1.25E-05
BENZO(G,H,I)PERYLENE	NA	NA	NA	NA	NA	6.52E-05	7.57E-05	1.46E-05	1.56E-04	2.28E-04	9.33E-05
BIPHENYL	NA	NA	NA	NA	NA	1.01E-03	1.17E-03	2.26E-04	2.41E-03	3.53E-03	1.44E-03
1,2-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	9.37E-04	1.09E-03	2.10E-04	2.24E-03	3.28E-03	1.34E-03
1,3+1,6+1,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	3.68E-03	4.27E-03	8.24E-04	8.77E-03	1.29E-02	5.26E-03
1,4+1,5+2,3-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.10E-03	1.28E-03	2.47E-04	2.63E-03	3.86E-03	1.58E-03
1,8-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	2.40E-05	2.79E-05	5.38E-06	5.73E-05	8.40E-05	3.44E-05
2,6+2,7-DIMETHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.68E-03	1.95E-03	3.77E-04	4.01E-03	5.88E-03	2.41E-03
FLUORANTHENE	NA	NA	NA	NA	NA	3.78E-04	4.39E-04	8.48E-05	9.02E-04	1.32E-03	5.41E-04
FLUORENE	NA	NA	NA	NA	NA	8.50E-04	9.87E-04	1.91E-04	2.03E-03	2.97E-03	1.22E-03
1-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	8.49E-03	9.87E-03	1.90E-03	2.03E-02	2.97E-02	1.22E-02
2-METHYLNAPHTHALENE	NA	NA	NA	NA	NA	1.83E-02	2.12E-02	4.09E-03	4.36E-02	6.39E-02	2.61E-02
NAPHTHALENE	NA	NA	NA	NA	NA	3.03E-02	3.52E-02	6.79E-03	7.23E-02	1.06E-01	4.34E-02
PHENANTHRENE	NA	NA	NA	NA	NA	1.32E-03	1.53E-03	2.96E-04	3.15E-03	4.62E-03	1.89E-03
PYRENE	NA	NA	NA	NA	NA	3.05E-04	3.54E-04	6.84E-05	7.28E-04	1.07E-03	4.37E-04
TOTAL	1.28E-05	1.49E-05	2.88E-06	3.06E-05	1.84E-05	1.03E+00	1.20E+00	2.31E-01	2.46E+00	3.61E+00	1.47E+00

Notes:

Source: ENSR 1995 RRISK.WQ1 R.N.: 3

TABLE 4-33
COMPARISON OF DETERMINISTIC AND PROBABILISTIC RESULTS
FOR ESTIMATED TOTAL INCREMENTAL CARCINOGENIC RISK
ARIZONA HAPS, ALL REGIONS
HUMAN HEALTH RISK ASSESSMENT

Study Region	Distribution of Excess Lifetime Cancer Risk				RME Total Excess Lifetime Cancer Risk	CTC Total Excess Lifetime Cancer Risk
	Mean	50%	95%	Max		
Phoenix	1.70E-04	1.59E-04	3.28E-04	7.93E-04	5.36E-04	9.77E-05
Tucson	1.26E-04	1.21E-04	2.28E-04	3.78E-04	3.14E-04	7.53E-05
Casa Grande	4.63E-05	4.33E-05	9.06E-05	1.67E-04	1.42E-04	2.92E-05
Payson	7.05E-05	6.94E-05	1.20E-04	2.17E-04	1.25E-04	4.20E-05

Notes:
RME - Reasonable Maximum Exposure
CTC - Central Tendency Case

2.8 2009.

Source: ENSR 1995 TBL433.WQ1 RN:2

TABLE 4-34
COMPARISON OF DETERMINISTIC AND PROBABILISTIC RESULTS
FOR ESTIMATED TOTAL NONCARCINOGENIC HAZARD INDEX
ARIZONA HAPS, ALL REGIONS
HUMAN HEALTH RISK ASSESSMENT

Study Region	Distribution of Noncarcinogenic Hazard Index				RME Hazard Index	CTC Hazard Index
	Mean	50%	95%	Max		
Phoenix	7.00E+00	7.00E+00	1.00E+01	1.80E+01	1.50E+01	6.00E+00
Tucson	3.88E+00	3.78E+00	5.57E+00	8.25E+00	7.08E+00	3.48E+00
Casa Grande	3.41E+00	3.30E+00	5.29E+00	8.02E+00	7.66E+00	3.05E+00
Payson	5.05E+00	4.97E+00	6.74E+00	1.01E+01	6.65E+00	4.52E+00

Notes:
RME - Reasonable Maximum Exposure
CTC - Central Tendency Case

Source: ENSR 1995 TBL433.WQ1 RN: 3

ATTACHMENT F-1

DOSE RESPONSE PROFILES FOR

- ELEMENTS
- VOLATILE ORGANIC COMPOUNDS
- POLYNUCLEAR AROMATIC HYDROCARBONS
- PESTICIDES

PLUS

- ECAO RISK ASSESSMENT ISSUE PAPERS

ATTACHMENT F-1

DOSE RESPONSE PROFILES FOR

- ELEMENTS
- VOLATILE ORGANIC COMPOUNDS
- POLYNUCLEAR AROMATIC HYDROCARBONS
- PESTICIDES

PLUS

- ECAO RISK ASSESSMENT ISSUE PAPERS

ELEMENTS

ARSENIC

Inorganic arsenic has been classified by the U.S. EPA as a known human carcinogen (Group A). IRIS (U.S. EPA, 1995) provides a cancer slope factor for the inhalation route of exposure. There was no available inhalation reference dose for arsenic.

Derivation of the Inhalation Cancer Slope Factor

IRIS (U.S. EPA, 1995) reports the inhalation unit risk for arsenic to be 4.3E-03 $(\mu g/m^3)^{-1}$. The inhalation cancer slope factor of 1.5E+01 $(mg/kg\text{-}day)^{-1}$ was calculated assuming a 70 kg human body weight and 20 m^3 air inhaled/day. The unit risk should not be used if the air concentration exceeds 2 $\mu g/m^3$, since above this concentration the slope factor may differ. The estimate is the geometric mean of values from two studied populations of smelter workers (U.S. EPA, 1984). Confidence in the CSF is high because a large study population was observed. Exposure assessments included air measurements for one smelter and both air measurements and urinary arsenic levels for another smelter. Observed lung cancer incidence was significantly increased over expected values. The range of the estimates derived from data from the two different exposure areas was within a factor of 6.

REFERENCES

U.S. EPA. 1984. Health Effects Assessment of Arsenic. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Emergency and Remedial Response, Washington, DC.

U.S. EPA. 1995. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

BARIUM

According to IRIS (U.S. EPA, 1995a), barium has not been evaluated by the U.S. EPA for evidence of carcinogenic potential. The dose-response assessment for barium, therefore, is based on noncarcinogenic health endpoints. An inhalation RfD is not available at this time on IRIS, but an reference concentration (RfC) is provided in the Health Effects Assessment Summary Tables (U.S. EPA, 1995b).

Derivation of the Inhalation Reference Dose

The Health Effects Assessment Summary Tables (U.S. EPA, 1995b) report an inhalation RfC of 5.0E-04 mg/kg-day. Based on this value, an inhalation RfD of 1.43E-04 was derived, assuming a 70 kg adult inhales 20 m^3 of air/day.

The RfC is based on a study by Tarasenko et al. (1977) which reported increased fetotoxicity in rats exposed to $BaCO_3$. Exposure of male rats to atmospheric concentrations of 3.62 mg Ba/m^3, 4 hours per day, for 4 months, resulted in general signs of toxicity that included decreased body weight and liver function, and increased mortality of fetuses following mating of treated males with untreated females. Exposure to 0.80 mg/m^3 resulted in no observed toxic effects in the adult males; however, reproductive performance was not reported for this group. The exposure level of 0.80 mg/m^3 is considered the NOEL. An uncertainty factor of 1000 was used to account for the subchronic nature of the study, interspecies variation, and in order to be protective of sensitive human subpopulations.

REFERENCES

Tarasenko, M., O. Promin and A. Silayev. 1977. Barium compounds as industrial poisons (an experimental study). J. Hyg. Epidem. Microbiol. Immunol. 21: 361.

U.S. EPA. 1995a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1995b. Health Effects Assessment Summary Tables (HEAST). Annual, F.Y. 1995. Office of Solid Waste and Emergency Response, Washington, DC.

CADMIUM

Cadmium is classified by the U.S. EPA as a Group B1 carcinogen (a probable human carcinogen) via inhalation. Insufficient data exist to classify cadmium as carcinogenic by the oral route. A unit cancer risk is listed in IRIS (U.S. EPA, 1995) for the inhalation route, however there is no inhalation RfD available.

Derivation of the Inhalation Cancer Slope Factor

IRIS (U.S. EPA, 1995) reports an inhalation unit risk of 1.8E-03 $(\mu g/m^3)^{-1}$. Assuming an inhalation rate of 20 m^3/day and a body weight of 70 kg, this converts to an inhalation cancer slope factor of 6.3 $(mg/kg\text{-}day)^{-1}$. Based on limited evidence from epidemiologic studies, the slope factor is supported by sufficient evidence of carcinogenicity in rats and mice by two routes.

A two-fold excess of cases of lung cancer was observed in a cohort of cadmium smelter workers. Urine cadmium data available for 261 workers suggested a highly exposed population. The investigators (Thun et al., 1985) were able to control for confounding factors to estimate lung cancer risk. An evaluation by the Human Health Assessment Group of such confounding factors as smoking and arsenic exposure indicated that the assumptions and methods used in accounting for them may not be valid. Because of the lack of clear cut causal evidence of cadmium exposure only, this study is considered to supply limited evidence of human carcinogenicity.

Excess lung cancer risk was also observed in three other studies which were compromised by the presence of other carcinogens (arsenic, smoking) in the exposure. Four studies of workers exposed to cadmium dust or fumes provided evidence of statistically significant positive association with prostate cancer, but the total number of cases was small in each study.

Inhalation exposure of Wistar rats to cadmium as cadmium chloride at concentrations of 12.5, 25 and 50 $\mu g/m^3$ for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors (Takenaka et al., 1983). Intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats but rather mammary tumors in females and tumors at multiple sites in males (Sanders and Mahaffey, 1984).

A slope factor was derived from cadmium chloride inhalation assay data in rats (Takenaka et al., 1983). While this estimate was higher than that derived from human data and thus more conservative, the human data was used because it was felt that it was more reliable because of

species variations in response, and the type of exposure (cadmium salt vs. cadmium fume and cadmium oxide).

REFERENCES

Sanders, C.L. and J.A. Mahaffey. 1984. Carcinogenicity of single and multiple intratracheal instillations of cadmium oxide in the rat. Environ. Res. 33: 227-233.

Takenaka, S., H. Oldiges, H. Konig, D. Hochrainer and G. Oberdorster. 1983. Carcinogenicity of cadmium chloride aerosols in W rats. J. Natl. Cancer Inst. 70: 367-373.

Thun, M.J., T.M. Schnorr, A.B. Smith and W.E. Halperin. 1985. Mortality among a cohort of U.S. cadmium production workers: An update. J. Natl. Cancer Inst. 74(2): 325-233.

U.S. EPA. 1995. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

CHROMIUM VI

Chromium VI is classified by U.S. EPA as a Group A carcinogen (known human carcinogen) by the inhalation route. An inhalation cancer slope factor is listed in HEAST (U.S. EPA, 1995). An inhalation reference dose (RfD) is not available for chromium VI.

Derivation of the Inhalation Cancer Slope Factor

HEAST (U.S. EPA, 1995) reports an inhalation cancer slope factor (CSF) of 4.1E+01 (mg/kg-day)$^{-1}$. This value is based on one of the many epidemiologic studies that have been performed. Dose-response relationships for lung tumors have been established in epidemiologic studies of chromate production facilities in the U.S. An epidemiological study by Mancuso (1975) was used as the basis for the development of the inhalation CSF. Specifically, the increased lung cancer mortality in the study by Mancuso (1975) was assumed to be due to Cr VI, which was further assumed to be no less than one-seventh of total chromium to which the workers were exposed. In order to account for the effects of cigarette smoking in lung cancer incidence, it was also assumed that the smoking habits of chromate workers were similar to those of the U.S. white male population.

REFERENCES

Mancuso, T.F. 1975. International Conference on Heavy Metals in the Environment, Toronto, Canada, Oct. 27-31. (Cited in U.S. EPA, 1984)

U.S. EPA. 1995. Health Effects Assessment Summary Tables (HEAST). Annual, F.Y. 1995. Office of Solid Waste and Emergency Response, Washington, DC.

MANGANESE

Manganese is classified by the U.S. EPA (1995) as a Class D carcinogen (not classifiable as to human carcinogenicity), and no inhalation cancer slope factors are provided. IRIS (U.S. EPA, 1995) reports an inhalation reference concentration (RfC) for manganese.

Derivation of the Inhalation Reference Dose

IRIS (U.S. EPA, 1995) reported an inhalation RfC 5.0E-05 mg/m^3. The RfC is based on two epidemiological studies (Roels et al. 1987, 1992) of worker exposure to manganese. This corresponds to an inhalation RfD of 1.43E-05, assuming a 70 kg adult inhales 20 m^3 air/day.

The 1987 (Roels et al.) study was a cross-sectional study of 141 male workers exposed to MnO_2, manganese tetroxide, and various Mn salts. The workers were matched with a control group of 104 male workers based on socioeconomic status, background environmental factors, work-load and work-shift characteristics.

The time weighted average (TWA) of total airborne Mn dust ranged from 0.07 to 8.61 mg/m^3, with an overall arithmetic mean of 1.33 mg/m^3. The duration of employment ranged from 1 to 19 years, with a mean of 7.1 years. A neurological examination, nuerobehavioral function tests (simple reaction time, short-term memory, eye-hand coordination, hand tremor), spirographic measurements, blood and urine tests, and self-administered questionnaires were used to assess potential effects of Mn exposures.

Significant differences were found between mean scores of the exposed group and the scores of the control group for the objective tests (i.e., reaction time; short-term memory). Subjective symptoms reported in the questionnaires were more prevalent for the exposed group than the controls for 20 of the 25 symptoms listed. Four items, fatigue, tinnitus, trembling fingers, and irritability were statistically significant. Based on the results of the study, a LOAEL of 0.97 mg/m^3 was identified.

Roels et al. (1992) conducted a cross-sectional study of 92 male workers exposed to manganese dioxide dust in a Belgian alkaline battery plant. A control group of 101 male workers were matched for age, height, weight, work schedule, coffee and alcohol consumption, and smoking. The exposed group were exposed to Mn for an average of 5.3 years. Airborne Mn concentration were determined using personal samplers in the breathing zone; resulting in a mean concentration of 0.948 mg/m^3 for total dust. An occupational lifetime integrated exposure was

Roels, H.A., P. Ghyselen, J.P. Buchet, E. Ceulemans, and R.R. Lauwerys. 1992. Assessment of the permissible exposure level to manganese in workers exposed to manganese dioxide dust. Br. J. Ind. Med. 49: 25-34.

U.S. EPA. 1995. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

Wennberg, A., A. Iregren, G. Struwe, G. Cizinsky, M. Hagman, and L. Johansson. 1991. Manganese exposure to steel smelters a health hazard to the nervous system. Scand. J. Work Environ. Health. 17: 255-262.

Wennberg, A., M. Hagman, and L. Johansson. 1992. Preclinical neurophysiological signs of parkinsonism in occupational manganese exposure. Neurotoxicology. 13: 271-274.

estimated for each worker by multiplying the measured concentrations by number of years at each job classification.

A self-administered questionnaire was administered and focused on occupational and medical histories, neurological complaints, and respiratory symptoms. Neurobehavioral functions were evaluated using short-term memory tests, reaction times, hand steadiness, and eye-hand coordination. Lung function, blood samples, and urine samples were also evaluated.

The Mn exposed workers performed worse than the controls for several nuerobehavioral functions, including visual reaction time, eye-hand coordination, and hand tremor. Roels et al. indicated that a LOAEL for airborne Mn dust could not be identified from the group data. The EPA derived a LOAEL of 0.15 mg/m^3 by dividing the geometric mean of an integrated respirable dust (IRD) concentration (0.793 mg/m^3 x years) by the average duration of worker exposure to Mn dust (5.3 years).

The results of the two studies indicate that the nervous system is a more sensitive target for Mn toxicity. The findings of the two studies are supported by other recent reports that provide comparable and consistent indication of nuerobehavioral function impairments in Mn exposed workers (Mergler et al., 1993; Iregren, 1990; Wennberg et al., 1991,1992).

The EPA's confidence in the principal studies (Roels et al., 1987, 1992) is medium. The two studies did not provide NOAELs for the nuerobehavioral effects, and the duration of exposures were limited in the studies. The 1987 study only measured total dust, while the 1992 study provided respirable and total dust. In addition, the 1987 study did not distinguish the species of Mn or proportions of Mn compounds to which workers were exposed, making it impossible to determine relative toxicities of the different compounds.

REFERENCES

Iregren, A. 1990. Psychological test performance in foundry workers exposed to low levels of manganese. Neurotoxicol. Teratol. 12: 673-675.

Mergler, D., G. Huel, R. Bowler et al. 1993. Nervous system dysfunction among workers with long-term exposure to manganese. Environ. Res. (In press).

Roels, H., R. Lauwerys, J.P. Buchet et al. 1987. Epidemiological survey among workers exposed to manganese: Effects on lung, central nervous system, and some biological indices. Am. J. Ind. Med. 11: 307-327.

NICKEL

The U.S. EPA has not evaluated nickel soluble salts, as a class of compounds, for evidence of human carcinogenic potential. Assessments of nickel refinery dust and nickel subsulfide indicate that inhalation of particulate forms of nickel leads to cancer. The U.S. EPA has classified nickel as a known human carcinogen by the inhalation route (Group A) (U.S. EPA, 1995).

The U.S. EPA derived inhalation cancer slope factors that are verified and are available on IRIS for both nickel refinery dust and nickel subsulfide (U.S. EPA, 1995). The inhalation cancer slope factor for nickel refinery dust will be used to represent environmental exposure situations since it is composed of several nickel moieties, whereas the applicability of the cancer slope factor for nickel subsulfide is more limited.

Derivation of the Inhalation Cancer Slope Factor

Ideally, the cancer slope factor would have been derived for the form of nickel with which potential human exposure is anticipated to occur. Since information regarding the chemical form of nickel is not available, the cancer slope factor for nickel refinery dust is assumed to be the most representative of potential environmental exposure situations since it consists of several nickel moieties (U.S. EPA, 1995).

The U.S. EPA derived the cancer slope factor for nickel refinery dust from several epidemiological studies which concluded that nickel caused lung and nasal tumors in sulfide nickel matte refinery workers in several different countries. The data used in the slope estimate were from Huntington, WV (Enterline and Marsh, 1982), Copper Cliff, Ontario (Chovil et al., 1981), Clydach, Wales (Peto et al., 1984), and Kristians, Norway (Magnus et al., 1982). These studies consistently report specificity of tumor site (nasal and lung), high relative risks, especially for nasal cancer, and a dose-response relationship by length of exposure. Excess risks were greatest in the dustier areas of the refineries.

The dose-response curves for nasal cancer were not used in deriving the slope estimate because nasal cancer risk from nickel is thought to be an occupational hazard associated only with the pyrometallurgical process, and these tumors are not found in the general public to the same extent as lung tumors. Thus, the data for lung cancer alone was used. For the four data sets analyzed, both the additive and multiplicative (relative) excess risk models were fitted whenever possible. The average relative risk model was applied to the data from Huntington, WV and Copper Cliff, Ontario.

MERCURY

Mercury has been classified by the U.S. EPA as Group D; i.e., not classifiable as to human carcinogenic potential (U.S. EPA, 1995). The dose-response assessment for mercury, therefore, is based on noncarcinogenic health endpoints.

Derivation of the Inhalation Reference Dose

IRIS (U.S. EPA, 1995a) reports an inhalation reference concentration for inorganic mercury of 0.3 $\mu g/m^3$. This corresponds to an RfD of 8.57E-05 mg/kg-day assuming 20 m^3 inhalation by a 70 kg adult. The reported value was based on several occupational studies involving exposed workers evaluated for neurotoxic effects (U.S. EPA, 1995). An uncertainty factor of 30 was applied to the concentration of 9 $\mu g/m^3$ to develop the reference concentration (U.S. EPA, 1995). No inhalation RfCs were reported for other forms of mercury. The U.S. EPA has a medium confidence level in this RfC.

REFERENCES

U.S. EPA. 1995. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

The unit risk estimates ranged from 1.5×10^{-5} to 4.6×10^{-4} $(\mu g/mg^3)^{-1}$. As a best estimate, the midpoint of the range, 2.4×10^{-4} $(\mu g/m^3)^{-3}$, was taken as the incremental unit risk resulting from a lifetime of exposure to nickel matte refinery dust. This unit risk should not be used if the air concentration exceeds 40 $\mu g/m^3$. From this unit risk, an inhalation cancer slope factor of 0.84 $(mg/kg/day)^{-1}$ was derived. The U.S. EPA's confidence in the cancer slope estimate is high since four data sets, all from humans offer a range of unit risk estimates which are consistent with each other (U.S. EPA, 1995).

REFERENCES

Chovil, A., R.B. Sutherland and M. Halliday. 1981. Respiratory cancer in a cohort of sinter plant workers. Br. J. Ind. Med. 38: 327-333.

Enterline, P.E. and G.M. Marsh. 1982. Mortality among workers in a nickel refinery and alloy manufacturing plant in West Virginia. J. Natl. Cancer Inst. 68(6): 925-933.

Magnus, K., A. Andersen and A. Hogetveit. 1982. Cancer of respiratory organs among workers at a nickel refinery in Norway. Int. J. Cancer. 30: 681-685.

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VOLATILE ORGANIC COMPOUNDS

ACROLEIN

IRIS (U.S. EPA, 1995a) classifies acrolein as Class C, a possible human carcinogen. There are no inhalation cancer slope factors available on IRIS or the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b). IRIS reports an inhalation reference concentration (RfC) for acrolein.

Derivation of the Inhalation Reference Dose

The inhalation RfD is based on an RfC of 2.0E-05 mg/m^3 reported in IRIS (U.S. EPA, 1995a), which corresponds to an RfD of 5.71E-06 mg/kg-day. This assumes a 70 kg adult will inhale 20 m^3 air/day.

The RfC is based on two inhalation studies by Kutzman (1981) and Feron et al. (1978). The Kutzman study was a subchronic study of male F344 rats exposed to acrolein concentrations of 0, 0.4, 1.4, and 4.0 ppm 6 hours/day, 5 days/week for 62 days. Twenty four of the exposed animals were assessed for pulmonary function. The animals were then sacrificed, and one lung was used for compositional testing, and the other lung was used for pathological examination. Rats exposed to 4 ppm had a high mortality rate and reduced weight gain among survivors of both sexes. Growth in the low and medium concentration levels was similar to control animals, and no remarkable pathology was observed in the extrarespiratory tissues. The nasal turbinate exhibited a concentration-dependant increase in submucosal lymphoid aggregates and rhinitis. A LOAEL of 0.4 ppm for respiratory effects in the nasal cavity was estimated from the results of the study.

Feron et al. (1978) exposed Syrian Golden hamsters, Wistar rats, and Dutch rabbits to acrolein concentrations of 0, 0.4, 1.4, and 4.9 ppm for 6 hours/day, 5 days/week for 13 weeks. Rats appeared to be most sensitive to the exposures. Exposure related histopathological changes were observed only in the respiratory tract. At the highest levels of exposure (4.9 ppm), marked changes in epithelial lining of the nasal cavity and neutrophilic infiltration were observed. At intermediate levels of exposure, squamous metaplasia and neutrophilic infiltration was "moderately affected". At the lowest exposure level, one male rat was reported to slight metaplastic and inflammatory changes. A LOAEL of 0.4 ppm for respiratory effects in the nasal cavity was estimated from the results of the study. The NOAEL and LOAEL for respiratory effects in the tracheobronchial region were estimated to be 1.4 and 4.9 ppm, respectively.

The LOAEL of 0.4 ppm was adjusted for exposure time, resulting in a NOAEL(ADJ) of 0.164 mg/m^3. A human equivalency conversion (HEC) factor was then applied to arrive at a NOAEL(HEC) of 0.02 mg/m^3.

To derive the RfC, an uncertainty factor of 1000 was applied to the NOAEL(HEC): 10 to account for sensitive human populations; 10 for both the uncertainty in the interspecies extrapolation using dosimetric adjustments and to account for the mild nature of the LOAEL; 10 to account for the lack of chronic studies. The U.S. EPA has high confidence in the principal study, medium confidence in the database, and a medium level of confidence in the derived RfC.

References

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U.S. EPA. 1995b. Health Effects Summary Tables (HEAST). Annual, F.Y. 1995. Office of Solid Waste and Emergency Response.

ACETALDEHYDE

IRIS (U.S. EPA, 1995) classifies acetaldehyde as a Class B2, probable human carcinogen, and provides an inhalation unit risk. IRIS also reports an inhalation reference concentration (RfC) for acetaldehyde.

Derivation of the Inhalation Cancer Slope Factor

An inhalation cancer slope factor of 7.7E-03 $(mg/kg\text{-}day)^{-1}$ was derived from an inhalation unit risk of 6.2E-4 ug/m^3, assuming a 70 kg adult inhales 20 m^3 air/day. The U.S. EPA calculated the unit risk based on a study of 420 male and 420 female albino SPF Wistar rats assigned to four groups of 105 males and 105 females each (Woutersen and Appelman, 1984; Woutersen et al., 1985). The rats were exposed by inhalation to atmospheres containing 0, 750, 1500, or 3000 ppm of acetaldehyde for 6 hours/day, 5 days/week, for 27 months. Interim sacrifices at 13, 26, and 52 weeks indicated one tumor in the 52 week sacrifice and no tumors in the earlier sacrifices.

The exposure to acetaldehyde increased the incidence of multiple respiratory tumors in both the male and female rats. Adenocarcinomas were increased significantly in both male and female rats at all exposure levels, however, squamous cell carcinomas were increased in male rats at middle and high doses and in females at high doses only.

In a concurrent study, 30 male and 30 female rats were exposed to the same concentrations of acetaldehyde for 52 weeks, followed by a recovery period of 26 or 52 weeks. The results of the study indicated increases in nasal tumors in both male and female rats.

Derivation of the Inhalation Reference Dose

The inhalation RfD is based on an RfC of 9.0E-03 mg/m^3 reported in IRIS (U.S. EPA, 1995), which corresponds to an RfD of 2.57E-03 mg/kg-day. This assumes a 70 kg adult will inhale 20 m^3 air/day.

The RfC is based on two short-term studies by Appelman et al. (1982, 1986). In the 1982 study, male and female Wistar rats for 6 hours/day, 5 days/week for 4 weeks to 0, 400, 1000, 2200, or 5000 ppm acetaldehyde. Compound related histopathological changes were observed only in the respiratory system, with the nasal cavity most severely affected. At the 400 ppm level, changes observed were slight to severe degeneration of the nasal olfactory epithelium; at 1000 to 2000 ppm levels, more severe degenerative changes were observed in the nasal cavity.

Degeneration also occurred in the laryngeal and tracheal epithelium at these levels. At 5000 ppm, changes included severe degenerative hyperplastic and metaplastic changes of the nasal, laryngeal and tracheal epithelium. Based on these results, a NOAEL of 273 mg/m^3 was estimated. This was adjusted for exposure time, resulting in a NOAEL of 48.75 mg/m^3. A human equivalency conversion (HEC) factor was then applied to arrive at a NOAEL(HEC) of 8.7 mg/m^3.

To derive the RfC, an uncertainty factor of 1000 was applied to the NOAEL(HEC): 10 to account for sensitive human populations; 10 for both the uncertainty in the interspecies extrapolation using dosimetric adjustments and to account for incompleteness in the data base; 10 to account for extrapolation from subchronic to chronic. The U.S. EPA has medium confidence in the principal study, a low confidence in the data base, and a low confidence in the RfC results.

References

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ACRYLONITRILE

The U.S. EPA (1995a) classifies acrylonitrile as Class B1, a probable human carcinogen, and provides an inhalation reference concentration (RfC) for acrylonitrile. The Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b) provide an inhalation cancer slope factor (CSF).

Derivation of the Inhalation Cancer Slope Factor

An inhalation cancer slope factor of 2.4E-01 $(mg/kg\text{-}day)^{-1}$ was derived from an inhalation unit risk of 6.8E-5 ug/m^3), assuming a 70 kg adult inhales 20 m^3 air/day. The unit risk is estimated from an occupational study of male textile workers exposed to acrylonitrile (O'Berg, 1980). The study observed 25 cases of cancer, including eight cases of respiratory cancer in 1345 workers exposed to acrylonitrile for 10 or more years at estimated concentrations of 5 to 20 ppm. The increased cancer incidence was observed with increased duration of exposure and length of follow-up time. The excess respiratory cancer was statistically significant both before and after evaluation for the contribution of smoking (5 observed vs. 1.6 expected).

Derivation of the Inhalation Reference Dose

The inhalation RfD is based on an RfC of 2.0E-03 mg/m^3 reported in IRIS (U.S. EPA, 1995a), which corresponds to an RfD of 5.71E-04 mg/kg-day. This assumes a 70 kg adult will inhale 20 m^3 air/day.

The RfC is based on two-year study by Quast et al. (1980). The study consisted of Sprague-Dawley rats (100/sex/concentration) exposed to acrylonitrile 6 hours/day, 5 days/week for 2 years to 0, 20, or 80 ppm acrylonitrile. A control group was exposed only to air. Additional rats were include in the study for interim sacrifices and evaluations at 6 and 12 months. Gross and histopathological evaluations of over 40 different organs were conducted, and the tissues that were observed to have adverse effects due to exposure were the respiratory tract and the nasal turbinates.

Significant degenerative and inflammatory changes were observed in the respiratory system, with the nasal cavity most severely affected. In the male rats exposed to 20 ppm acrylonitrile, there was a slight increase in respiratory epithelium hyperplasia in the nasal turbinates, and a statistically significant increase in hyperplasia of the mucous secreting cells. Females in the 200 ppm group had a slight increase in inflammation of the nasal turbinates, and a statistically significant increase in the flattening of the respiratory epithelium of the nasal turbinates. In the

80 ppm exposure group, the above effects were more severe, and were characterized by suppurative rhinitis, hyperplasia, focal erosions, and squamous metaplasia of the respiratory epithelium. No effects were observed in the olfactory epithelium, trachea, or lower respiratory system in males or females in any of the concentration groups. Based on these results, a LOAEL of 43 mg/m^3 (20 ppm) was estimated for effects to the epithelium of the extrathoracic region of the respiratory system.

The LOAEL of 43 mg/m^3 was adjusted for exposure time, resulting in a NOAEL(ADJ) of 7.7 mg/m^3. A human equivalency conversion (HEC) factor was then applied to arrive at a NOAEL(HEC) of 1.9 mg/m^3.

To derive the RfC, an uncertainty factor of 1000 was applied to the NOAEL(HEC): 10 to protect unusually sensitive human populations; 3 to adjust from a minimally adverse LOAEL to a NOAEL; 3 for the uncertainty in the interspecies variability because of the use of dosimetric adjustments and to account for the mild nature of the LOAEL; 10 to account for the lack of an inhalation bioassay in a second species and the lack of reproductive data by the inhalation route with the existence of an oral study that shows reproductive effects. The U.S. EPA has a medium level of confidence in the derivation of the RfC.

References

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U.S. EPA. 1995a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1995b. Health Effects Summary Tables (HEAST). Annual, F.Y. 1995. Office of Solid Waste and Emergency Response.

AMMONIA

There are no available carcinogenic dose-response values for ammonia in IRIS (U.S. EPA, 1995a) or the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b). IRIS contains an inhalation reference concentration (RfC) for ammonia.

Derivation of the Inhalation Reference Dose

The inhalation RfD is based on an RfC of 1.0E-01 mg/m^3 reported in IRIS (U.S. EPA, 1995a), which corresponds to an RfD of 2.86E-02 mg/kg-day. This assumes a 70 kg adult will inhale 20 m^3 air/day.

The RfC is based on an epidemiological study (Holness et al., 1989) of workers exposed to ammonia in a soda ash facility. Two groups of workers (52 exposed/31 control) were evaluated, with weight being the statistically significant difference determined in the two groups. The average age of the workers was 38.9 years, and the duration of exposure was 12.2 years. Exposure concentrations were based on personal sampling of the workers over one work shift (average 8.4 hours). The mean TWA concentration for the exposed was 9.2 ppm (6.4 mg/m^3) and 0.3 ppm (0.21 mg/m^3) for the controls.

A questionnaire was administered to obtain information on work histories, exposure histories, and eye, skin, and respiratory symptoms. Spirometry tests were performed at the beginning and end of each work shift on the first workday of the week and the last workday of the week. The difference between the two groups were evaluated for both the questionnaire and lung functions.

The results of the evaluation indicated no significant difference between the two groups with regard to subjective symptomatology or spirometry changes. Based on these results, a NOAEL of 9.2 ppm (6.4 mg/m^3) was established for the study, which was adjusted for an occupational exposure to a NOAEL(ADJ) of 2.3 mg/m^3.

To derive the RfC, an uncertainty factor of 30 was applied: 10 to account for sensitive individuals in the population; 3 to account for database deficiencies, including a lack of chronic studies and a lack of reproductive and developmental information. The U.S. EPA has a medium level of confidence in the RfC.

References

Holness, D.K., J.T. Purdham and J.R. Nethercott. 1989. Acute and chronic respiratory effects of occupational exposure to ammonia. Am. Ind. Hyg. Assoc. J. 50: 646-650.

U.S. EPA. 1995a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1995b. Health Effects Assessment Summary Tables (HEAST). Annual F.Y. 1995. Office of Solid Waste and Emergency Response, Washington, D.C.

BENZENE

The U.S. EPA has classified benzene as a human carcinogen (category A) due to evidence in humans of nonlymphocytic leukemia and evidence of other forms of cancer in rodents. In addition, supporting studies have shown benzene to cause chromosomal aberrations in bone marrow cells and lymphocytes, in both humans and animals. Reference doses are not provided by IRIS (U.S. EPA, 1995a) or Health Effects Assessment Summary Tables (U.S. EPA, 1995b). The ECAO has provided a provisional inhalation reference concentration (RfC) for benzene. The ECAO information is provided at the end of this Attachment.

Derivation of the Inhalation Cancer Slope Factor

The inhalation CSF for benzene is estimated to be 2.9E-02 $(mg/kg\text{-}day)^{-1}$ (U.S. EPA, 1994). The CSF value is based on three occupational inhalation studies: Rinsky et al. (1981), Ott et al. (1978), and Wong et al. (1983). Rinsky et al. observed 748 workers exposed to benzene for at least 24 years at 10-100 ppm 8-hour TWA. Seven leukemia deaths were recorded, which was a statistically significant incidence. Increases of leukemia and lymphatic and hematopoietic cancers were dose-dependent. The Ott et al. study observed 3 deaths from leukemia among 594 workers followed for 23 years, although the increase was not statistically significant. Wong et al. (1983) reported on the mortality of chemical workers exposed to benzene for at least a 6-month period. Dose-dependent increases were seen in leukemia and lymphatic and hematopoietic cancers. Confidence in the CSF is relatively high due to adequate time, number of cohorts, and significance of leukemia incidence. Recent investigations have suggested that benzene carcinogenicity arises from blood cell toxicity at high doses. Therefore, utilization of pharmacokinetic data would likely cause CSF estimates to be lower than the current EPA value.

REFERENCES

U.S. EPA. 1995a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1995b. Health Effects Assessment Summary Tables (HEAST). Annual, F.Y. 1995. Office of Solid Waste and Emergency Response, Washington, DC.

Ott, M.G., J.C. Townsend, W.A. Fishbeck and R.A. Langner. 1978. Mortality among individuals occupationally exposed to benzene. Arch. Environ. Health. 33: 3-10.

Rinsky, R.A., R.J. Young and A.B. Smith. 1981. Leukemia in benzene workers. Am. J. Ind. Med. 2:217-245.

Wong, O., R.W. Morgan and M.D. Whorton. 1983. Comments on the NIOSH study of leukemia in benzene workers. Technical report submitted to Gulf Canada, Ltd., by Environmental Health Associates.

1,3-BUTADIENE

IRIS (U.S. EPA, 1995a) classifies 1,3-butadiene as a Class B2, probable human carcinogen, and provides an inhalation unit risk. There is no available inhalation reference concentration (RfC) on IRIS or in the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b).

Derivation of the Inhalation Cancer Slope Factor

An inhalation cancer slope factor of 9.8E-01 $(mg/kg\text{-}day)^{-1}$ was derived from an inhalation unit risk of 2.8E-04 $\mu g/m^3$), assuming a 70 kg adult inhales 20 m^3 air/day. The U.S. EPA calculated the unit risk based on two lifetime rodent studies (NTP, 1984; Hazelton Laboratories, 1981).

In the NTP study (1984), 50 male and 50 female B6C3F1 mice were exposed to 625 or 1250 ppm of 1,3-butadiene for 6 hours/day, 5 days/week. Exposure began at 8-9 weeks of age, and all mice were sacrificed after 60-61 weeks due to excessive deaths among the treated mice. Increases in tumors were observed throughout the body, including hemangiosarcomas of the heart, lymphomas and alveolar/bronchiolar adenomas/carcinomas.

In the Hazelton Laboratory study (1981), 110 male and 110 female Charles River CD rats were exposed to 1000 or 8000 ppm of 1,3-butadiene for 6 hours/day, 5 days/week for 111 weeks (males) and 105 weeks (female). There was treatment related increase in mortality, some of which was attributed to nephropathies in males. Significant increases were observed in both common and uncommon tumors, including mammary gland tumors, thyroid follicular adenomas and carcinomas, and Leydig cell adenomas.

The tumor incidence in mice occurs at much lower exposure doses than for rats. Recent experimental data indicate that mice are much more sensitive to the effects of 1,3-butadiene than rats, and that humans are much more like rats in their metabolism of 1,3-butadiene (Himmelstein et al., 1994).

References

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U.S. EPA. 1995b. Health Effects Summary Tables (HEAST). Annual F.Y. 1995. Office of Solid Waste and Emergency Response, Washington, D.C.

CARBON TETRACHLORIDE

IRIS (U.S. EPA 1995a) provides oral RfD and oral CSF values. In addition, IRIS provides an inhalation unit risk value. An inhalation RfD is not available in IRIS or in the Health Effects Assessment Summary Tables (U.S. EPA 1995b). The inhalation CSF was chosen by extrapolation of the oral data, as only oral exposures have been used in animal bioassays. Carbon tetrachloride is classified as a B2, or probable human, carcinogen (U.S. EPA 1992).

Derivation of the Oral Cancer Slope Factor

The oral CSF is listed as 1.3E-01 $(mg/kg\text{-}day)^{-1}$, based on an NCI study from 1976 and 1977 and several other experiments (U.S. EPA, 1995a). Data on human carcinogenicity is inadequate: 3 liver cancer cases have been reported and the available epidemiological results suggest carcinogenicity, but do not provide definitive evidence. However, animal carcinogenicity by the oral route has been sufficiently demonstrated in every rodent species tested with carbon tetrachloride. In the principal study, 50 male and 50 female rats were dosed by corn oil gavage for 5 days/wk for 78 weeks, at 47 and 94 mg/kg for males, or 80 and 159 mg/kg for females. After 110 weeks, 7/50 males and 14/50 females had survived the low dose. In the same study, 100% of mice dosed at 250 mg/kg expressed liver tumors compared to 97% tumors in mice dosed at 2500 mg/kg (NCI, 1976a,b, 1977). Liver tumors were significant even in L mice, a strain with a naturally low incidence of liver cancer (Edwards et al., 1942). Throughout these experiments, low doses demonstrated higher cancer levels than high doses, due to greater fatality at high doses before carcinogenic development could occur.

The supporting data show that carbon tetrachloride is not mutagenic; in fact, it is difficult to establish the mechanism of carcinogenicity. Confidence in the CSF is low, as the studies were considered weak.

Derivation of the Inhalation Cancer Slope Factor

IRIS (U.S. EPA, 1995a) provides a unit risk of 1.5E-05 $(\mu g/m^3)^{-1}$. Based on oral exposure data, this value adjusts for a 40% absorption rate by humans. Assuming an inhalation rate of 20 m^3/day and a body weight of 70 kg, the unit risk is converted to a cancer slope factor of 5.25E-02 $(mg/kg\text{-}day)^{-1}$. This slope factor should not be used if the air concentration exceeds 70 $(\mu g/m^3)$, as it may not be appropriate above this level.

For a discussion of confidence in the inhalation cancer slope factor, see the Derivation of the Oral Cancer Slope Factor section.

Derivation of the Inhalation Reference Dose

An inhalation reference dose of 5.71E-04 mg/kg-day was provided by ECAO. The ECAO information is provided at the end of this attachment.

REFERENCES

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CHLOROBENZENE

An inhalation reference concentration (RfC) for chlorobenzene is available in the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995a). The U.S. EPA has classified chlorobenzene as a Group D carcinogen (not classifiable as to human carcinogenicity) based on a lack of human data, inadequate animal data, and negative genetic toxicity data (U.S. EPA, 1995b).

Derivation of the Inhalation Reference Dose

HEAST (U.S. EPA, 1995) report an inhalation reference concentration for chlorobenzene of 2E-2 mg/m^3 and an RfD of 5.7E-3 mg/kg-day. The reported value was based on a 120-day intermittent inhalation study of rats. A LOAEL of 75 ppm is chosen as the basis for the chronic inhalation RfD with an uncertainty factor of 10,000.

REFERENCES

U.S. EPA. 1995a. Health Effects Assessment Summary Tables (HEAST). Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1995b. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

CHLOROFORM

Chloroform is classified by the U.S. EPA as a Group B2 carcinogen (probable human carcinogen). Cancer slope factors are listed in IRIS (U.S. EPA, 1995) for both the oral and inhalation routes of exposure. The toxicity of chloroform has also been assessed for noncarcinogenic endpoints and IRIS (U.S. EPA, 1995) lists an oral RfD, however, no inhalation RfD is available.

Derivation of the Inhalation Cancer Slope Factor

The inhalation slope factor of 8.1E-02 $(mg/kg\text{-}day)^{-1}$ is based on oral gavage data from a study by NCI (1976). Vehicle control animals were run concurrently and housed with test animals. All treated animals experienced decreased body weight gain. Survival was reduced in high-dose males and in all treated females.

Experimental data for this compound support complete absorption of orally administered chloroform under conditions of this assay. There are no apparent species differences in this regard. Extrapolation of metabolism-dependent carcinogenic responses from mice to humans on the basis of body surface area is supported by experimental data. The slope factor is the geometric mean calculated from male (0.033) and female (0.2) data.

Slope factors derived from male rat kidney tumor data of 0.024 $(mg/kg\text{-}day)^{-1}$ (NCI, 1976) and studies by Roe et al. (1979) of 0.1 $(mg/kg\text{-}day)^{-1}$ are generally supportive of the risk estimate.

Review of Chloroform Inhalation CSF Issues

Chloroform is classified by the US EPA as a Group B2 carcinogen (US EPA, 1995a): a probable human carcinogen based on what EPA considers to be sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans. The underlying assumption of regulatory risk characterization for compounds with known or assumed potential carcinogenic effects is that no threshold dose exists. Thus, the characterization of carcinogens assumes that there is some finite level of risk associated with each non-zero dose, i.e., even with exposure to a single molecule. This model was developed based on the scientific community's understanding of direct-acting carcinogens, of which cancer chemotherapeutic drugs were the prime example, since both animal and human data were available. Although the model may be appropriate for direct-acting genotoxic agents (i.e., those chemicals that bind directly to DNA to exert their effects), it may still be overly conservative even for these agents, since the model does not take

into account DNA repair mechanisms that can protect the DNA, especially from low levels of chemical exposure.

There is an increasing body of scientific evidence suggesting that a second mechanism of carcinogenesis exists, that of nongenotoxic cytotoxic carcinogens. These are agents that do not bind to DNA but whose carcinogenicity is presumed to be a secondary event resulting from cytotoxicity (the induction of cell death) and excessive cell proliferation in response to the cell death. It is during this proliferative response that "precancerous" cells may proliferate and develop into tumors. Research in this area, specifically with chloroform, has indicated that for nongenotoxic cytotoxic agents, a threshold for cytotoxicity exists, and therefore, a threshold for carcinogenicity exists. Therefore, it would be more appropriate to regulate these chemicals based on a threshold mechanism of action, much like the regulatory treatment of noncarcinogens.

Chloroform has been studied as a model compound of this threshold mechanism of carcinogenesis. The US EPA has incorporated some of this information into its regulation of chloroform, but not completely, and not consistently.

The first cancer bioassay for chloroform was conducted by the National Cancer Institute (NCI) (NCI, 1976). It was an oral gavage study in which rats and mice were administered chloroform dissolved in corn oil in a single large dose once per day via on oral tube or syringe. An increased frequency of kidney tumors was noted in male rats, and a significant increase in liver tumors was noted for both sexes of mice. Based on this study, the US EPA developed an oral CSF of 8.1×10^{-2} $(mg/kg\text{-}day)^{-1}$ for chloroform, based on the mouse data. This value was adopted as the CSF for inhalation exposures as well.

In 1985, Jorgenson et al. administered chloroform in drinking water to rats and mice in a two year cancer bioassay. A significant increase in kidney tumors was observed only in the highest dose group in male rats. Liver tumor incidence was not significantly increased in mice, despite the fact that they received the same total daily dose as the mice in the NCI gavage study. The only difference was that the mice in the NCI study received a single large dose of chloroform once a day, and the mice in the Jorgenson study received small but frequent doses of chloroform as they drank their water throughout the day. This latter exposure regimen more closely mimics potential human environmental exposures to chloroform.

The US EPA revised its estimate of the oral CSF for chloroform based on the Jorgenson study to the current value of 6.1×10^{-3} $(mg/kg\text{-}day)^{-1}$. By lowering the CSF value, EPA was recognizing that chloroform exposures were not as toxic as first believed based on the NCI gavage data. However, a concomitant change was not made in the inhalation CSF, which is still based on the

NCI gavage study, even though potential human environmental exposures to chloroform in air would be expected to be continuous, rather than a single large exposure each day. The inhalation CSF for chloroform should have been changed to 6.1×10^{-3} $(mg/kg\text{-}day)^{-1}$, when the oral CSF was changed. Using this value would result in 13.3-fold lower inhalation risks in a risk assessment compared to the current inhalation CSF.

As stated above, these CSF for chloroform are calculated using an EPA model appropriate for direct-acting carcinogens. More recent work on the mechanism of action of chloroform has been conducted by the Chemical Industry Institute of Toxicology (CIIT), and summarized two articles:

- Risk Assessment Issues Associated with Chloroform-Induced Mouse Liver Tumors. BE Butterworth, JL Larson, RB Conolly, SJ Borghoff, GL Kedderis, and DC Wolf. CIIT Activities, Vol. 14, No. 2, pp 1-7.

- Risk Assessments for Chloroform. BE Butterworth, RB Conolly, MV Templin, and DC Wolf. CIIT Activities, Vol. 14, No. 3, pp 8-11.

CIIT researchers note that when chloroform is tested for genotoxicity, the results are almost uniformly negative, indicating that chloroform is not a direct acting carcinogen. The CIIT researchers have been studying the effects of chloroform on cell toxicity, cell death and regenerative cell proliferation following gavage, drinking water and inhalation exposures. Female mice administered chloroform by gavage exhibit substantial necrosis, inflammation and regenerative cell proliferation in the liver after just 4 days and continuing up through three weeks of exposure. On the other hand, female mice exposed to chloroform in drinking water at concentrations resulting in a daily dose similar to that of the gavage group did not exhibit any cellular changes, even after three weeks of exposure.

These observations have been extended to inhalation exposures in as yet unpublished studies by CIIT presented at the December, 1994 meeting of the Society for Risk Analysis and the March, 1994 meeting of the Society of Toxicology. The CIIT work has been complemented by a two year inhalation cancer bioassay performed by the Japan Bioassay Laboratory, work that also has not yet been published, but was presented by the CIIT researchers at the aforementioned meetings. In the Japanese study, mice were exposed to chloroform in air at 0, 5, 30, and 90 ppm. Significant increases in liver tumors only occurred in the 90 ppm dose group. CIIT has studied cellular effects of inhalation exposures in mice to the same air concentrations of chloroform, and detected significant changes only in the 30 and 90 ppm dose groups. These data also support the hypothesis that induction of cytotoxic effects are necessary precursors to chloroform induced tumor formation.

Mechanistically, how are these phenomenon explained? Both animals and humans can metabolize and detoxify low doses of chloroform into non-toxic products that are safely excreted from the body. At high doses, the enzymes responsible for detoxifying chloroform can be overwhelmed and toxic intermediates may accumulate in the cells. These intermediates can induce cell injury and cell death. The single gavage doses in the NCI assay were large enough to overwhelm the animals' ability to safely metabolize the chloroform, cell injury and cell death resulted, followed by regenerative cell proliferation and tumor induction. In the Jorgenson drinking water study, the small doses of chloroform received throughout the day were easily metabolized and excreted by the animals. It was only at the highest concentrations in the drinking water study that tumorigenic effects were found. The same is true for the inhalation bioassay results. When exposures to air concentrations of chloroform were below 30 ppm, chloroform was easily metabolized and excreted; above 30 ppm, cell injury and cell death occurred because the metabolic capability was exceeded. Note that although low levels of cellular changes were observed in mouse livers at 30 ppm in the CIIT study, tumors were only observed at the 90 ppm exposure group in the Japanese study.

The CIIT researchers have applied these data to a risk assessment for chloroform. They state:

> The weight of evidence indicates that chloroform is acting through a non-genotoxic/cytotoxic mode of action. In this case, tumor formation is thought to result from initiation and promotional events which are secondary to induced cytolethality and regenerative cell proliferation. Thus, no increased risk of tumors would be anticipated at doses that do not induce cytolethality, designated as a no observed adverse effect level (NOAEL).

Applying the NOAEL approach to chloroform, the CIIT researchers suggest that a virtually safe dose (VSD), similar to a reference dose (RfD) for noncarcinogens, would be 0.01 ppm (49.6 $\mu g/m^3$) in air by defining the NOAEL of the CIIT results at 10 ppm, and applying an uncertainty factor of 1000. The VSD based on the Japanese bioassay would be 0.03 ppm (148.8 $\mu g/m^3$) in air by defining the NOAEL at 30 ppm, and applying an uncertainty factor of 1000. The current US EPA derived VSD at a 1×10^{-5} risk level is 0.00008 ppm (0.397 $\mu g/m^3$).

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1,4-DICHLOROBENZENE

The U.S. EPA has classified 1,4-dichlorobenzene as a group B2 carcinogen (possible human carcinogen based on limited evidence of carcinogenicity in animals and inadequate or lack of human data). IRIS (U.S. EPA, 1995) provides an inhalation reference concentration for this compound, both of which are currently under review. An inhalation cancer slope factor (CSF) was obtained from California Environmental Protection Agency (CalEPA, 1994) information.

Derivation of the Inhalation Cancer Slope Factor

The inhalation CSF of 4.0E-02 $(mg/kg\text{-}day)^{-1}$ is based on a 103 week study of male and female mice and rats (CalEPA, 1994). Hepatocellular adenoma and carcinoma of the adrenal gland were observed in both male and female mice. Pheochromocytoma of the renal tubular gland was observed in male mice only, and adenocarcinoma was observed in male rats.

Derivation of the Inhalation Reference Concentration

The RfC for 1,4-dichlorobenzene is 8.0E-01 mg/m^3 (IRIS, 1995). This corresponds to an inhalation RfD of 2.3E-01 mg/kg-day. The value is based on a two-generation reproductive study of Sprague-Dawley rats exposed to 1,4-dichlorobenzene vapor at concentrations of 0, 50, 150, or 450 ppm for 6 hours/day, 7days/week for ten weeks (Chlorobenzene Producers Association, 1986). The rats were then mated for three weeks, and selected weanlings were exposed for 11 weeks and then mated. The primary effects were reduced body weights and weight gain, reduced food consumption, increased incidence of tremors, and nasal and ocular discharges in adult males exposed to 150 ppm. A statistically significant increase in liver weights in adult male rats of the 150 and 450 ppm groups. Increased kidney weight was also observed in both the male and female parental rats.

Based on the study, a NOAEL of 301 mg/m^3 (50 ppm) was established, and an adjusted NOAEL of 75 mg/m^3 was derived. To calculate the RfC, an uncertainty factor of 100 was applied: 10 to account for sensitive human populations; a factor of 3 to account for interspecies differences since dosimetry adjustments were used; and an additional factor of 3 since the NOAEL was based on a subchronic study rather than a chronic study. The U.S. EPA confidence is medium for the RfC results.

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DICHLOROMETHANE (METHYLENE CHLORIDE)

IRIS (U.S. EPA, 1995a) classifies dichloromethane as a Class B2, probable human carcinogen, and provides an inhalation unit risk. IRIS does not contain an inhalation reference dose (RfD), however, the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b) provide an inhalation reference concentration (RfC) for dichloromethane.

Derivation of the Inhalation Cancer Slope Factor

An inhalation cancer slope factor of 1.6E-03 $(mg/kg\text{-}day)^{-1}$ was derived from an inhalation unit risk of 4.7E-07 per (ug/m^3), assuming a 70 kg adult inhales 20 m^3 air/day. The U.S. EPA calculated the unit risk based on a study of 50 male and 50 female groups of F344/N rats and B6C3F1 mice exposed to dichloromethane by inhalation for 6 hours/day, 5 days/week, for 2 years (NTP, 1986). The exposure concentrations were 0, 1000, 2000, and 4000 ppm for rats and 0, 2000, or 4000 ppm for mice. Survival was decreased for male and female mice and female rats. Mammary adenomas and fibroadenomas were significantly increased in male and female rats, as were mononuclear cell leukemias in female rats. Mice of both sexes showed significant increases in hepatocellular adenomas and carcinomas, and alveolarbronchiolar adenomas and carcinomas. In addition, there was a significant increase in the number of lung tumors in both sexes of mice.

Derivation of the Inhalation Reference Dose

The inhalation RfD is based on an RfC of 3.0E-00 mg/m^3 reported in HEAST (U.S. EPA, 1995b), which corresponds to an RfD of 8.6E-01 mg/kg-day. This assumes a 70 kg adult will inhale 20 m^3 air/day.

The RfC is based on a two year intermittent inhalation study of rats exposed to dichloromethane (Nitschke et al., 1988). The study results showed toxic effects to the liver. Based on the results of the study, a NOAEL of 694.8 mg/m^3 was calculated. An uncertainty factor of 100 was applied to develop the RfC.

References

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ETHYLENE DIBROMIDE (EDB)

The U.S. EPA (IRIS, 1995a) classifies ethylene dibromide (EDB) as Class B2, a probable human carcinogen, and provides an inhalation unit risk. The Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b) provide an inhalation reference concentration (RfC).

Derivation of the Inhalation Cancer Slope Factor

An inhalation cancer slope factor of 7.7E-01 $(mg/kg\text{-}day)^{-1}$ was derived for EDB from an inhalation unit risk of 2.2E-4 ug/m^3, assuming a 70 kg adult inhales 20 m^3 air/day. The unit risk is estimated from a rodent study conducted by NTP (1982). Fischer 344 rats and B6C3F1 mice of both sexes were exposed to EDB vapors of 0, 10, or 40 ppm 6 hours/day, 5 days/week for their lifespan. The incidence of nasal cavity carcinomas and adenocarcinomas in rats of both sexes and alveolar/bronchiolar carcinomas in female rats and mice of both sexes were significantly increased over the control group.

Derivation of the Inhalation Reference Dose

An inhalation RfC of 2.0E-04 mg/m^3 reported in HEAST (U.S. EPA, 1995b), which corresponds to an RfD of 5.71E-05 mg/kg-day. This assumes a 70 kg adult will inhale 20 m^3 air/day.

The RfC is derived from occupational studies in which sperm effects were observed in workers intermittently exposed to EDB (Ratcliffe et al., 1987; Schrader et al., 1988) through inhalation. Based on these results, a LOAEL of 88 ppb was reported in HEAST (1995). An uncertainty factor of 1000 was applied to the LOAEL.

References

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U.S. EPA. 1995a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

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ETHYLENE OXIDE

There are no available carcinogenic or noncarcinogenic dose-response values for ethylene oxide in IRIS (U.S. EPA, 1995a). Ethylene oxide is classified by the U.S. EPA (1995a) as a B1, probable human carcinogen. The Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b) provide an inhalation cancer slope factor (CSF), however, no inhalation reference concentration (RfC) for noncancer effects was reported.

Derivation of the Inhalation Cancer Slope Factor

The inhalation CSF for ethylene oxide is 3.5E-01 $(mg/kg\text{-}day)^{-1}$. The CSF is based on a two-year intermittent inhalation study of rats (Snellings et al., 1981). The study indicated increases in the incidence of leukemia and brain cancer in the study animals exposed to ethylene oxide.

References

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FORMALDEHYDE

U.S. EPA (1995a) has classified formaldehyde as a B1, probable human carcinogen, based on limited evidence in humans and sufficient evidence in animals. In addition, supporting studies have demonstrated mutagenic and genotoxic effects of formaldehyde in vitro in mammalian cells as well as other systems. Both IRIS (U.S. EPA, 1995a) and the Health Effects Assessment Summary Tables (U.S. EPA, 1995b) report inhalation cancer slope factors for the potential carcinogenic effects of formaldehyde by the inhalation route of exposure.

Derivation of the Inhalation Cancer Slope Factor

The inhalation CSF for formaldehyde is 4.5×10^{-2} $(mg/kg/day)^{-1}$, and is based on an inhalation study in rats performed by Kerns et al. (1983). F344 rats were exposed to 0, 2, 5, 6 and 14.3 ppm formaldehyde 6 hours/day, 5 days/week for 24 months followed by up to 6 months of non exposure. Body weight was not significantly affected by exposure. Mortality in the high dose group was significantly elevated with respect to controls. Squamous cell carcinomas were observed in the nasal cavities of animals in the high and low dose groups (tumor incidence: 94/140 and 2/153, respectively) and occurred in a non-linear dose-response manner. The linearized multistage model was used to calculate the unit risk. There is limited epidemiological evidence that correlates human exposure with lung and nasopharyngeal cancer deaths.

REFERENCES

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N-HEXANE

There are no available carcinogenic dose-response values for n-hexane in IRIS (U.S. EPA, 1995a) or the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1995b). IRIS contains an inhalation reference concentration (RfC) for n-hexane.

Derivation of the Inhalation Reference Dose

An RfC of 2.0E-01 mg/m^3 is reported in IRIS (U.S. EPA, 1995a), which corresponds to an RfD of 5.71E-02 mg/kg-day, is assuming a 70 kg adult will inhale 20 m^3 air/day.

The RfC is based on an epidemiological study (Sanagi et al., 1980) of workers exposed in a factory producing tungsten carbide alloys. Two groups of workers (14 exposed/14 control) were matched based on age, stature, weight, alcohol consumption and smoking habits. The exposure durations ranged from 1 to 12 years, with an average of 6.2 years. Both groups underwent clinical neurological examinations with regard to cranial nerves, motor and sensory systems, reflexes, coordination, and gait. Neurophysiological studies included electromyography on muscles of the forearm and leg and nerve stimulation.

No neurological abnormalities were noted in the clinical examinations. The neurophysical tests showed a decrease in the mean motor nerve velocity of the exposed group when compared with the controls. In addition, the residual latency of motor nerve conduction of the posterior tibial nerve in the exposed group was significantly slower in the exposed group. Based on the results of the study, a LOAEL of 204 mg/m^3 was estimated. This LOAEL was adjusted for occupational exposure for a LOAEL (ADJ) of 73 mg/m^3.

To derive the RfC, and uncertainty factor of 300 was applied: 10 to account for sensitive populations; 10 for the use of a LOAEL rather than a NOAEL; 3 for lack of data on reproductive and chronic respiratory effects. The U.S. EPA has medium confidence in the RfC for n-hexane.

References

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U.S. EPA. 1995b. Health Effects Assessment Summary Tables (HEAST). Annual, F.Y. 1995. Office of Solid Waste and Emergency Response. Washington, D.C.

TETRACHLOROETHENE (TETRACHLOROETHYLENE)

The U.S. EPA has withdrawn the carcinogenicity assessment for tetrachloroethylene (U.S. EPA, 1995). IRIS does not report an inhalation oral RfC for noncarcinogenic health effects (U.S. EPA, 1995). The ECAO (1993) reports a provisional cancer slope factor and a provisional RfC (ECAO, 1995) for tetrachloroethene. The ECAO reported tetrachloroethene as a B2 carcinogen, (possible human carcinogen based on limited evidence of carcinogenicity in animals and inadequate or lack of human data). The provisional CSF for tetrachloroethene is 2.0E-03 $(mg/kg\text{-}day)^{-1}$. The provisional RfC is reported as 3.99E-01 mg/m^3, which, based on a 70 kg adult inhaling 20 m^3 air/day, corresponds to an inhalation RfD of 1.14E-01 mg/kg-day. The ECAO information for the RfC is provided at the end of this Attachment.

REFERENCES

U.S. EPA. 1995. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

TOLUENE

IRIS (U.S. EPA, 1995) provides a reference air concentration for inhalation exposure. The U.S. EPA has assigned toluene a human carcinogenicity classification of D, or not classifiable. This is due to a lack of positive animal bioassays (NTP, 1989) or human studies.

Derivation of the Inhalation Reference Dose

The inhalation RfC for toluene is 0.4 mg/m^3 (U.S. EPA, 1995). This is equivalent to 0.114 mg/kg-day, assuming a 70 kg person breathes 20 m^3 of air a day. The value is based on a study that examined neurobehavioral effects resulting from human occupational exposure to toluene (Foo et al., 1990). Although co-exposure to others solvents in the work place was not addressed, and poor correlation-response relationships resulted for the battery of neurotoxicity tests, a LOAEL of 88 ppm was identified from this study. The average number of years worked by the study population was 5.7 +/- 3.2 yrs.

Because there is a large body of information that relates exposure to toluene with CNS effects, this study was used to develop an RfC, despite its draw backs, since it is one of the few chronic studies performed in humans. An uncertainty factor of 300 is applied: 10 to account for intraspecies variability, 10 to account for use of a LOAEL, and 3 to account for data base deficiencies. The U.S. EPA has a medium level of confidence in the RfC.

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NTP (National Toxicology Program). 1989. Toxicology and carcinogenesis studies of toluene (CAS No. 108-88-3) in F344/N rats and B5C3F1 mice (inhalation studies). Technical Report Series No. 371. Research Triangle Park, NC.

TRICHLOROETHENE (TRICHLOROETHYLENE)

The carcinogenicity assessment for trichloroethylene has been withdrawn from IRIS (U.S. EPA, 1995a). Trichloroethylene was formerly classified by the U.S. EPA as a Class B2 carcinogen (a probable human carcinogen), and cancer slope factors were provided for both oral and inhalation exposures. No RfDs were available in IRIS or the Health Effects Assessment Summary Tables (U.S. EPA, 1995b). The ECAO (1993) provided a provisional inhalation cancer slope factor for trichloroethene.

REFERENCES

U.S. EPA. 1995a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1995b. Health Effects Assessment Summary Tables (HEAST). Annual, F.Y. 1995. Office of Solid Waste and Emergency Response, Washington, DC.

TRIMETHYL BENZENE

An inhalation reference dose for trimethyl benzene of 1.71E-03 mg/kg-day was provided by ECAO. The ECAO information is provided at the end of this attachment.

VINYL CHLORIDE

U.S. EPA has classified vinyl chloride as a human carcinogen, group A, due to evidence of liver, brain, and lung cancers arising from occupational exposures. In addition, vinyl chloride is known to cause potentially fatal circulatory and nerve damage. IRIS (U.S. EPA, 1995a) does not provide any information about vinyl chloride, but the Health Effects Assessments Summary Tables report an inhalation cancer slope factor, with IRIS input pending (U.S. EPA, 1995b).

Derivation of the Inhalation Cancer Slope Factor

The inhalation unit risk of 8.5×10^{-5} $(\mu g/m^3)^{-1}$, which corresponds to an inhalation cancer slope factor of 0.3 $(mg/kg/day)^{-1}$, is based upon a study by Maltoni et al. (1980, 1981), in which rats were dosed with 1 to 30,000 ppm vinyl chloride for 1 year, and mice and hamsters were dosed with 50 to 30,000 ppm for 30 weeks, with one to two years observation time. All rodent species developed liver cancers at statistically significant levels at or above 50 ppm (Maltoni et al., 1980, 1981). Unit risk calculations assume 50% absorption.

REFERENCES

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POLYNUCLEAR AROMATIC HYDROCARBONS

POLYNUCLEAR AROMATIC HYDROCARBONS (PAH)

Polynuclear aromatic hydrocarbons (PAH) are a class of related chemicals that are components of petroleum products, coal, and coal tar. They are also formed from the incomplete combustion of fuels. In hazardous waste site investigations, site media are typically analyzed for eighteen PAH. For this risk assessment, the dose-response values used are based on an inhalation cancer slope factor (CSF) for benzo(a)pyrene [B(a)P] derived by the California Environmental protection Agency (CalEPA, 1994) and U.S. EPA's comparative potency approach as described below. ENSR has used information provided by the Environmental Criteria and Assessment Office (ECAO, 1995) on noncancer inhalation dose-response values for PAH.

Carcinogenic Risk Assessment

For carcinogenic risk assessment, ENSR has used the U.S. EPA developed comparative potency approach to calculate benzo(a)pyrene toxic equivalents (U.S. EPA, 1993). Of the 18 commonly detected PAH, U.S. EPA has classified seven of them as "probable human carcinogens (class B2)." They include: benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(1,2,3-c,d)pyrene, dibenz(a,h)anthracene, and chrysene.

Benzo(a)pyrene is one of the most potent of the potentially carcinogenic PAH in animal experiments. A review of the scientific literature indicates that the other PAH are considerably less potent than B(a)P. To perform carcinogenic risk assessment for the other six potentially carcinogenic PAH, one needs an estimate of their carcinogenic potency.

A comparative potency approach was developed for U.S. EPA in which experimental studies on B(a)P and other PAH are used to derive comparative potency factors that relate the potency of other PAH to B(a)P. Then the CalEPA inhalation cancer slope factor for B(a)P can be used to estimate the potential cancer risk posed by exposure to a mixture of potentially carcinogenic PAH. The U.S. EPA derived B(a)P Toxic Equivalent Factors are listed in the table below.

Benzo(a)Pyrene Toxic Equivalent Factors

Compound	Toxic Equivalent Factors
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenz(ah)anthracene	1.0
Indeno(1,2,3-cd)pyrene	0.1
Source: U.S. EPA, 1993	

Derivation of the Inhalation Cancer Slope Factor for Benzo(a)pyrene

The inhalation cancer slope factor is based on an inhalation unit risk derived by the CalEPA of 1.1E-03 $(\mu g/m^3)^{-1}$. This unit risk corresponds to a cancer slope factor of 3.9E+00 $(mg/kg\text{-}day)^{-1}$, assuming a 70 kg adult inhales 20 m^3 air/day.

The CalEPA unit risk was based on a study of hamster exposed to B(a)P through inhalation (Thyssen et al., 1981). In the study, Syrian golden hamsters were exposed B(a)P concentrations of 2.2, 9.5, or 46.5 mg/m^3 condensed onto sodium chloride particles. Control animals were unexposed. Survival time was decreased for the highest exposed animals from 96 weeks to 60 weeks; survival time did not change for those in the lower exposure groups. No tumors were observed in the control group or in the group exposed to the lowest concentration of 2.2 mg/m^3 B(a)P. The incidence of tumors increased in a dose-dependant manner in the respiratory system for the remaining two groups of animals. Tumors were observed in the nasal cavity, larynx, trachea, pharynx, esophagus, and forestomach. No lung tumors were observed in any of the animals. The tumor types were classified as papillomas, papillary polyps, and squamous cell carcinomas.

Noncarcinogenic Risk Assessment

All PAH compounds have the potential for noncarcinogenic health effects. However, the U.S. EPA has derived oral reference doses for only six of the PAH: naphthalene, pyrene, acenaphthene, fluorene, anthracene, and fluoranthene and an inhalation RfC only for naphthalene. Based on information provided by the Environmental Criteria and Assessment Office (ECAO, 1995), the dose-response value developed for naphthalene will be used to assess the noncarcinogenic effects of all PAH compounds in this assessment. The ECAO information is provided as an attachment at the end of this Attachment

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PESTICIDES

CHLOROPYRIFOS

There are no available carcinogenic dose-response values on IRIS (U.S. EPA, 1995a) or in the Health Effects Assessment Summary Tables (HEAST, 1995b) for chloropyrifos. Chloropyrifos has not been evaluated for noncarcinogenic effects through the inhalation route. In this assessment, the chronic oral reference dose (RfD) reported in IRIS for chloropyrifos is used to evaluate inhalation exposures.

Derivation of the Oral Reference Dose

The RfD for chloropyrifos is 3.0E-03 mg/kg-day (IRIS, 1995a). It is based on a study performed by the Dow Chemical Company (Dow Chemical, 1972). Sixteen human male volunteers were administered chloropyrifos by capsule at doses of 0, 0.014, 0.03, or 0.10 mg/kg/day. The low and mid doses were administered for 20 days, and the high dose was administered for 9 days. Compared to a control group, the mean plasma cholinesterase was inhibited by approximately 65%. An uncertainty factor of 10 was applied to account for the range of human sensitivity for cholinesterase inhibition. The U.S. EPA has a medium level of confidence in this RfD.

References

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DISULFOTON

There are no available carcinogenic dose-response values on IRIS (U.S. EPA, 1995a) or in the Health Effects Assessment Summary Tables (HEAST, 1995b) for disulfoton. Disulfoton has not been evaluated for noncarcinogenic effects through the inhalation route. In this assessment, the chronic oral reference dose (RfD) reported in IRIS for disulfoton is used to evaluate inhalation exposures.

Derivation of the Oral Reference Dose

The RfD for disulfoton is 4.0E-05 mg/kg-day (IRIS, 1995a). It is based on a study performed by the Mobay Chemical Company (Mobay Chemical, 1985). Disulfoton was fed to male and female Fischer 344 rats at concentrations of 0.8, 3.3, or 13 ppm in the diet for 105 weeks. Dose related inhibition of cholinesterase was observed for plasma, erythrocyte, and brain in both sexes of all the treated groups. Optic nerve degeneration was also observed in the low, mid, and high dose females, but was only statistically significant in the mid and high dose groups. An uncertainty factor of 1000 was applied: 100 to account for interspecies differences and the range of human sensitivity for cholinesterase inhibition, plus a factor of 10 to account for the lack of a NOAEL. The U.S. EPA has a medium level of confidence in this RfD.

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METHYL PARATHION

There are no available carcinogenic dose-response values on IRIS (U.S. EPA, 1995a) or in the Health Effects Assessment Summary Tables (HEAST, 1995b) for methyl parathion. Methyl parathion has not been evaluated for noncarcinogenic effects through the inhalation route. In this assessment, the chronic oral reference dose (RfD) reported in IRIS for methyl parathion is used to evaluate inhalation exposures.

Derivation of the Oral Reference Dose

The RfD for methyl parathion is 2.5E-04 mg/kg-day (IRIS, 1995a). It is based on a study performed by the Monsanto Company (Monsanto, 1984). Methyl parathion was fed to sixty male and female rats at concentrations of 0.5, 5, or 50 ppm in the diet for 2 years. Based on effects in hematological parameters, a NOEL of 0.5 ppm was established. The effects observed included slightly reduced hemoglobin, hematocrit, and RBCs in the mid and high dose males, and moderately reduced in the high dose females. An uncertainty factor of 100 was applied to account for inter- and intraspecies differences in the extrapolation to humans. The U.S. EPA has a medium level of confidence in this RfD.

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PENTACHLORONITROBENZENE

There are no available carcinogenic dose-response values on IRIS (U.S. EPA, 1995a) for pentachloronitrobenzene. An oral cancer slope factor (CSF) is provided in the Health Effects Assessment Summary Tables (HEAST, 1995b), and is used in the carcinogenic assessment of inhalation exposure to pentachloronitrobenzene. Pentachloronitrobenzene has not been evaluated for noncarcinogenic effects through the inhalation route. Therefore, the chronic oral reference dose (RfD) reported in IRIS is used to evaluate inhalation exposures in this assessment.

Derivation of the Oral Reference Dose

The RfD for pentachloronitrobenzene is 3.0E-03 mg/kg-day (IRIS, 1995a). It is based on a study performed by the Olin Mathieson Chemical Corporation (Olin Mathieson, 1968). Pentachloronitrobenzene was fed to dogs (four males and four females/group) at concentrations of 0, 30, 180, or 1080 ppm in the diet for 2 years. Liver weight increases, increased liver-to-body weight ratios, elevated serum alkaline phosphatase levels, and microscopically observed cholestatic hepatosis were observed in the highest dose groups. Based on the observed effects, a NOEL of 30 ppm was established. An uncertainty factor of 300 was applied: 100 to account for inter- and intraspecies differences, and 3 since the data base for chronic toxicity is incomplete. The U.S. EPA has a medium level of confidence in this RfD.

Derivation of the Oral Cancer Slope Factor

The U.S. EPA has classified pentachloronitrobenzene as a Class C, possible human carcinogen. The oral CSF of 2.6E-01 $(mg/kg\text{-}day)^{-1}$ reported in HEAST (1995b) was derived from a 72 week oral study of mice (Innes et al., 1969). The study results indicated an increase in liver tumors.

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Olin Mathieson Chemical Corporation. 1968. MRID No. 00001666, 00083328, 00114201; HED Doc. No. 005188. Available from the EPA.

U.S. EPA. 1995a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1995b. Health Effects Assessment Summary Tables (HEAST). Annual F.Y.I. 1995. Office of Solid Waste and Emergency Response. Washington, D.C.

TRIFLURALIN

There is no available dose-response information available for carcinogenic or noncarcinogenic effects of trifluralin through the inhalation route. An oral cancer slope factor (CSF) and an oral reference dose (RfD) are provided in IRIS (1995), and are used in the assessment of inhalation exposures to trifluralin.

Derivation of the Oral Reference Dose

The RfD for trifluralin is 7.5E-03 mg/kg-day (IRIS, 1995). It is based on a study performed by the Hoechst Aktiengesellschaft (Hoechst, 1984). Trifluralin was fed to six male and six female beagle dogs at concentrations of 0, 30, 150, or 750 ppm in the diet for 1 year. At 750 ppm, there was decreased weight gain in both male and female dogs. An increase in methemoglobin in mid and high dose males and females was also observed. There was an increase in liver weight in males receiving 150 and 750 ppm and in females receiving 750 ppm. Based on the increased liver weights and the increased methemoglobin, an NOEL of 30 ppm was estimated. An uncertainty factor of 100 was applied to account for inter- and intraspecies differences. The U.S. EPA has a high level of confidence in this RfD.

Derivation of the Oral Cancer Slope Factor

The U.S. EPA has classified trifluralin as a Class C, possible human carcinogen. The oral CSF of 7.7E-03 $(mg/kg\text{-}day)^{-1}$ reported in IRIS (1995) was derived from a study of 120 F344 rats (60 male/60 female) fed dietary doses of 0, 813, 3250, or 6500 ppm for 2 years (Emmerson et al., 1980). The study results indicated a statistically significant increase in the incidence of bladder papillomas and renal pelvis carcinomas in the highest dose level in male and female rats, respectively. An increase in follicular cell tumors of the thyroid gland were also observed in the highest male dose group.

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Hoechst Aktiengesellschaft. 1984. MRID No. 00151908. Available from the EPA.

Attachment

(94-007/04-11-94)

Risk Assessment Issue Paper for:
Derivation of a Provisional Inhalation RfC for
Carbon Tetrachloride (CASRN 56-23-5)

Introduction

Review documents available for carbon tetrachloride include a Health Assessment Document (U.S. EPA, 1984), a Drinking Water Criteria Document (U.S. EPA, 1985), an Updated Health Effects Assessment (U.S. EPA, 1989) and an Updated Toxicological Profile (ATSDR, 1992a). These documents were examined for information pertinent to derivation of an RfC for carbon tetrachloride. Computer searches of TOXLINE (1986-1992), TSCATS, HSDB and RTECS were performed in August, 1992 and screened for relevant information. Updated computer literature searches of TOXLINE (1992-1994), MEDLINE (1992-1994) and RTECS were conducted and screened in March, 1994. The OHEA CARA list (U.S. EPA, 1991, 1994), NTP status reports (NTP, 1994a, 1994b) and ATSDR Toxicological Profile Information Sheet (ATSDR, 1992b) were also consulted.

An inhalation RfC for carbon tetrachloride has not been discussed by the RfD/RfC Work Group (U.S. EPA, 1995a) and is not available on IRIS (U.S. EPA, 1995b) or in the HEAST (U.S. EPA, 1995c). However, ATSDR (1992a) derived an intermediate inhalation MRL of 0.01 ppm for carbon tetrachloride.

Review of Pertinent Literature

No studies were located regarding the subchronic or chronic toxicity of inhaled carbon tetrachloride vapor to humans. In animals, several subchronic inhalation studies were available. In the earliest of these studies, Smyth et al. (1936) exposed groups of 24 guinea pigs (strain not specified) and 24 Wistar-derived rats (mixed sexes of both species) to 50, 100, 200 or 400 ppm (315, 630, 1260 or 2520 mg/m^3) of carbon tetrachloride vapor (>99% pure), 8 hours/day 5 days/week for up to 10.5 months. The guinea pigs in this study received a purely vegetarian diet, but because the authors felt that low calcium in this diet may have affected the toxicity results, additional groups of 16 guinea pigs fed diets supplemented with calcium were tested at concentrations of 25 ppm (157 mg/m^3), as well as 50, 100 and 200 ppm. In addition to the rats and guinea pigs, groups of 4 monkeys (species and sex not specified) were exposed to 50 or 200 ppm using the same protocol. Use of controls was not described although controls apparently were included in the study. All animals were weighed weekly. Blood counts (all species) and urinalysis (guinea pigs and monkeys) were performed monthly. The fertility of rats and guinea pigs, which were housed in mixed-sex groups and produced litters during the study, was monitored. All animals that survived to scheduled sacrifice (including some animals which were sacrificed only after recovery periods of

varying durations), and most of those dying during the study, were examined for gross pathology. Tissue samples for histopathological examination were taken from the liver, kidney, adrenal gland, spleen, heart, sciatic and optic nerves and ocular muscle. Serum chemistry analyses were performed on some animals as well. No statistical tests were conducted. Guinea pigs of all exposure groups, including those that received diets supplemented with calcium, suffered substantial mortality (≥25-80% among "uninfected" guinea pigs). Mortality in controls was not reported. In contrast, mortality among "uninfected" rats was limited to 2 animals exposed to 400 ppm. No monkeys died during the study. Compared to controls, body weight gain was reported to be markedly reduced among survivors in all groups of guinea pigs. Body weight gain was also reduced by about 30% among rats exposed to 400 ppm. Too few litters were born to guinea pigs during the study to determine if exposure had any effect, but in rats fertility was reduced in the 200 and 400 ppm groups. In guinea pigs, fatty changes in the liver were seen at all dose levels, and cirrhosis developed at ≥50 ppm. In rats, fatty changes were seen at >50 ppm and cirrhosis at ≥100 ppm. In monkeys, mild fatty degeneration of the liver was found at both 50 and 200 ppm. Other pathological changes in animals exposed to these concentrations included renal tubular degeneration, degeneration of the adrenal glands (with necrosis in guinea pigs) and damage to the sciatic nerve. This study did not include concentrations low enough to identify a NOAEL for any of the 3 species tested. For guinea pigs, the low concentration of 25 ppm was a FEL that produced substantial mortality. For rats and monkeys, the low concentration of 50 ppm was a LOAEL that produced fatty changes in the liver.

Adams et al. (1952) conducted studies in which Wistar-derived rats (15-25/sex), outbred guinea pigs (5-9/sex), outbred rabbits (1-2/sex) and rhesus monkeys (1-2 of either sex) were exposed to carbon tetrachloride vapor (>99% pure), 7 hours/day and 5 days/week for 6 months at concentrations of 5, 10, 25, 50, 100, 200 or 400 ppm (31, 63, 157, 315, 630, 1260 or 2520 mg/m^3). Matched control groups, both unexposed and air-exposed, were included in these experiments. Animals were observed frequently for appearance and general behavior, and weighed twice weekly. Selected animals were used for hematological analyses periodically throughout the study. Moribund animals and those surviving to scheduled sacrifice were necropsied. The lungs, heart, liver, kidneys, spleen and testes were weighed, and sections from these and 10 other tissues were prepared for histopathological examination. In many cases, terminal blood samples were collected and used for serum chemistry analyses, and part of the liver was frozen and used for lipid analyses. The t test was used to make statistical comparisons. Guinea pigs were the most sensitive species tested. The primary target of carbon tetrachloride in guinea pigs was the liver. Liver effects progressed from a statistically significant increase in relative liver weight among females at 5 ppm to include slight-to-moderate fatty degeneration and increases in liver total lipid, neutral fat and esterified cholesterol at 10 ppm, and cirrhosis at 25 ppm. Liver effects became progressively more severe at higher concentrations. Growth retardation was first observed at 25 ppm and progressed to rapid loss of weight at 200 ppm. In the kidney, slight tubular degeneration was first observed at 200 ppm and increased kidney weight at 400 ppm. Mortality was increased at ≥100 ppm. A similar progression of effects was seen in rats, with no effects at 5 ppm, mild liver changes at 10 ppm, cirrhosis at 50 ppm and liver necrosis, kidney effects, testicular atrophy, growth depression and mortality at ≥200 ppm. In rabbits, 10 ppm was without effect, 25 ppm produced mild liver changes, 50 ppm produced moderate liver changes and 100 ppm produced growth depression. Monkeys were the most resistant

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- CARBON TETRACHLORIDE
- NAPHTHALENE
- TETRACHLOROETHYLENE

U.S. EPA. 1995. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

species tested, with evidence of adverse effects (mild liver lesions and increased liver lipid) only at 100 ppm, the highest concentration tested. This study identified a LOAEL of 5 ppm in guinea pigs and NOAEL and LOAEL values, respectively, of 5 and 10 ppm in rats, 10 and 25 ppm in rabbits and 50 and 100 ppm in monkeys, all based on hepatotoxic effects. The intermediate inhalation MRL calculated by ATSDR (1992a) was based on the occurrence of liver effects in rats in this study, using the same NOAEL and LOAEL values cited here.

Prendergast et al. (1967) exposed groups of 15 Sprague-Dawley or Long-Evans rats, 15 Hartley guinea pigs, 3 New Zealand rabbits, 2 beagle dogs and 3 squirrel monkeys (sexes not specified) to carbon tetrachloride vapor ("highest purity available") either by continuous exposure to 6.1 or 61 mg/m^3 (1 or 10 ppm) for 90 days or intermittent exposure (8 hours/day, 5 days/week) to 515 mg/m^3 (82 ppm) for 6 weeks. The control group consisted of 304 rats, 314 guinea pigs, 48 rabbits, 34 dogs and 57 monkeys. In order to generate the 6.1 mg/m^3 concentration, the researchers found it necessary to dilute the carbon tetrachloride in 61 mg/m^3 of n-octane. Therefore, a vehicle control group exposed to 62 mg/m^3 of n-octane was included in this study. Animals were observed routinely for signs of toxicity and weighed monthly. Blood samples for hematological analysis were taken at the end of the exposure period. Following sacrifice, animals were necropsied and sections of the heart, lung, liver, spleen and kidney were taken for histopathological examination. Serum chemistry and liver lipid analyses were performed on some animals. No statistical tests were conducted. Intermittent exposure to 515 mg/m^3 resulted in the death of 3 guinea pigs and 1 monkey. Body weight gain was reduced in all species, and all species except rats actually lost weight during the study. Mottled livers were seen in all species except dogs. Histopathological examination of the liver revealed fatty changes that decreased in severity from guinea pigs to rats to rabbits to dogs to monkeys. Liver lipid content of guinea pigs was increased about 3 fold compared to controls. The only other effect noted was interstitial inflammation in the lungs of all species. Continuous exposure to 61 mg/m^3 resulted in the deaths of 3/15 guinea pigs. Body weight gain was depressed in all species and monkeys appeared visibly emaciated. Gross examination showed the presence of enlarged/discolored livers in all species except dogs. Microscopic examination revealed fatty changes in the liver that were most prominent in rats and guinea pigs, but present in the other species as well. Lung effects were not reported in this group. Continuous exposure to 6.1 mg/m^3 produced reduced growth in all species, although the only histopathological findings were nonspecific inflammatory changes in the liver, kidney, heart and lungs. No effects were noted in the n-octane control group. The results of this study suggest a LOAEL of 6.1 mg/m^3 for all species tested based on reduced growth, but the study was limited by failure to compare growth to controls using statistical tests. It is not clear whether inflammatory changes observed in the lungs of some exposed animals occurred in controls as well.

More recent inhalation studies of carbon tetrachloride toxicity were performed for purposes other than determination of NOAEL and LOAEL values and were not useful for risk assessment for various reasons, including use of only a single exposure level (usually a high one), brief daily exposure periods and short study durations (Bogers et al., 1987; Hall et al., 1991; Plummer et al., 1990; Sakata et al., 1987; Vazquez et al., 1990)

As noted above, Adams et al. (1952) found testicular degeneration in rats exposed to 200 or 400 ppm (1260 or 2520 mg/m^3) of carbon tetrachloride vapor 7 hours/day, 5 days/week for 6 months. Testicular degeneration was also reported in rats given repeated i.p. doses of 1.5 ml/kg (Chatterjee, 1966; Kalla and Bansal, 1975). Therefore, there is reason to suspect that carbon tetrachloride might affect reproductive performance, and in fact, Smyth et al. (1936) found that fertility was reduced in rats exposed to 200 or 400 ppm (1260 or 2520 mg/m^3) of carbon tetrachloride vapor 8 hours/day 5 days/week for up to 10.5 months. However, reproduction was only studied incidentally in this experiment. Reproductive performance was also monitored in an oral study in which rats of an unspecified strain (18/sex/group) were fed for up to 2 years on experimental diets that had been fumigated with carbon tetrachloride for 48 hours (Alumot et al., 1976). The initial concentrations of carbon tetrachloride in the food were 0, 80 and 200 ppm. The fumigated diet was stored in sealed containers and feeding time was restricted to a single hour during the day and two hours at night so as to prevent excessive volatilization of carbon tetrachloride from the food. The investigators calculated that 60-70% of the carbon tetrachloride present in the food initially was actually present in food consumed by the rats. Based on this estimate and reference values for rat body weight (male = 0.38 kg, female = 0.229 kg) and food consumption (male = 0.03 kg/day, female = 0.021 kg/day) from U.S. EPA (1987), it was calculated that approximate carbon tetrachloride doses of 0, 4 and 10 mg/kg-day and 0, 5 and 12 mg/kg-day were delivered to males and females, respectively. The animals were housed in groups of 6 and segregated by sex for the first 6 weeks of the experiment. At this time the females, now 3 months old, were mated with untreated males. Mating was repeated approximately every 2 months, but using treated males rather than controls. For mating, each male was housed with two females for a 10-day period, during which the animals received the control diet After the mating period, the animals were returned to their original cages. Females were weighed two times a week, and were transferred to individual cages upon gaining 60 g. Litter size and weight were recorded at parturition and again 10 days later when the dams were returned to their original cages. Treatment with carbon tetrachloride had no effect on the reproductive parameters monitored (male and female fertility, litter size, and pup mortality and body weight at birth and weaning). There was widespread occurrence of chronic respiratory disease in animals from all groups after 14 months, but this probably did not affect these results since most reproductive activity took place during the first year of the study (only 7 successful matings occurred during the second year). Treatment-related parental toxicity was not reported, but only parental body weight was monitored concurrently with the reproductive part of the study. No evidence of liver toxicity was found by serum analyses or biochemical tests at the end of the study. In conclusion, this study found no evidence of reproductive or maternal effects at doses of 10-12 mg/kg-day in the feed.

The developmental effects of carbon tetrachloride have been studied following inhalation exposure. Groups of 22-23 pregnant female Sprague-Dawley rats were exposed to concentrations of 0, 334 or 1004 ppm (0, 2101 or 6316 mg/m^3) for 7 hours per day on gestation days 6-15 (Schwetz et al., 1974). Exposures to the two different dose levels were not performed concurrently, so two separate control groups were used. Data from the two control groups were combined except where they differed significantly (e.g., incidence of delayed ossification of sternebrae). The rats were observed daily throughout pregnancy. Food intake was monitored every other day during the experiment, and body weight was determined on days 6, 13 and 21 of gestation. Following sacrifice

on gestation day 21, the number and uterine position of live, dead and resorbed fetuses were recorded. The fetuses were weighed, measured, and examined for external anomalies. Half of the fetuses in each litter were prepared so as to enable detection of soft tissue anomalies upon subsequent examination, and the remainder were prepared and examined for skeletal abnormalities. The litter was considered the unit of treatment and observation when comparing the results from the different exposure groups. Nonpregnant female rats were exposed simultaneously with the pregnant rats in order to monitor effects on the liver. Serum GPT was determined in these rats throughout exposure and some were sacrificed for gross examination of the liver at the end of the exposure period. The remainder were sacrificed 6 days later (corresponding to the end of gestation in the pregnant rats) for GPT analysis, gross examination of the liver, and determination of liver weight. Significant reductions in fetal body weight and crown-rump length were found in both treated groups. The incidence of delayed ossification of the sternebrae was significantly elevated in the high-dose group compared to the concurrent control, but not compared to the low-dose group or its concurrent control. No other effects on the fetus were reported. Maternal toxicity was also observed in both dose groups. Food consumption and body weight were significantly reduced compared to controls, and hepatotoxicity was indicated by significantly elevated serum GPT, gross changes in liver appearance, and significantly increased liver weight. This study, therefore, detected both maternal and developmental toxicity at 334 ppm.

Several studies of the developmental toxicity of carbon tetrachloride following oral exposure have recently been conducted. Hamlin et al. (1993) treated pregnant female B6D2F1 mice with 0, 82.6 or 826 mg/kg of carbon tetrachloride by gavage in corn oil on days 1-5 of gestation. In this strain, days 1-5 of gestation are characterized by sequential cleavage of the fertilized oocyte to generate a hatched blastocyte, with implantation occurring on day 5 and organogenesis occurring subsequently. Therefore, dosing in this study was limited to the pre-implantation period. A total of 31 pregnant females were included in the experiment, with a minimum of 8 in each dose group (actual group sizes were not reported). Dams were allowed to give birth; litter size was recorded and neonates were weighed, measured for crown-rump length and checked for obvious birth defects. During lactation, the pups were weighed and measured for crown-rump length weekly. Lower incisor eruption and eye opening were assessed in all pups on postpartum days 11 and 15, respectively. Pups were weaned on postpartum day 22 and sacrificed. Dams were weighed weekly during pregnancy and on postpartum day 22 just prior to sacrifice. The liver and kidneys from the dams were removed and weighed. Tissue samples were collected, but not examined for histopathology in time for this report. Treatment with carbon tetrachloride had no effect on dam body weight during pregnancy or on absolute or relative liver or kidney weight at sacrifice. Treatment also had no effect on litter size, pup size at birth, the timing of developmental milestones (incisor eruption and eye opening) or pup growth through weaning (a statistically significant difference in body weight between high-dose pups and controls on day 15 postpartum was not considered to be biologically significant by the researchers since crown-rump length was not affected and no other body weight differences were found). No stillbirths or malformations were observed. The value of this study is limited because the results were not well reported and no data were shown.

In two recent abstracts, Narotsky et al. (1992, 1993) report that carbon tetrachloride induced

full-litter resorption in F-344 rats when pregnant rats were treated with 150 mg/kg by gavage in corn oil during organogenesis (GD 6-15 or GD 6-7). This dose level also caused maternal weight loss on GD 6-8. Resorption rates in surviving litters were unaffected by treatment.

Derivation of RfC

A provisional RfC for carbon tetrachloride can be derived from the study by Adams et al. (1952). This study delineated the progression of toxic effects associated with carbon tetrachloride inhalation and identified NOAEL/LOAEL values in four species. Guinea pigs were the most sensitive species tested, with a LOAEL of 5 ppm (31 mg/m^3), the lowest concentration included in the study. Designation of 31 mg/m^3 as a LOAEL for guinea pigs was based on a statistically significant increase in relative liver weight in females exposed to this concentration. Although increased liver weight does not necessarily indicate a toxic effect, in this case there is a clear progression of liver effects from increased liver weight to fatty changes to cirrhosis with increasing exposure concentration. Therefore, increased liver weight in this case was considered to be an early manifestation of carbon tetrachloride hepatotoxicity and 31 mg/m^3 was taken to be a LOAEL. It should be noted, however, that ATSDR (1992a) considered 31 mg/m^3 to be a NOAEL in guinea pigs and chose to use rats as the basis for the intermediate inhalation MRL.

To calculate the RfC, the LOAEL of 31 mg/m^3 is multiplied by the exposure period (7 hr/24 hr)(5 d/7 d) to obtain a duration-adjusted $LOAEL_{ADJ}$ of 6.5 mg/m^3. The human equivalent concentration (HEC) for an extrarespiratory effect produced by a vapor is calculated by multiplying the $LOAEL_{ADJ}$ by the ratio of blood:gas partition coefficients in animals and humans (L_A/L_H). Since a value for L_A is not available for guinea pigs, a default value of 1 is used for L_A/L_H and the $LOAEL_{HEC}$ becomes 6.5 mg/m^3. An uncertainty factor (UF) of 3000 was calculated from factors of 3 for use of a minimal LOAEL; 3 for interspecies extrapolation using a dosimetric adjustment; 3 for lack of a complete database including adequate study of respiratory, reproductive and developmental endpoints; 10 for use of a subchronic study and 10 to protect sensitive subpopulations. Dividing the $LOAEL_{HEC}$ of 6.5 mg/m^3 by the UF of 3000 produces a provisional RfC of 2E-3 mg/m^3 for carbon tetrachloride.

A provisional RfC of 2E-3 mg/m^3 was calculated for carbon tetrachloride based on hepatotoxicity in guinea pigs inhalation-exposed for 6 months by Adams et al. (1952). Confidence in the principal study is low because group sizes were small (5-9 guinea pigs/sex/group) and data regarding the incidence of histopathological lesions were not provided. Confidence in the data base is low because no toxicity studies of chronic duration were available and effects on the respiratory tract, reproduction and development were not adequately studied. Low confidence in the RfC follows.

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ATTACHMENT F-2

PROBABILITY DISTRIBUTIONS

ATTACHMENT F-2

PROBABILITY DISTRIBUTIONS

Body Weight

Receptor(s): Young Child Resident, and Young Child Lifetime Resident

Units: kg

@Risk Function: LOGNORM (14.9,4.0)

Justification:

Data on the body weight of children are available from the U.S. Public Health Service in the National Health and Nutrition Examination Survey (NHANES II). These data are reported in one year increments up to 20 years of age. In a review of these data, Lloyd and Burmaster (1994) found that Lognormal distributions give consistently strong fits to the NHANES II data across all age groups for each gender. This observation is consistent with previously published results for adult men and women (Brainard and Burmaster, 1992). Finley et al. (1994) also conducted a review of this data and reported similar observations.

Using the parameters for mean and standard deviation provided in Finley et al. (1994), lognormal distributions were fit to each yearly increment for male and female children ages 0.5-6 years. The final distribution for the group was fit to the group as a whole by randomly sampling body weight values from each one year increment, with equal probability. The resulting probability density function has a lognormal distribution with an arithmetic mean and standard deviation of 14.9 and 4.0 kg, respectively.

Outputs:

The distribution for body weight of the Young Child Lifetime Resident has these summary statistics: arithmetic mean = 14.88; median = 14.32; 95th percentile = 22.24; and maximum = 44.80. Figure 1 illustrates the probability density function for the Young Child Lifetime Resident's body weight.

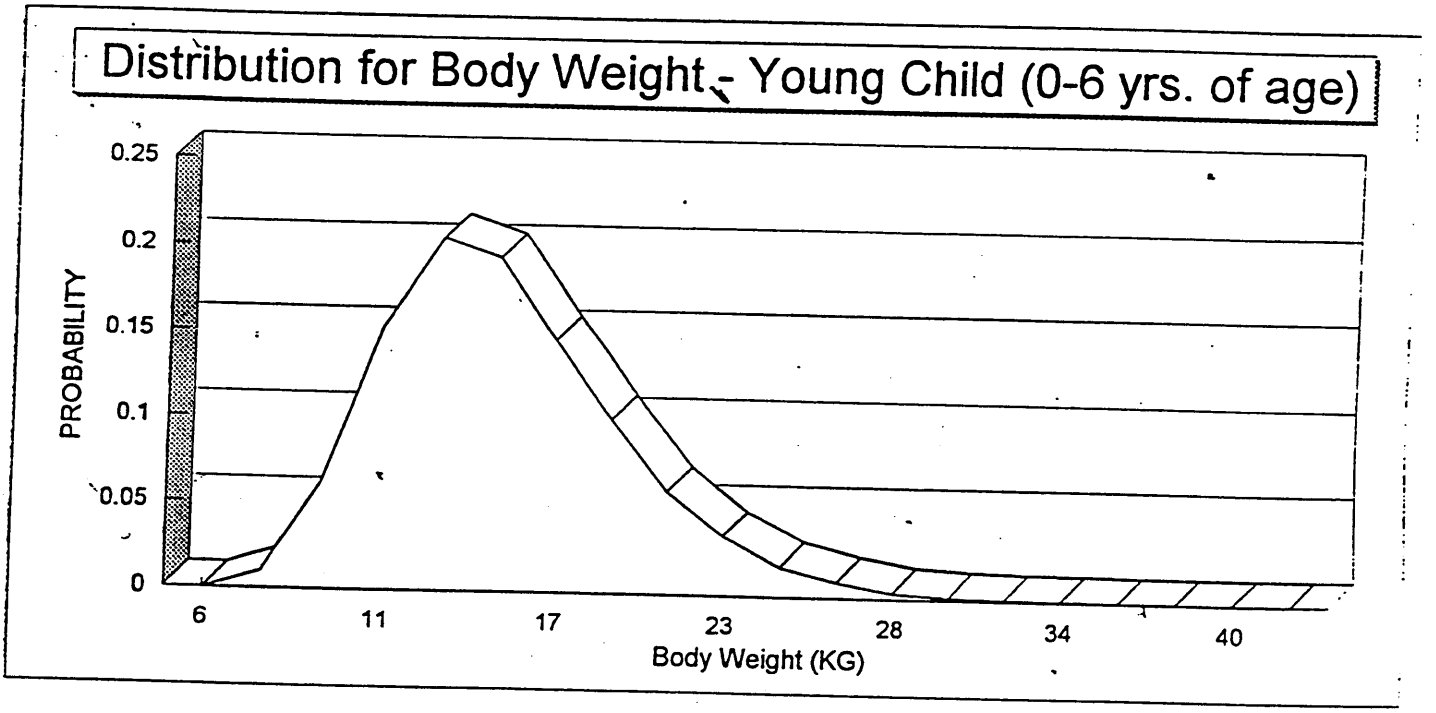

Figure 1 Distribution for Body Weight - Young Child (0-6 years of age)

Body Weight

Receptor: Older Child Lifetime Resident

Units: kg

@Risk Function: LOGNORM(42.1,16.1)

Justification:

Data on the body weight of children are available from the U.S. Public Health Service in the National Health and Nutrition Examination Survey (NHANES II). These data are reported in one year increments up to 20 years of age. In a review of these data, Lloyd and Burmaster (1994) found that Lognormal distributions give consistently strong fits to the NHANES II data across all age groups for each gender. This observation is consistent with previously published results for adult men and women (Brainard and Burmaster, 1992). Finley et al. (1994) also conducted a review of this data and reported similar observations.

Using the parameters for mean and standard deviation provided in Finley et al. (1994), lognormal distributions were fit to each yearly increment for male and female children ages 6-18 years. The final distribution for the group was fit to the group as a whole by randomly sampling body weight values from each one year increment, with equal probability. The resulting probability density function has a lognormal distribution with an arithmetic mean and standard deviation of 42.1 and 16.1 kg, respectively.

Outputs:

The distribution for body weight of the Older Child Lifetime Resident has these summary statistics: arithmetic mean = 42.02; median = 39.25; 95th percentile = 72.65; and maximum = 166.00 kg. Figure 2 illustrates the probability density function for the Older Child Lifetime Resident's body weight.

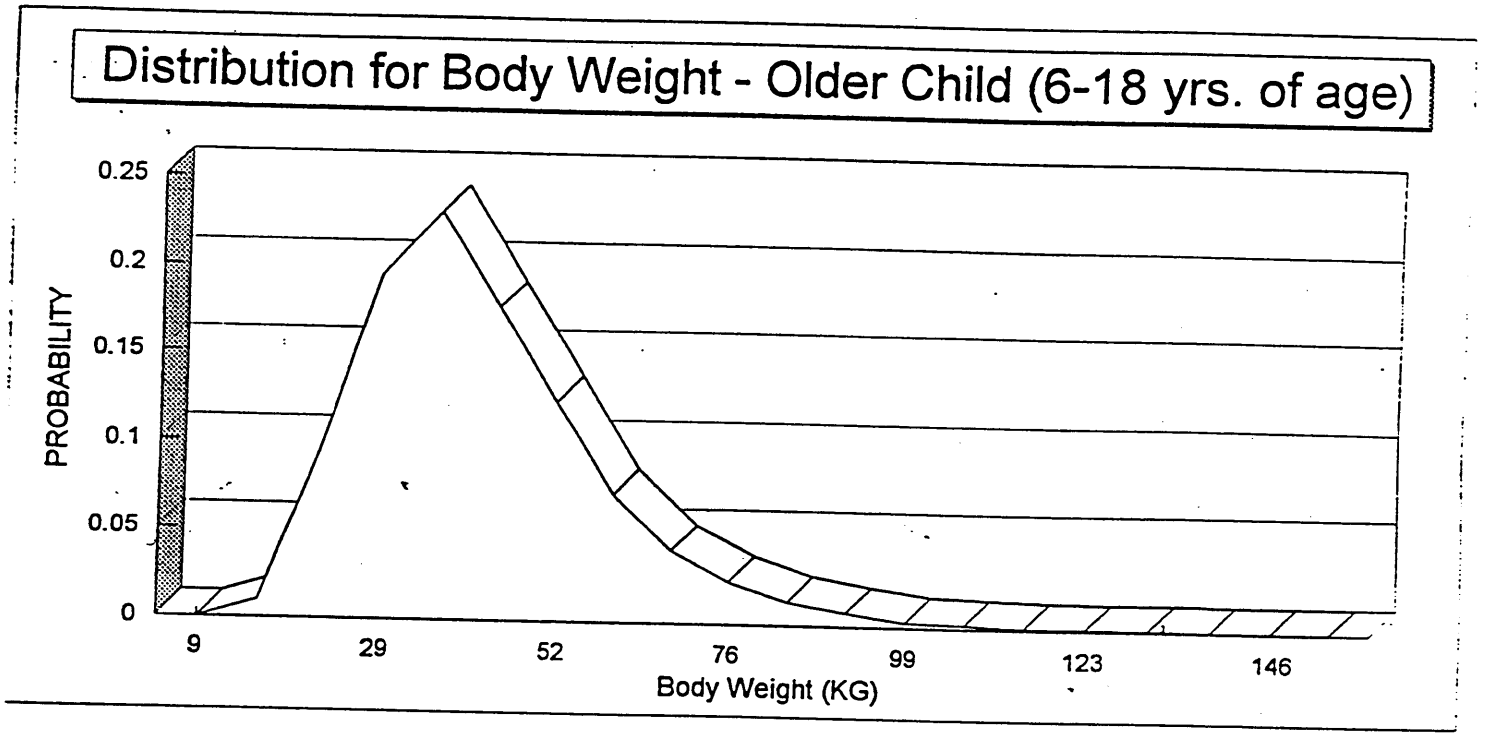

Figure 2 Distribution for Body Weight - Older child (6-18- Years of age)

Body Weight

Receptor: Adult Lifetime Resident

Units: kg

@Risk Function: LOGNORM(71.0,15.9)

Justification:

Data on height and weight is available from the U.S. Public Health Service in the National Health and Nutrition Examination Survey(NHANES II). These data cover the civilian noninstitutionalized population of the United States from the ages of six months to 74 years. In a review of these data, Brainard and Burmaster (1992) found that for men and women ages 18-74 years body weight is well fit for men and satisfactorily fit for women by a lognormal distribution. Finley et al (1994) developed cumulative adult body weight distributions for both sexes combined based on the NHANES II data as adjusted by Brainard and Burmaster (1992). Finley et al. (1994) found the body weights of adults of both sexes over the age of eighteen fit a lognormal distribution with a mean of 71.0 kg and a standard deviation of 15.9 kg.

Integrating the above information, a Lognormal distribution having a mean of 71.0 kg and a standard deviation of 15.9 kg was used to represent the body weight of the Adult Lifetime Resident.

Outputs:

The distribution for body weight of the Adult Lifetime Resident has these summary statistics: arithmetic mean = 70.76; median = 68.88; 95th percentile = 99.65; and maximum = 162.90 kg. Figure 3 illustrates the probability density function (PDF) for the Adult Lifetime Resident's body weight.

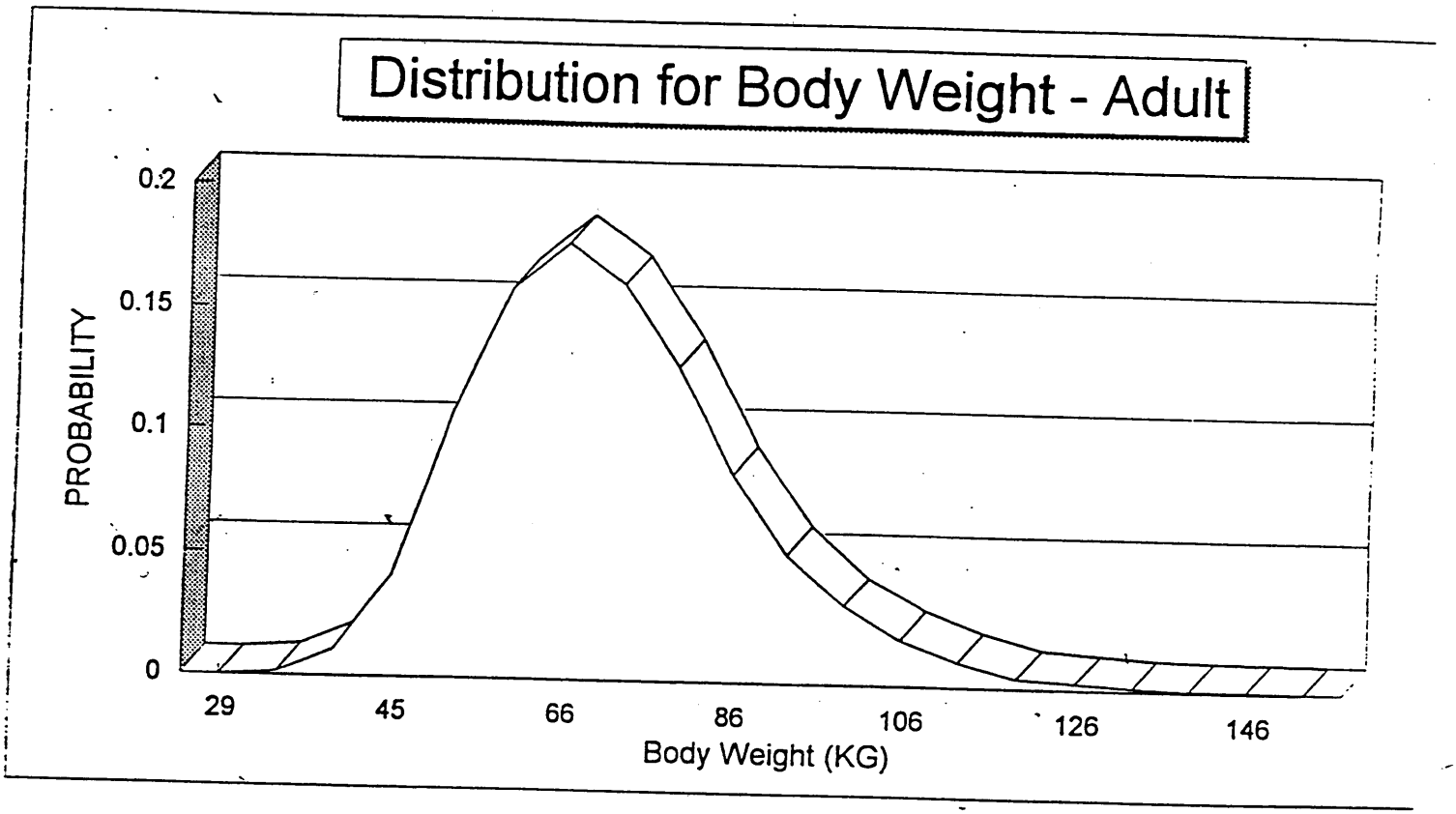

Figure 3 Distribution for Body Weight - Adult

Inhalation Rate

Receptor(s): Young Child Resident, and Young Child Lifetime Resident

Units: m^3/day

Formula: BMR*A*H*VQ where BMR is the basal metabolic rate, A is the ratio of daily energy expenditure to daily BMR, H is 0.05m^3 of O_2/MJ, and VQ is the ventilatory equivalent (ratio of air inhaled to oxygen uptake) (Finley et al. 1994).

@Risk Function: (0.168*(body weight) + 0.972) * 1.6 * 0.05 * (lognorm2(ln(27),ln(1.18))

Layton (1993) presents methods for estimating inhalation rate which are based on the oxygen uptake associated with energy expenditure (basal metabolic rate, BMR). The method utilized here in deriving a distribution for inhalation rate incorporates individual variation in metabolism and efficiency of oxygen uptake, both of which affect inhalation rates (Finley et al. 1994). The formula for calculating the inhalation rates incorporates distributions for body weight and ventilatory equivalent (ratio of air inhaled to oxygen uptake). BMR is correlated with body weight. Layton (1993) characterizes ventilatory equivalent using a lognormal distribution. Body weight is characterized by a lognormal distribution having a mean of 14.9 kg, and standard deviation of 4.0 kg. The ventilatory equivalent is characterized by a lognormal distribution with geometric mean of 27 and standard deviation of 1.18. The equation for BMR was derived from the BMR equations for different age groups reported by Layton (1993). Layton (1993) identified daily energy ratios (represented by A in the above equation) which are consistent with the results of other studies (Basiotis et al., 1989). The value of A utilized in deriving the distribution of inhalation rate for the Young Child Lifetime Resident is an average for males and females.

Outputs:

The distribution for inhalation rate of the Young Child Lifetime Resident has these summary statistics: arithmetic mean = 7.59; median = 7.34; 95th percentile = 11.23; and maximum = 21.30 m^3/day. Figure 4 illustrates the probability density function for the Young Child Lifetime Resident's inhalation rate.

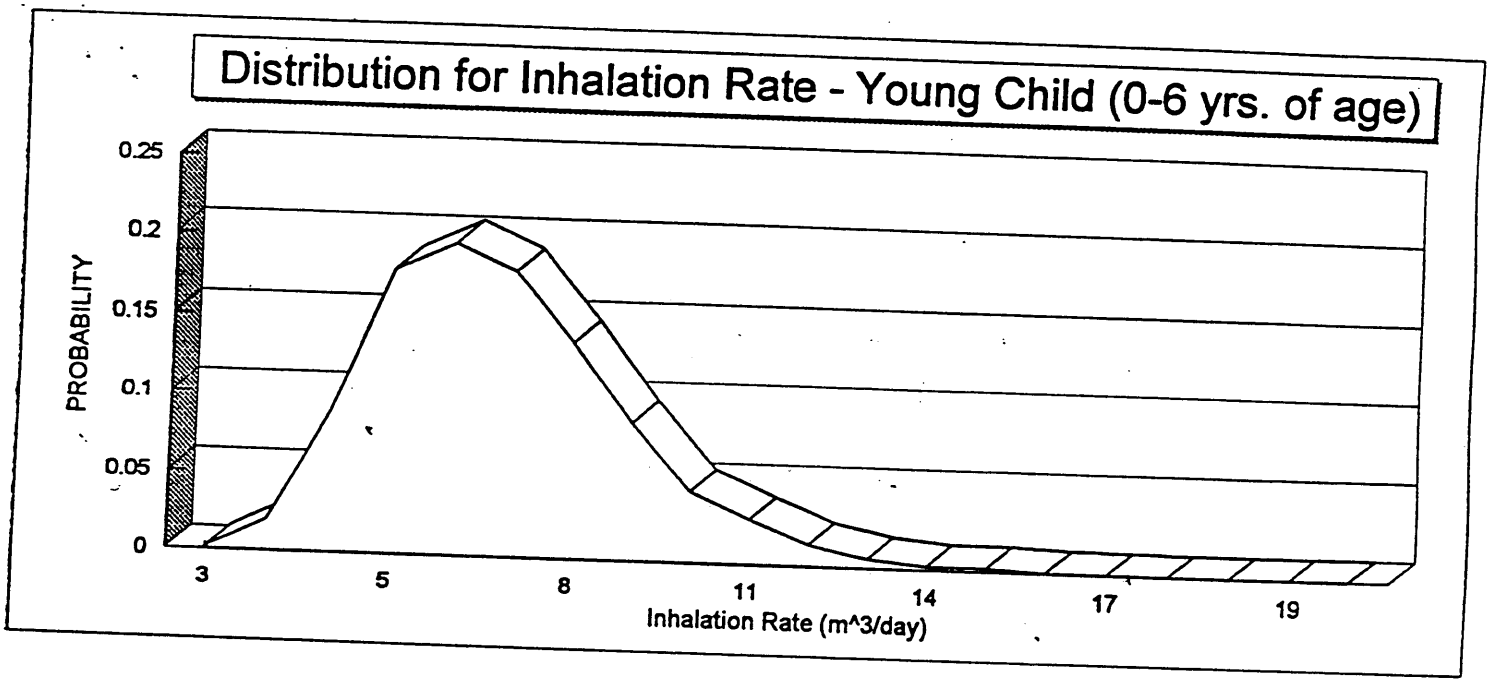

Figure 4 Distribution for Inhalation Rate - Young Child (0-6 years of age)

Inhalation Rate

Receptor(s): Older Child Lifetime Resident

Units: m^3/day

Formula: BMR*A*H*VQ where BMR is the basal metabolic rate, A is the ratio of daily energy expenditure to daily BMR, H is 0.05m^3 of O_2/MJ, and VQ is the ventilatory equivalent (ratio of air inhaled to oxygen uptake) (Finley et al. 1994).

@Risk Function: (0.065*(body weight) + 2.826) * 1.6 * 0.05 * (lognorm2(ln(27),ln(1.18))

Layton (1993) presents methods for estimating inhalation rate which are based on the oxygen uptake associated with energy expenditure (basal metabolic rate, BMR). The method utilized here in deriving a distribution for inhalation rate incorporates individual variation in metabolism and efficiency of oxygen uptake, both of which affect inhalation rates (Finley et al. 1994). The formula for calculating the inhalation rates incorporates distributions for body weight and ventilatory equivalent (ratio of air inhaled to oxygen uptake). BMR is correlated with body weight. Layton (1993) characterizes ventilatory equivalent using a lognormal distribution. Body weight is characterized by a lognormal distribution having a mean of 42.1 kg, and standard deviation of 16.1 kg. The ventilatory equivalent is characterized by a lognormal distribution with geometric mean of 27 and standard deviation of 1.18. The equation for BMR was derived from the BMR equations for different age groups reported by Layton (1993). Layton (1993) identified daily energy ratios (represented by A in the above equation) which are consistent with the results of other studies (Basiotis et al., 1989). The value of A utilized in deriving the distribution of inhalation rate for the Older Child Lifetime Resident is an average for males and females.

Outputs:

The distribution for inhalation rate of the Older Child Lifetime Resident has these summary statistics: arithmetic mean = 12.16; median = 11.73; 95th percentile = 17.75; and maximum =36.00 m^3/day. Figure 5 illustrates the probability density function for the Older Child Lifetime Resident's inhalation rate.

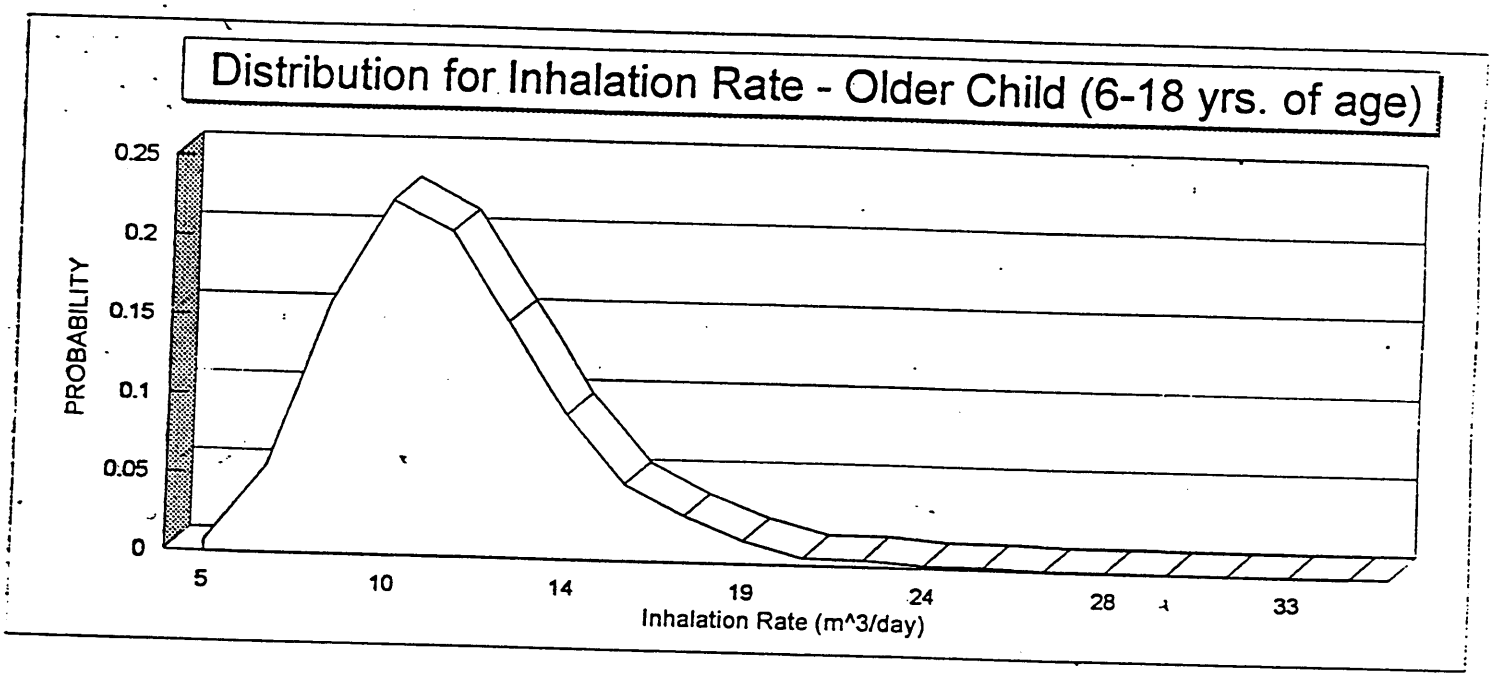

Figure 5 Distribution for Inhalation Rate - Older Child (6-18 years of age)

Inhalation Rate

Receptor(s): Adult Lifetime Resident

Units: m^3/day

Formula: BMR*A*H*VQ where BMR is the basal metabolic rate, A is the ratio of daily energy expenditure to daily BMR, H is 0.05m^3 of O_2/MJ, and VQ is the ventilatory equivalent (ratio of air inhaled to oxygen uptake) (Finley et al. 1994).

@Risk Function: (0.052*(body weight) + 3.031) * 1.49 * 0.05 * (lognorm2(ln(27),ln(1.18))

Layton (1993) presents methods for estimating inhalation rate which are based on the oxygen uptake associated with energy expenditure (basal metabolic rate, BMR). The method utilized here in deriving a distribution for inhalation rate incorporates individual variation in metabolism and efficiency of oxygen uptake, both of which affect inhalation rates (Finley et al. 1994). The formula for calculating the inhalation rates incorporates distributions for body weight and ventilatory equivalent (ratio of air inhaled to oxygen uptake). BMR is correlated with body weight. Layton (1993) characterizes ventilatory equivalent using a lognormal distribution. Body weight is characterized by a lognormal distribution having a mean of 71.0 kg, and standard deviation of 15.9 kg. The ventilatory equivalent is characterized by a lognormal distribution with geometric mean of 27 and standard deviation of 1.18. The equation for BMR was derived from the BMR equations for different age groups reported by Layton (1993). Layton (1993) identified daily energy ratios (represented by A in the above equation) which are consistent with the results of other studies (Basiotis et al., 1989). The value of A utilized in deriving the distribution of inhalation rate for the Adult Lifetime Resident is an average for males and females.

Outputs:

The distribution for inhalation rate of the Adult Lifetime Resident has these summary statistics: arithmetic mean = 13.70; median = 13.37; 95th percentile = 18.95; and maximum = 32.24 m^3/day. Figure 6 illustrates the probability density function for the Adult Lifetime Resident's inhalation rate.

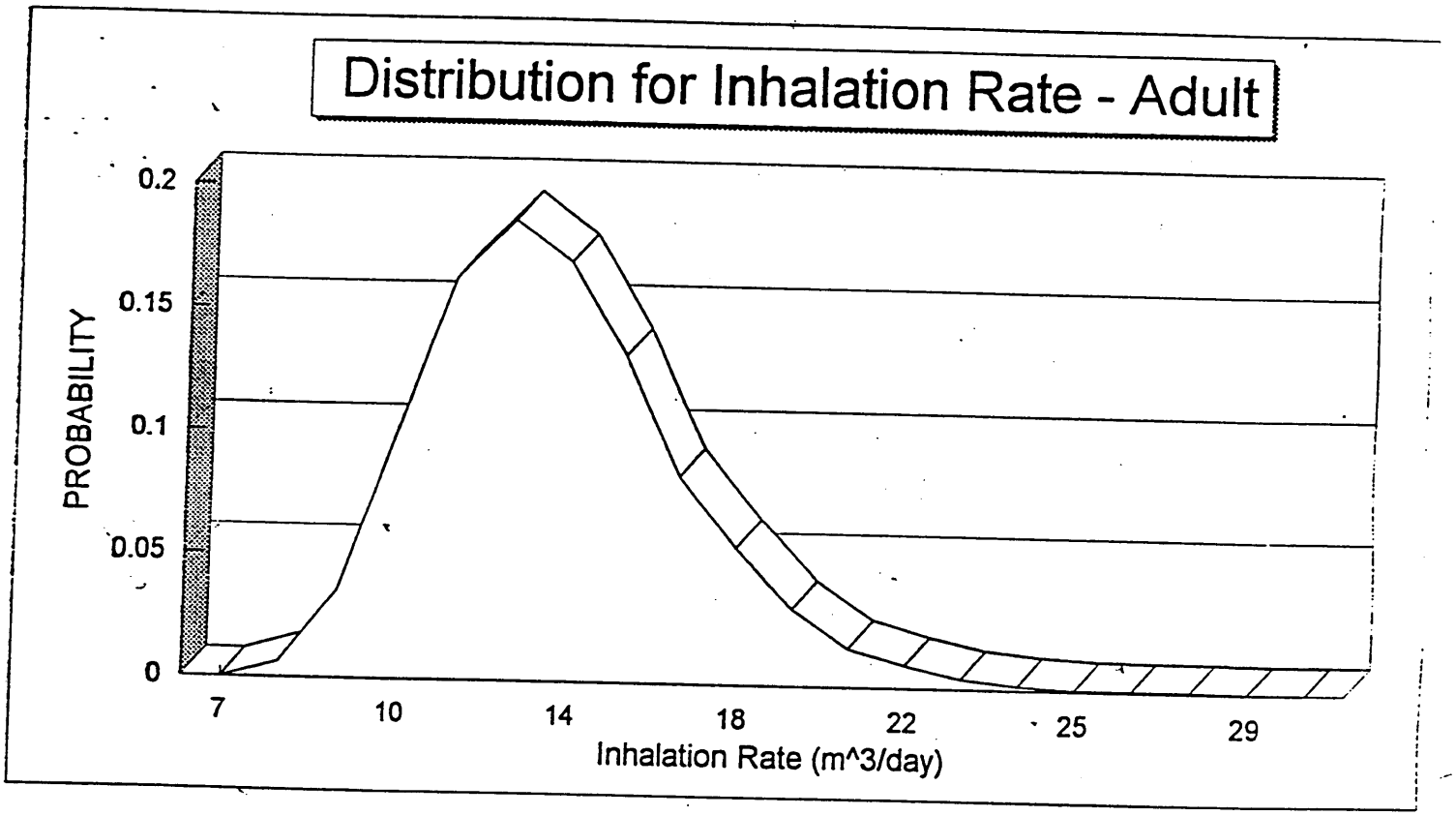

Figure 6 Distribution for Inhalation Rate - Adult

Exposure Frequency

Receptors(s): Young Child Lifetime Resident, Older Child Lifetime Resident, Adult Lifetime Resident and Young Child Resident

Units: days/year

@Risk Function: INT(UNIFORM(344,365))

Justification:

A distribution for exposure frequency was derived, based on best professional judgement, for all receptors evaluated in the probabilistic analysis. This judgement took into account the climate of the region and the possibility of absence from the region due to travel (vacation or business). We have assumed that the temperate nature of the climate in Arizona allows for the possibility of outdoor activities, and thus exposure to compounds of interest present in the air, virtually everyday of the year. We have also assumed that it is likely residents will travel outside of their home counties at least one day per year and possibly as many as 21 days per year. In the derivation of the exposure frequency distribution it was also assumed that every day in the range of days (1-21) spent traveling outside the county had an equal likelihood of occurrence. On this basis a Uniform distribution with a minimum value of 344 days and a maximum value of 365 was chosen to represent exposure frequency for all of the receptors considered in the probabilistic analysis. In order to consider on integers within the range of possible exposure frequencies, the INT function was used in conjunction with @Risk Uniform distribution function.

Outputs:

The distribution for exposure frequency for all receptors considered in the probabilistic analysis has these summary statistics: arithmetic mean = 354; median = 354; 95th percentile = 363; and maximum = 365 days per year. Figure 7 illustrates the probability density function of exposure frequency for all receptors evaluated in the probabilistic analysis.

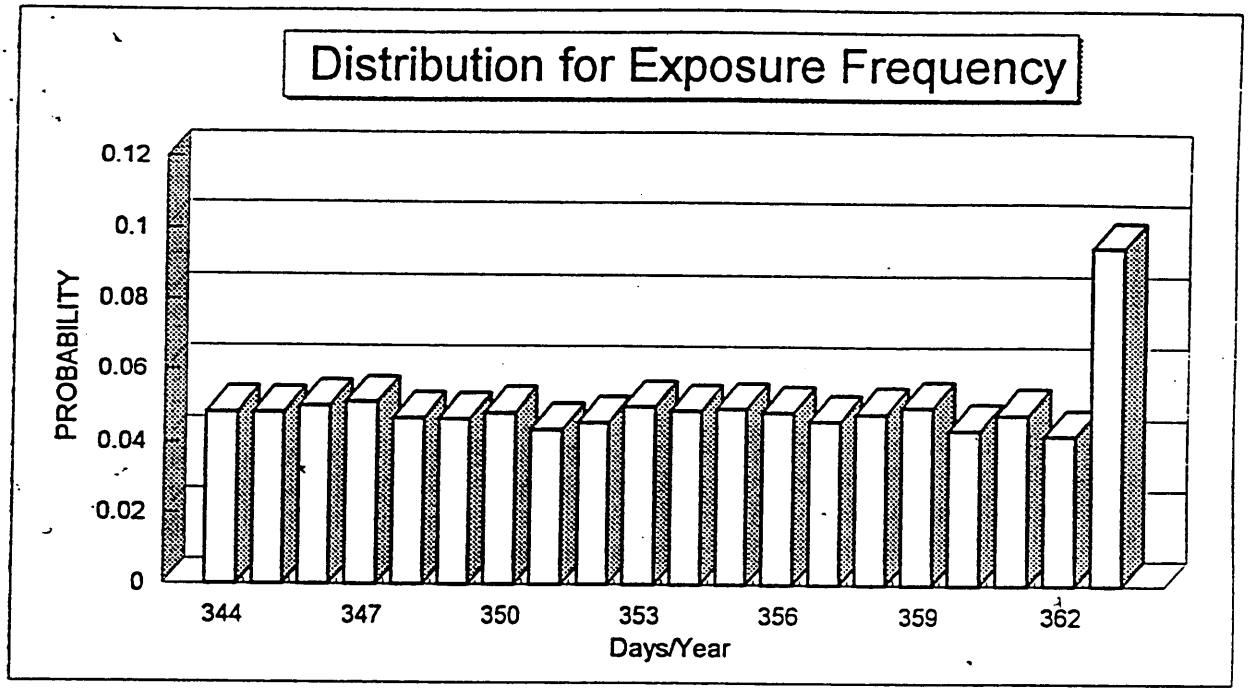

Figure 7 Distribution for Exposure Frequency

Exposure Duration

Receptor(s): Young Child Resident, and Young Child Lifetime Resident

Units: years

@Risk Function: CUMUL(1,85,12,21,34,50,59,76,0.25,0.5,0.75,0.9,0.95,0.99)

Justification:

Since the Arizona HAPs research program is evaluating exposure to HAPs on a regional basis, a distribution of residential occupancy period within a region (regional occupancy period) is a more appropriate estimator of exposure to HAPS than a distribution of residential occupancy period within a single house.

In order to estimate a distribution for regional occupancy period, ENSR has developed a PC-based version of the U.S. EPA's "ROPSIM" model. ENSR has validated this model by reconstructing the results presented by the authors of the EPA model for single residence occupancy periods. U.S. census data for years spent in the current county was used as input to the model to develop a probabilistic distribution for years spent in the current county. For the HAPs evaluation, it is assumed that the per-county value is predictive of a per-region value. A cumulative distribution, with a minimum of 1.0 and a maximum of 85 years, was fit to the resulting statistics. In a separate cell, a formula was constructed which referred to the value picked from the distribution during each iteration. All values greater than six years were set equal to 6 to reflect the age range of the Young Child.

Outputs:

The distribution for the Young Child Lifetime Resident's Exposure Duration has these summary statistics: arithmetic mean = 5.71; median = 6.0; 95th percentile = 6.0; and maximum = 6.0 years. Figure 8 illustrates the probability density function for the Young Child Lifetime Resident's Exposure Duration.

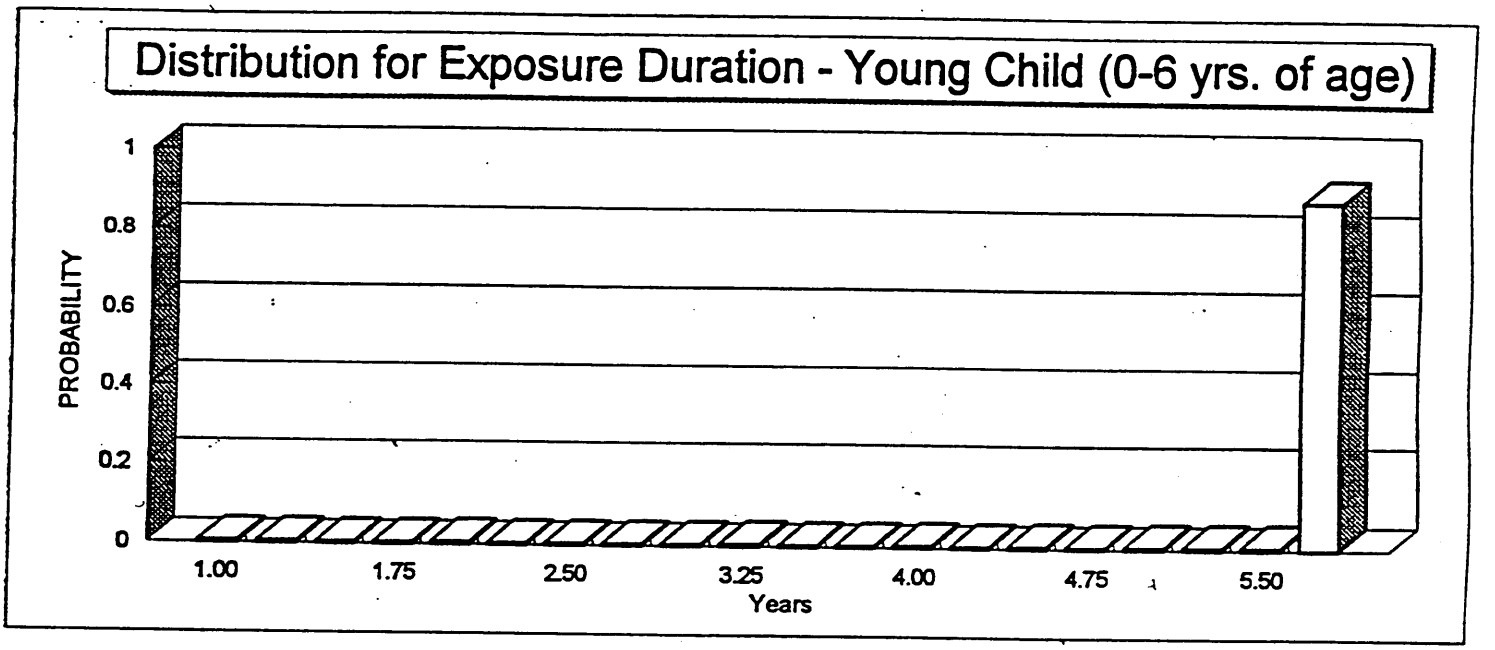

Figure 8 Distribution for Exposure Duration - Young child (0-6 years of age)

Exposure Duration

Receptor(s): Older Child Lifetime Resident

Units: years

@Risk Function: CUMUL(1,85,12,21,34,50,59,76,0.25,0.5,0.75,0.9,0.95,0.99)

Justification:

Since the Arizona HAPs research program is evaluating exposure to HAPs on a regional basis, a distribution of residential occupancy period within a region (regional occupancy period) is a more appropriate estimator of exposure to HAPS than a distribution of residential occupancy period within a single house.

In order to estimate a distribution for regional occupancy period, ENSR has developed a PC-based version of the U.S. EPA's "ROPSIM" model. ENSR has validated this model by reconstructing the results presented by the authors of the EPA model for single residence occupancy periods. U.S. census data for years spent in the current county was used as input to the model to develop a probabilistic distribution for years spent in the current county. For the HAPs evaluation, it is assumed that the per-county value is predictive of a per-region value. A cumulative distribution, with a minimum of 1.0 and a maximum of 85 years, was fit to the resulting statistics. In a separate cell, a formula was constructed which referred to the value picked from the distribution during each iteration. All values greater than twelve years were set equal to 12 to reflect the age range of the Older Child.

Outputs:

The distribution for the Older Child Lifetime Resident's Exposure Duration has these summary statistics: arithmetic mean = 10.60; median = 12.00; 95th percentile = 12.00; and maximum = 12.00 years. Figure 9 illustrates the probability density function for the Older Child Lifetime Resident's Exposure Duration.

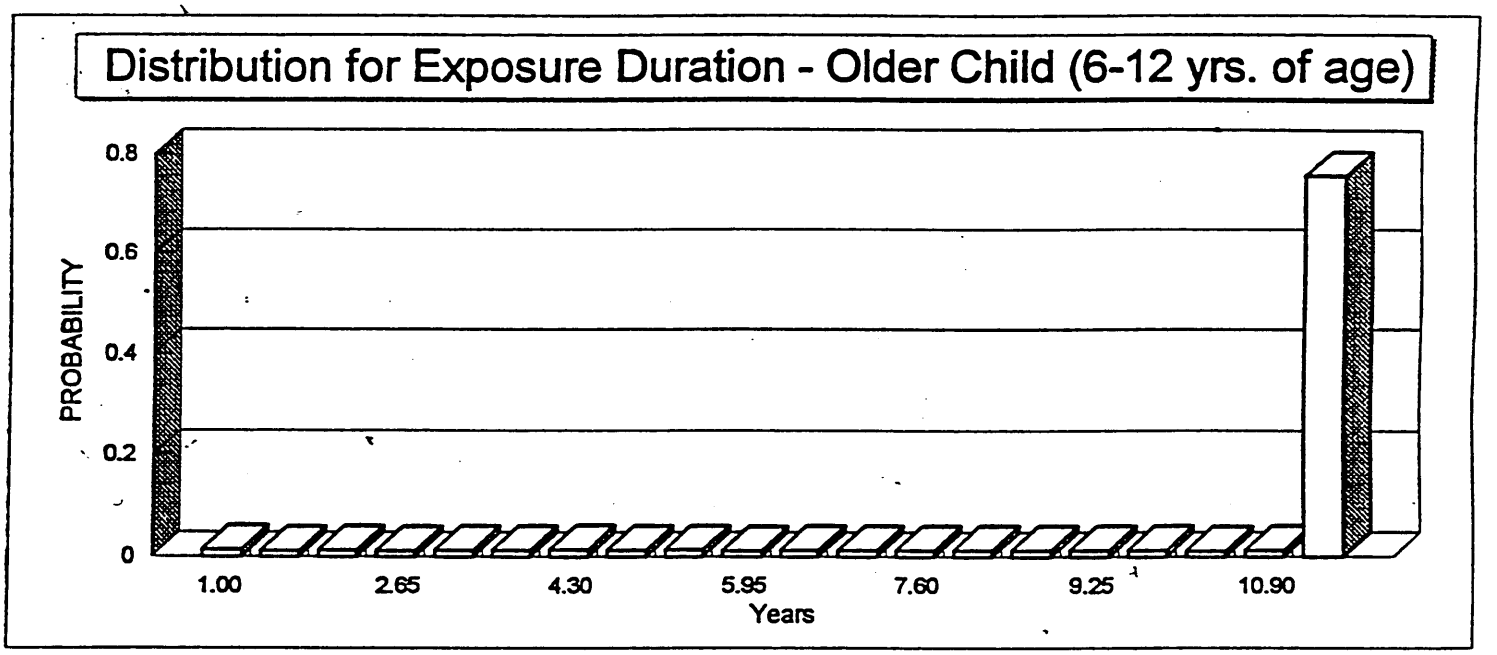

Figure 9 Distribution for Exposure Duration - Older Child (6-12 years of age)

Exposure Duration

Receptor(s): Adult Lifetime Resident

Units: years

@Risk Function: CUMUL(1,85,12,21,34,50,59,76,0.25,0.5,0.75,0.9,0.95,0.99)

Justification:

Since the Arizona HAPs research program is evaluating exposure to HAPs on a regional basis, a distribution of residential occupancy period within a region (regional occupancy period) is a more appropriate estimator of exposure to HAPS than a distribution of residential occupancy period within a single house.

In order to estimate a distribution for regional occupancy period, ENSR has developed a PC-based version of the U.S. EPA's "ROPSIM" model. ENSR has validated this model by reconstructing the results presented by the authors of the EPA model for single residence occupancy periods. U.S. census data for years spent in the current county was used as input to the model to develop a probabilistic distribution for years spent in the current county. For the HAPs evaluation, it is assumed that the per-county value is predictive of a per-region value. A cumulative distribution, with a minimum of 1.0 and a maximum of 85 years, was fit to the resulting statistics. In a separate cell, a formula was constructed which referred to the value picked from the distribution during each iteration. All values greater than sixty-seven years were set equal to 67 to reflect the age range of the Adult.

Outputs:

The distribution for the Adult Lifetime Resident's Exposure Duration has these summary statistics: arithmetic mean = 24.79; median = 20.71; 95th percentile = 58.42; and maximum = 67.00 years. Figure 10 illustrates the probability density function for the Adult Lifetime Resident's Exposure Duration.

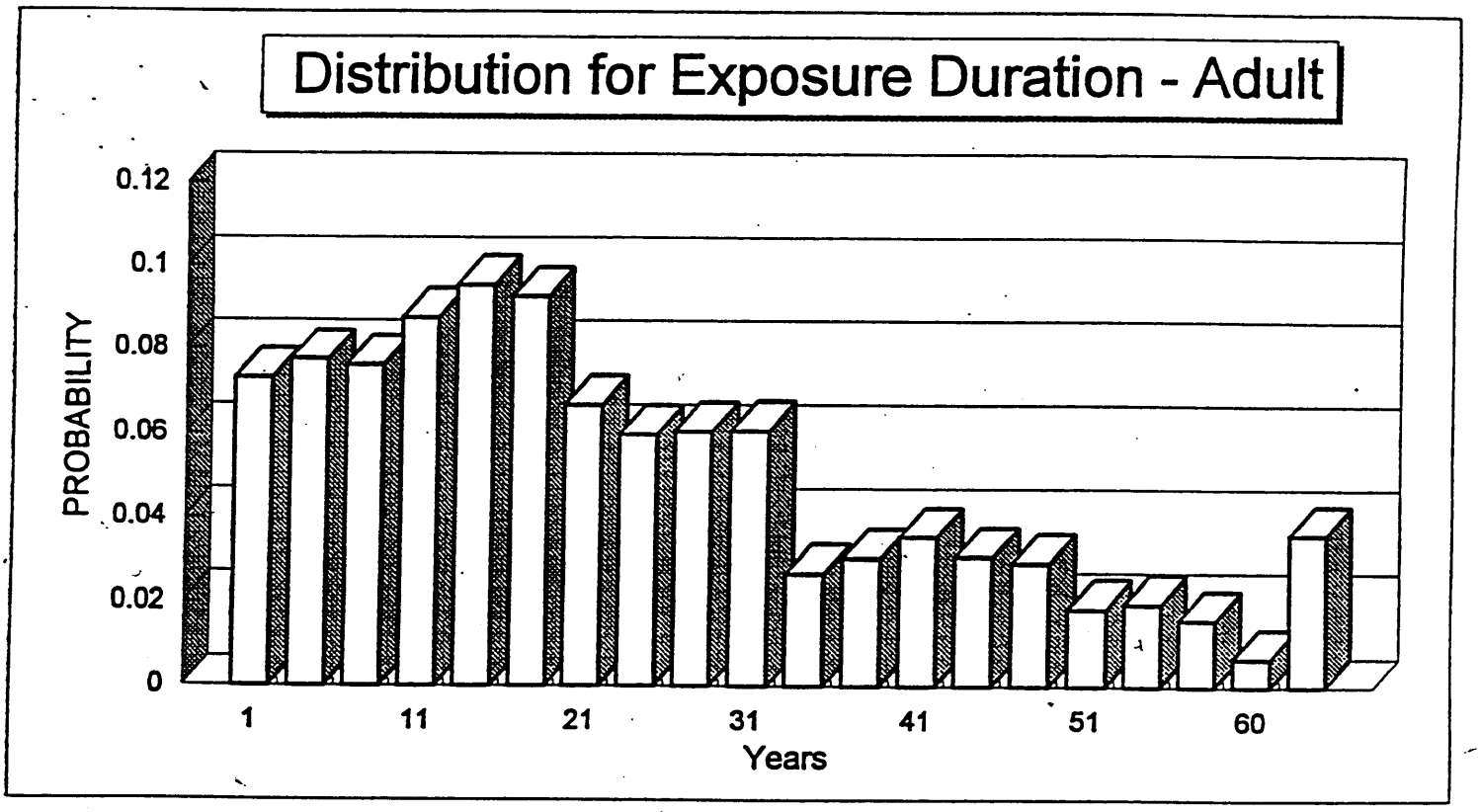

Figure 10 Distribution for Exposure Duration - Adult

Risk Assessment Issue Paper for: Derivation of Provisional Chronic RfC for Tetrachloroethylene (CASRN 127-18-4)

INTRODUCTION

Guidelines for oral, but not inhalation, exposure to tetrachloroethylene have developed by EPA. A chronic RfD of 0.01 mg/kg/day is available on IRIS (U.S. EPA, 1995). In addition, the HEAST (U.S. EPA, 1994b) has a subchronic oral RfD of 0.1 mg/kg/day for tetrachloroethylene. Neither subchronic or chronic inhalation RfCs are available on IRIS or HEAST. The Drinking Water Regulations and Health Advisories list (U.S. EPA, 1994a) indicates an MCLG of zero, an MCL of 0.005 mg/L, 1- and 10-day health advisories of 2 mg/L (child), longer-term health advisories of 1 mg/L (child) and 5 mg/L (adult), and a DWEL of 0.5 mg/L. Documents listed on the CARA list (U.S. EPA, 1994c) include a Water Quality Criteria Document (U.S. EPA, 1985a), a Health Effects Assessment (U.S. EPA, 1988) and several Health Assessment Documents (U.S. EPA, 1982, 1983, 1985b, 1986). ATSDR has prepared a toxicological profile on tetrachloroethylene. An acute inhalation MRL of 0.6 ppm and an intermediate inhalation MRL of 0.009 ppm were derived for tetrachloroethylene. No chronic inhalation MRL was derived. An intermediate oral MRL of 0.1 mg/kg/day was derived, but no acute or chronic oral MRLs were derived for tetrachloroethylene. The odor threshold for tetrachloroethylene in air is ≈300 mg/m^3 (50 ppm) (Carpenter, 1937).

To identify research reports pertinent to the derivation of a provisional RfC for tetrachloroethylene, the EPA and ATSDR documents (as cited above) were reviewed. In addition, a computerized search of the literature was conducted from the HSDB, RTECS, ETIC, Toxline, Medline, and DART (1985-1993). The oral database was also considered.

REVIEW OF PERTINENT LITERATURE

Tetrachloroethylene is widely used for dry cleaning fabrics and for metal-degreasing operations (ATSDR, 1993). Various symptoms have been reported by individuals exposed acutely to tetrachloroethylene vapors, irritation of mucosa (eyes, upper respiratory tract), gastrointestinal distress, alteration of liver and kidney function parameters, central nervous system (CNS) effects (fatigue, dizziness, weakness, headache, memory loss, behavioral changes, EEG disturbances) (Carpenter, 1937; Freed and Kandel, 1988; Hake and Stewart, 1977; Rowe et al., 1952; Coler and Rossmiller, 1953; Stewart et al., 1970). Acute exposures to high concentrations of tetrachloroethylene also result in anesthesia (ATSDR, 1993). Following prolonged exposures, the brain, liver, and kidney have been reported as the target organs in humans. Because of its high lipophilicity, tetrachloroethylene is stored in tissues with high lipid content, such as perirenal fat, brain, and liver tissues (NTP, 1986). The respiratory system has not been demonstrated to be a major target organ of inhalation exposure to tetrachloroethylene.

Human Occupational Studies. Data regarding the toxicity of inhaled tetrachloroethylene in humans

for longer durations are from occupational studies on exposed workers in the dry-cleaning industry. The data from these studies are limited due to inadequate monitoring data (which may not accurately represent exposures to the workers over the chronic durations).

Lauwerys et al. (1983) studied workers (24 females, 2 males) (average age of 32.9 years) employed in 6 dry-cleaning shops in Belgium. The time-weighted-average (TWA) exposure to tetrachloroethylene was 20.8 ppm (141 mg/m^3)(range of 8.9–37.5 ppm), with a mean exposure duration of 6.4 years (0.1–25 years). A group of 33 individuals, occupationally exposed to organic solvents in a chocolate factory or occupational health service, represented the control group, and was matched with the exposed group for gender, age, and educational level. No significant differences were found in subjective symptoms reported by both groups, although memory loss (7/26 vs. 3/33 in controls) and difficulty in falling asleep (11/26 vs. 6/33 in controls) were noted. Also, the exposed group exhibited no significant alterations in psychomotor tests (critical flicker fusion, simple and 9-choice visual reaction time, and sustained attention tests) performed before and after the workshift. Furthermore, the levels of albumin and β_2-microglobulin in urine and β_2-microglobulin and SGPT in serum were not statistically different. The study results were limited because the size of the groups was small and the exposure concentration was based on monitoring data collected from a single work day. The free-standing NOAEL for the study was 20.8 ppm ($NOAEL_{HEC}$ = 34 mg/m^3, assuming an average work day of 8 hours/day, 5 days/week) for neurological and renal effects.

In a recent study by Mutti et al. (1992), renal function was assessed in 9 men and 41 women (average age of 41 years) from dry-cleaning shops who were exposed to 14.8 ppm (100 mg/m^3) tetrachloroethylene (ranging from trace amounts to 85 ppm) for 10 years. The controls consisted of 50 blood donors matched by age, gender, and tobacco, alcohol, and drug consumption with the exposed workers. Indicators of renal toxicity were measured. Urinary albumin, transferrin, brush-border antigens BBA, BB50, and HF5, tissue non-specific alkaline phosphatase, and fibronectin, as well as serum anti-glomerular basement membrane antibodies and laminin fragments, were significantly altered in the exposed group compared to controls. Furthermore, the frequencies of abnormal values for urinary albumin, β_2-microglobulin, transferrin, IgG, glycosaminoglycans, and antigens BBA and HF5 were significantly greater in the exposed group compared to controls. These findings suggest there were diffuse structural and functional changes (i.e., membrane disturbances) within the kidney. However, serum and urinary β_2-microglobulin and serum creatinine levels were not significantly affected, indicating that major impairment of renal function did not occur. The investigators concluded that the significance of these changes cannot be easily assessed, and may be due to a shift of physiological adaption or response to other stimuli. Also, the failure to correlate the renal changes with the intensity or duration of exposure limited the study. The LOAEL for the study was 14.8 ppm ($LOAEL_{HEC}$ = 24 mg/m^3, duration adjusted for 8 hours/day, 5 days/week) for renal effects.

Vyskocil et al. (1990) examined the effect of tetrachloroethylene exposure on kidney function in female workers from five dry-cleaning shops. Sixteen female workers (average age of 42 years) were exposed to 157 mg/m^3 tetrachloroethylene (range of 9–799 mg/m^3) for an average of 9 years (1–18 years); air sampling was carried out by workers for a one-year period. In three of the dry-cleaning shops, 20–80% of the individual values were greater than 250 mg/m^3. Control and exposed groups were matched for age, gender, exposure duration, smoking habit, and alcohol and analgesic

consumption. Lysozyme in the urine was significantly increased ($p<0.05$) in the exposed workers compared to controls, but urinary excretion of albumin, β_2-microglobulin, LDH, or total protein was not affected. However, the prevalences of abnormal values for the urinary parameters were not statistically different between the groups. The investigators concluded that it was difficult to interpret the effect (lysozymuria) as a result of tetrachloroethylene-induced tubular damage since the other sensitive markers of tubular dysfunction were normal. The study was also limited by the small size of the test groups, inadequate monitoring data for the entire exposure period, the large range of mean exposure concentrations and durations for the exposed group, and the lack of correlation between the increased urinary lysozyme effect and the intensity of exposure. A LOAEL of 157 mg/m^3 ($LOAEL_{HEC}$ = 37 mg/m^3, duration adjusted for 8 hours/day, 5 days/week) was determined for the study based on renal effects.

In a cross-sectional study by Franchini et al. (1983), 57 workers (mostly females; average age of 43 years), employed in dry-cleaning shops for an average of 13.9 years, were exposed to a TWA of 10 ppm tetrachloroethylene (68 mg/m^3) (based on the urinary metabolite excretion; 7.8 mg trichloroacetic acid/g creatinine). Air sampling data were not available. There were two control groups (80 and 81 unexposed individuals) which were not matched (by age or gender) to the exposed group. Significantly altered renal function parameters (increased β-glucuronidase and lysozymuria) were reported, however, these changes were considered mild effects by the investigators. No other organs or parameters were examined. The study was limited because a range of the estimated exposure concentrations was not reported and the investigators did not demonstrate a correlation between exposure concentrations with the renal parameters. The small sample size and lack of matched controls were also limitations of the study. A LOAEL of 10 ppm ($LOAEL_{HEC}$ = 16 mg/m^3, duration adjusted for 8 hours/day, 5 days/week) was determined for renal effects.

Cai et al. (1991) evaluated the subjective symptoms, hematology, and serum biochemistry in 56 tetrachloroethylene-exposed workers (29 men, 27 women) (average age of 35 years) from 3 dry-cleaning workshops who were employed for an average of 36.3 months (1–120 months). There were a few individuals who had TWA of nearly 100 ppm, but most were exposed to lower concentrations, with a TWA of 20 ppm (136 mg/m^3) (3.8–94.4 ppm). The control group consisted of workers from the same factories with no solvent exposures. The subjects were interviewed regarding symptoms that occurred during work and symptoms that have occurred in the past 3-month period. The exposed workers reported significant increases in the prevalence of subjective symptoms, including nasal irritation, joint pain, rough skin, and CNS related effects (dizziness, forgetfulness, fainting, loss in hearing capacity, floating sensation, drunken feeling, heavy feeling in head). However, the reported symptoms may have occurred only on a single day when tetrachloroethylene concentrations were extremely high or be unrelated to tetrachloroethylene exposure and furthermore, the results from the interview may be confounded by the potential for recall bias. The investigators did not attempt to correlate the exposure concentrations with the subjective symptoms, even though the range of TWA values varied greatly in exposed individuals. The subjects were also examined for alterations in hematology (hemoglobin, white cell count) and liver and kidney function indicators (ASAT, ALAT, γ-GTP, ALP, LAR, total bilirubin, BUN, creatinine), but no differences were found between the control and exposed groups. A LOAEL of 100 ppm ($LOAEL_{HEC}$ = 32 mg/m^3, duration adjusted for 8 hours/day, 5 days/week) was determined for the study based on subjective CNS symptoms.

Ferroni et al. (1992) performed neurobehavioral tests on 60 female dry-cleaning shop workers (average age of 39.7 years) exposed to tetrachloroethylene for 10.1 years. The exposure concentration was 15 ppm (102 mg/m^3) (range of 1–67 ppm); air samples were collected during various 4-hour periods over a working week. The control group of 30, recruited in an industrial cleaning plant where solvents were never used, was matched to the exposed group for age, gender, and vocabulary test score. The study demonstrated that the reaction times were prolonged in all tests for the exposed group compared to the controls. It was not indicated, however, when tests were performed (i.e., before or after a work shift). Also, the exposed workers exhibited significantly higher basal levels of serum prolactin compared to their matched controls during the proliferative phase of the menstrual cycle, suggesting that tetrachloroethylene may cause neuroendocrine effects. The investigators reported that there was a lack of correlation between the test scores and the exposure concentrations or the duration of exposure, and furthermore, no correlation was seen between the increased prolactin levels and the exposure variables. The study was also limited by the small sample size and inadequate air sampling (i.e., not representative of the duration of the exposure period). The LOAEL for the study was 15 ppm ($LOAEL_{HEC}$ = 24 mg/m^3, duration adjusted for 8 hours/day, 5 days/week) based on neurological effects.

Seeber et al. (1989) conducted a battery of psychological tests and questionnaires on dry-cleaning shop workers (12 males, 89 females) (average age of 38 years) exposed to 205 mg/m^3 tetrachloroethylene vapors for chronic durations (appeared to be a duration of 127–141 months, although this was not clearly reported in the study). The control group was matched by age, gender, intellectual level, and daily alcohol consumption. A significant impairment ($p<0.01$) in perceptual function, attention, and intellectual function was found in the exposed group compared to controls. The exposed group was also separated into two subgroups, individuals with low exposures (TWA of 83.4 ppm) and those with high exposures (TWA of 363 ppm). However, no significant differences were seen in the psychological tests between the low- and high-exposed groups, indicating a lack of a concentration-response trend. The study was also limited by a failure to correlate the measured parameters with individual exposure concentrations. A LOAEL of 205 mg/m^3 ($LOAEL_{HEC}$ = 49 mg/m^3) was determined for the study based on impaired neurobehavioral functions.

Chronic inhalation studies. In an NTP (1986) study, Fischer 344 rats (50/sex/group) were exposed to 0, 200, and 400 ppm (1356 and 2713 mg/m^3) tetrachloroethylene, 6 hours/day, 5 days/week, for 103 weeks (duration adjusted to 242 and 484 mg/m^3). The concentration selections were based on a 13-week study (discussed below under Subchronic inhalation studies section). Survival was reduced in male rats (control, 46%; 200 ppm, 40%; 400 ppm, 24%), most likely due to the increased incidence of mononuclear cell leukemia. Body weights were comparable among groups. A high incidence of tubular karyomegaly (nuclear enlargement) was exhibited in the exposed groups; 2%, 76%, and 94% incidence in the control, 200-, and 400-ppm males, respectively, and 0%, 16%, and 40% in the females, respectively. Proliferative lesions (tubular cell hyperplasia and adenocarcinomas) were also seen in the kidneys of male rats. No hepatic effects were evident after the chronic exposure. Therefore, a LOAEL of 200 ppm ($LOAEL_{HEC}$[1] = 242 mg/m^3) was determined for the study based on renal karyomegaly.

For the animal inhalation studies, $L(N)OAEL_{HEC}$ = L(N)OAEL[duration adjusted] x (animal blood:air partition coefficient)/(human blood:air coefficient). The HEC was calculated assuming periodicity was attained.

NTP (1986) also exposed groups of B6C3F1 mice (50/sex/group) to 0, 100, or 200 ppm (678 and 1356 mg/m^3) tetrachloroethylene, 6 hours/day, 5 days/week, for 103 weeks (duration adjusted to 121 and 242 mg/m^3). Survival was slightly higher in males (control, 92%; 100 ppm, 50%; 200 ppm, 64%) than in females (72%, 62%, and 38%, respectively). Body weights were comparable among the groups. The incidence of renal tubular karyomegaly was higher in exposed groups for both sexes compared to controls (8%, 35%, and 92% in males; 0%, 33%, 76% in females). The incidence of nephrosis was greater in exposed females compared to controls (10%, 29%, 50%), but comparable for the male groups. Hepatocellular degeneration (4%, 16%, 28% in males; 2%, 4%, 26% in females) and necrosis (2%, 12%, 30% in males; 6%, 10%, 18% in females) were presented in the liver of both sexes. In addition, nuclear inclusions were observed in males (4%, 10%, 18%). Hepatocellular degeneration was characterized by cytoplasmic vacuolation, hepatocellular necrosis, inflammatory cell infiltrates, increased pigmentation in cells, oval cell hyperplasia, and regenerative foci. Hepatocellular adenoma and carcinoma were also seen in exposed mice. Acute passive congestion in the lungs was also found in the males (2%, 16%, 20%). A LOAEL of 100 ppm ($LOAEL_{HEC}$ = 121 mg/m^3) was determined for the study based on renal and hepatic effects.

In a study by Briving et al. (1986), four-month-old Mongolian gerbils (3/sex/group) were exposed to 0 or 120 ppm (814 mg/m^3) tetrachloroethylene continuously for 12 months. Body weight and brain weights were not significantly different between the two groups. Levels of taurine were significantly decreased in the hippocampus and in the posterior part of the cerebellar vermis, while levels of glutamine were significantly elevated in the hippocampus after tetrachloroethylene exposure. No other amino acids or glutathione were altered in the brain. The small number of animals in each group and the insufficient exposure levels limit the study results. The LOAEL for the study was 120 ppm ($LOAEL_{HEC}$ = 814 mg/m^3) based on neurological effects.

Kyrklund et al. (1984) exposed male Mongolian gerbils (6/group) to 0 or 120 ppm (814 mg/m^3) tetrachloroethylene continuously for 12 months. Exposure resulted in an alteration in the fatty acid composition of the phospholipid, phosphatidylethanolamine, in the cerebral cortex and hippocampus. Body and brain weights did not differ between control and exposed animals. However, the study did not examine the brain histologically, a small number of animals was used for each group, and only one concentration level was tested. The LOAEL for this study was 120 ppm ($LOAEL_{HEC}$ = 814 mg/m^3) based on neurological effects.

Subchronic inhalation studies. Subchronic inhalation studies have been conducted in rats, mice, and gerbils, with the major effects observed in the kidney, liver, and CNS. The exposure duration for many of the studies were for less than a month.

In a 13-week subchronic study by NTP (1986), Fischer 344 rats (10/sex/group) inhaled 0, 100, 200, 400, 800, and 1600 ppm (678, 1356, 2713, 5426, and 10851 mg/m^3) tetrachloroethylene, 6 hours/day, 5 days/week (duration adjusted to 121, 242, 484, 969, and 1938 mg/m^3). Death occurred in 40% of the males and 70% of the females in the 1600-ppm group. The high-concentration group also had final mean body weights which were 20% lower than that of controls for males and 11% lower for females. A concentration-related increase in the incidence of hepatic congestion was exhibited in the rats (males: control, 10%; 200 ppm, 20%; 400 ppm, 30%; 800 ppm, 50%; 1600 ppm, 70%; females: 0%, 10%, 50%, 50%, 89%, respectively); severity was also

concentration related. However, the 100-ppm group was not examined for this effect. Lung congestion developed in 70% of the animals exposed to 1600 ppm, but was not demonstrated in the animals of the other groups. A NOAEL of 200 ppm ($NOAEL_{HEC}$ = 242 mg/m^3) and a LOAEL of 400 ppm ($LOAEL_{HEC}$ = 484 mg/m^3) was determined for the study based on the hepatic effect.

NTP (1986) also exposed B6C3F1 mice (10/sex/group) to the same concentrations and subchronic duration as the rats. Death occurred only in the 1600-ppm group; 20% of the males and 40% of the females. Clinical signs in the exposed mice included a hunched appearance and lack of movement at 400 ppm, panting and irritation at 800 ppm, and uncoordination and unconsciousness at 1600 ppm. Liver lesions (leukocytic infiltration, centrilobular necrosis, and bile stasis) developed in 80% of the males and 50% of the females in the 400-ppm group and in nearly 100% of the animals in the 800- and 1600-ppm groups, with effects reaching only mild severity at 1600 ppm. Mitotic alterations in the liver (not concentration related) were exhibited in the males (control, 0%; 200 ppm, 30%; 400 ppm, 50%; 800 ppm; 50%; 1600 ppm, 10%), however, the 100-ppm group were not examined. None of the females demonstrated mitotic alterations in the liver. Karyomegaly of tubular epithelial cells in the kidneys was seen in 60% of the males and 80% of the females in the 200-ppm group, and in almost 100% of the animals in the 400-, 800-, and 1600-ppm groups, with the effect reaching only mild severity at 1600 ppm. The effect was not evaluated in the 100-ppm group. The LOAEL was 200 ppm ($LOAEL_{HEC}$ = 242 mg/m^3) based on renal and hepatic effects. A NOAEL could not be determined for the study.

In a 30-day study by Kjellstrand et al. (1984), NMRI mice (10/sex/group) were exposed continuously to 0, 9, 37, 75, 150 ppm (61, 251, 509, 1017 mg/m^3) tetrachloroethylene. Final body weights were significantly decreased in the males exposed to 75 and 150 ppm. The absolute liver weights were significantly increased (not concentration related) for all exposed groups, with abnormal gross pathological appearance (enlarged and yellowish colored liver, vacuolization of hepatocytes) reported to be detectable at 9 ppm and most apparent at 150 ppm (more pronounced in males than in females). However, incidence and severity data were not presented on the histological effects to determine if histological changes were concentration related. The investigators noted that the morphology alterations disappeared after a 5–120-day recovery period following exposure to 150 ppm. In addition, a significant increase in plasma butylcholinesterase activity occurred with exposure to 37 ppm and higher. Kidney and spleen weights were not affected by tetrachloroethylene exposure. However, histopathology was not performed on these organs. The LOAEL for the study was 9 ppm ($LOAEL_{HEC}$ = 61 mg/m^3) for hepatic effects (increased liver weight, gross pathology).

Kylin et al. (1965) reported on female mice (strain not identified) (20/group) inhaling 200 ppm (1356 mg/m^3) tetrachloroethylene vapor, 4 hours/day, 6 days/week, for 1, 2, 4 or 8 weeks (duration adjusted to 194 mg/m^3). Only the liver and kidneys were examined. Compared to the controls, an increased incidence of fatty degeneration in the liver was observed in exposed animals, with greater severity at the longer exposure periods. A two-fold increase in liver fat was also reported in exposed group. Kidney histopathology revealed no differences between the control and exposed groups. The LOAEL for the study was 200 ppm ($LOAEL_{HEC}$ = 194 mg/m^3) based on hepatic effects.

Odum et al. (1988) exposed groups of Fischer 344 rats (5/sex/group) to 0, 200 or 400 ppm (1356 and 2713 mg/m^3) tetrachloroethylene, 6 hours/day, for 28 consecutive days (duration adjusted

to 333 and 678 mg/m^3). The relative liver weights were not affected in the exposed rats. A slight, but significant, increase in hepatic palmitoyl CoA oxidase activity (marker for peroxisomal β-oxidation) was seen in the males and females of the two exposed groups. Centrilobular hepatocellular hypertrophy (consisting of proliferation of smooth endoplasmic reticulum), with a concomitant loss of glycogen, was exhibited in the males of both exposed groups and the females of the 400-ppm group. The LOAEL for the study was 200 ppm ($LOAEL_{HEC}$ = 333 mg/m^3) based on hepatic effects.

Odum et al. (1988) also examined B6C3F1 mice exposed to the same concentrations and duration as the rats. The relative liver weights were statistically elevated in exposed mice compared to controls. Palmitoyl CoA oxidase activity in the liver was significantly increased (effect more pronounced in mice than in rats). Histopathology revealed lipid accumulation in centrilobular hepatocytes for the 200- and 400-ppm groups, as well as centrilobular eosinophilia and centrilobular fatty vacuolation in the 400-ppm group; the effects were similar in males and females. A significant increase in the volume of cytoplasm with peroxisomes was seen in the hepatocytes of both exposed groups. A LOAEL of 200 ppm ($LOAEL_{HEC}$ = 333 mg/m^3) was determined for hepatic effects.

Green et al. (1990) exposed groups of Fischer 344 rats and B6C3F1 mice (5/group) to 400 ppm (2713 mg/m^3) tetrachloroethylene, 6 hours/day, for 28 consecutive days (duration adjusted to 678 mg/m^3). Kidney weight and histopathology were evaluated and were found to be comparable for the control and exposed groups. No other organs were examined. Therefore, a free-standing NOAEL of 400 ppm ($NOAEL_{HEC}$ = 678 mg/m^3) was determined based on kidney effects.

Soni et al. (1990) exposed groups of male Sprague-Dawley rats to 0, 50, 200, or 600 ppm (339, 1356, and 4069 mg/m^3) tetrachloroethylene continuously for 4 weeks. The erythrocyte δ-aminolevulinate dehydratase activity was significantly reduced in the 200- and 600-ppm groups, while serum transaminase activity was significantly increased in the 600-ppm group. Hepatic microsomal protein and cytochrome b_5 were significantly elevated for all exposed groups and NADPH cytochrome c reductase was increased in the 600-ppm group compared to controls. Cytochrome P_{450} in the kidneys was significantly increased for all exposed groups, although levels decreased at 600 ppm compared to those at 50 and 200 ppm. The investigators concluded that tetrachloroethylene alters mixed function oxidase and heme metabolism. The LOAEL for the study was 50 ppm ($LOAEL_{HEC}$ = 339 mg/m^3) based on hepatic effects.

In an older study by Carpenter (1937), male and female rats (24/group) (strain not specified) were exposed to 70, 230, or 470 ppm (475, 1560, or 3188 mg/m^3) tetrachloroethylene, 8 hours/day, 5 days/week, for 7 months (duration adjusted to 113, 371, 759 mg/m^3). The rats in the 230- and 470-ppm groups developed mild and nonspecific liver changes (including decreased hepatic glycogen). The control group consisted of 18 unexposed rats. The authors reported congested kidneys with granular swelling at 230 ppm. At 470 ppm, an increase in renal secretion, with cloudy swelling and desquamation in the kidneys, was reported in rats immediately after cessation of exposure and at the end of a 46-day recovery period. Splenic congestion and increased hemosiderin deposits were also exhibited in the rats exposed to 230 or 470 ppm. Study limitations include the use of sick animals (parasites, pneumonia), nonstandard study protocols, rats of undefined strain, and inadequate controls. A NOAEL of 70 ppm ($NOAEL_{HEC}$ = 113 mg/m^3) and LOAEL of 230 ppm ($LOAEL_{HEC}$ = 371 mg/m^3) were determined for the study based on hepatic, renal, and splenic effects.

There were several inhalation studies that assessed the neurological effects of tetrachloroethylene exposure in gerbils and rats. Karlsson et al. (1987) described the effect of tetrachloroethylene on DNA concentrations in brains of Mongolian gerbils (4/sex/group) exposed to 0 or 60 ppm (407 mg/m^3) tetrachloroethylene continuously for 3 months, followed by a 4-month recovery period. The DNA concentration per wet weight in the frontal cerebral cortex was significantly decreased in the exposed animals compared to controls. Body and brain weights and total protein concentrations for different brain areas were not affected by tetrachloroethylene exposure. The LOAEL of the study was 60 ppm ($LOAEL_{HEC}$ = 407 mg/m^3) based on neurological effect.

In a study by Rosengren et al. (1986), Mongolian gerbils (4/sex/group) were exposed continuously to 0, 60, and 320 ppm (407 and 2170 mg/m^3) for 3 months, followed by a 4-month recovery period. Eight sex-matched littermates served as controls. Body and brain weights were not significantly altered after tetrachloroethylene exposure, although the wet weights of the frontal cerebral cortex were significantly decreased. The brain was the only organ examined. The S-100 concentrations per wet weight (indicative of astroglial hypertrophy and/or proliferation) were significantly elevated in the occipital cerebral cortex, hippocampus, anterior cerebellar hemispheres, and posterior cerebellar vermis and decreased in the frontal cerebral cortex in the high-concentration group, but the S-100 concentrations were unaffected in the low-concentration group. The exposed groups showed a decrease in DNA concentration in the frontal cerebral cortex, as was seen by Karlsson et al. (1987). The study was limited because other target organs were not examined. A LOAEL of 60 ppm ($LOAEL_{HEC}$ = 407 mg/m^3) was determined for the study based on neurological changes.

In a similar experiment as the chronic gerbil study by Kyrklund et al. (1984), Kyrklund et al. (1987) exposed Mongolian gerbils (6/group; sex not reported) continuously to 0 or 320 ppm (2170 mg/m^3) for 3 months, resulting in altered fatty acid pattern of the cerebral cortex and hippocampus. Brain weight was slightly decreased (statistically significant) compared to controls, but body weight was not affected. A LOAEL of 320 ppm ($LOAEL_{HEC}$ = 2170 mg/m^3) was determined for neurological effect.

Kyrklund et al. (1990) exposed male Sprague-Dawley rats (8/group) to 0 or 320 ppm (2170 mg/m^3) tetrachloroethylene vapors continuously for 90 days. There was a significant increase in the relative liver weights after exposure, but there was no effect on brain weights. Extraction of lipids from the cerebral cortex revealed no changes in the lipid class concentration following tetrachloroethylene exposure. However, alterations in the fatty acid pattern of ethanolamine phosphoglyceride were seen. Following exposure to 320 ppm for a shorter duration of 30 days (Kyrklund et al. 1988), male Sprague-Dalwey rats (number of animals not reported) had a significant increase in the relative liver and brain weights compared to controls. The cerebral cortex of the brain was analyzed, and the total cerebral phospholipids and cholesterol were slightly decreased and the fatty acid pattern of ethanolamine phosphoglyceride was altered. The LOAEL for both studies was 320 ppm ($LOAEL_{HEC}$ = 2170 mg/m^3) based on neurological changes. Therefore, the Kyrklund et al. studies show that tetrachloroethylene exposure may cause alterations in lipid composition and fatty

acid pattern in the cerebral cortex of rats and gerbils. However, the investigators noted that the changes were different for the rats and gerbils, which may be due to species differences in the fatty acid metabolism.

Wang et al. (1993) described the effects of tetrachloroethylene on cytoskeletal elements of glial and neuronal cells in male Sprague-Dawley rats (8/group) inhaling 300 ppm (2035 mg/m^3) for 4 weeks or 600 ppm (4069 mg/m^3) for 4 or 12 week continuously. A control group was included for each exposed group. Body weight gain, as well as brain weight, was significantly reduced in the animals exposed to 600 ppm. Of the three brain areas examined, changes were seen in the frontal cerebral cortex and brain stem (significant decrease in brain region weight, total DNA content, and total protein) after exposure to 600 ppm, while no effects were seen in the hippocampus. There was also decreases in glial fibrillary acid protein and S-100 protein in the frontal cerebral cortex, brain stem, and hippocampus, which the investigators concluded may indicate a reduction in the number of glial cells in these brain regions. A NOAEL of 300 ppm ($NOAEL_{HEC}$ = 2035 mg/m^3) and a LOAEL of 600 ppm ($LOAEL_{HEC}$ = 4069 mg/m^3) were determined for the study based on neurological effects.

Reproductive/Developmental Studies. Schwetz et al. (1975) exposed groups of pregnant Sprague-Dawley rats (18) and Swiss Webster mice (12) to 300 ppm (2035 mg/m^3) tetrachloroethylene, 7 hours/day, on gestational days 6–15. A slight, but significant, increase in the incidence of resorptions was seen in the rat. There were no increased incidences of gross or skeletal anomalies in the exposed rat fetuses compared to controls. In mice, fetal body weights were significantly decreased. Increases in soft tissue alteration (subcutaneous edema) and skeletal anomalies (delayed ossification of skull bones, unfused centers of ossification) were seen in litters of exposed mice. A LOAEL of 300 ppm ($LOAEL_{HEC}$[2] = 2035 mg/m^3) was determined for development effects in mice (decreased fetal weight; delayed ossification) and for reproductive effects in rats (increased fetal resorptions).

In a behavioral developmental study by Nelson et al. (1980), pregnant Sprague-Dawley rats (19 or 21/group) were exposed to 100 ppm (678 mg/m^3) tetrachloroethylene on days 14–20 of gestation or 900 ppm tetrachloroethylene (6104 mg/m^3) on days 7–13 or days 14–20 of gestation. Three control groups, for each exposure group, were also included. Seven behavioral tests were performed on litters during the ages of 4–46 days to measure various CNS functions at different stages of development. No neurobehavioral effects were exhibited by the offspring exposed to 100 ppm *in utero*. A significant decrease in brain acetylcholine levels was found in 21-day-old offspring exposed to 900 ppm for either gestational period, as well as a decrease in dopamine levels in offspring exposed during gestational days 7–13. In this latter group, there was decreased performance during tests of neuromuscular ability (ascent on a wire mesh screen and rotorod balancing) on certain days. Offspring (before weaning) from dams exposed to 900 ppm on days 14–20 performed poorly on the ascent test on test day 14 only, but later in development, their performance in the rotorod balancing test was superior to controls, and they were more active in an open-field test. No microscopic brain lesions were seen. The NOAEL was 100 ppm ($NOAEL_{HEC}$

Exposures during the gestation period are not duration adjusted.

= 678 mg/m^3) and the LOAEL was 900 ppm ($LOAEL_{HEC}$ = 6104 mg/m^3) for the study based on developmental effects.

Carpenter (1937) reported that no fertility effects were apparent in rats exposed to 230 and 470 ppm (1560 and 3188 mg/m^3) tetrachloroethylene, 8 hours/day, 5 days/week, for 7 months. Therefore, a NOAEL of 470 ppm ($NOAEL_{HEC}$ = 3188 mg/m^3) for reproductive effects was determined. Study limitations include use of sick animals (parasites, pneumonia), nonstandard protocols, heterogenous rats, and inadequate controls. Hardin et al. (1981) reported that no maternal or fetal toxicity or teratogenicity was found in rats exposed to 500 ppm (3391 mg/m^3) tetrachloroethylene on days 1–19 and rabbits on days 1–24 of gestation. Therefore, a NOAEL of 500 ppm ($NOAEL_{HEC}$ = 3391 mg/m^3) was given for this study based on developmental and reproductive effects.

In the last few years, investigators have attempted to evaluate the effects of tetrachloroethylene on reproductive outcomes in humans. Most study findings suggest potential reproductive effects, however, they were limited due to lack of exposure concentration data and potential biases in data collection.

Eskenazi et al. (1991a) determined the effects of tetrachloroethylene exposure on human semen quality. In a comparison of 34 dry cleaners with 48 laundry workers, the overall percentage of abnormal sperm was similar for the two groups and was, by standard clinical measurements, within normal limits. However, sperm of dry cleaners were significantly more likely to be round and less likely to be narrow than the sperm of laundry workers; these effects corresponded to expired air levels in the workers. In another study, Eskenazi et al. (1991b) reported that the number of pregnancies and live births and the rates of spontaneous abortion were similar for 17 wives of dry-cleaning workers compared to 32 wives of laundry workers during a 2-year period. However, the wives of the dry cleaners were more than twice as likely to have a history of attempting to become pregnant for more than 12 months or to have sought care for an infertility problem than the wives of laundry workers. Definitive conclusions could not be made based on the study results due to the inadequate sample size.

Kyyronen et al. (1989) determined the effects of tetrachloroethylene exposure during the first trimester of pregnancy. A significant association was found between exposure and spontaneous abortions. However, potential sources of bias in the study may have occurred, including the following: (1) women who had suffered spontaneous abortion were less eager to participant in the study and (2) selective self reporting in the collected questionnaires.

In a cohort study by Olsen et al. (1990), reproductive outcomes were evaluated in women employed in the dry-cleaning work in Sweden, Denmark, and Finland. A significant increase in spontaneous abortions was found in the Finnish data, but no effects were seen on birth weight and congenital malformations. Again, bias may have influenced the findings. For example, women interviewed may have exaggerated their responses regarding exposure during pregnancy and thus provided inaccurate information.

DERIVATION OF PROVISIONAL CHRONIC RfC

Based on the available information in humans and animals, the critical effects of tetrachloroethylene exposure was determined to be hepatotoxicity and renal toxicity. The RfC is derived from the chronic mouse study by NTP (1986), supported by numerous animal studies showing the same critical endpoints. Neurological effects were also reported in animals, however, these effects generally occurred at higher concentrations than the hepatic and renal effects. Human data support the findings that the renal effect is a critical endpoint, and also demonstrate that neurological effects may develop with chronic exposures to low tetrachloroethylene concentrations. There was a strong preference to derive the RfC based on the human occupational data, with either neurological effects or renal toxicity as the critical effect, because the exposure concentrations from the human studies were lower than those identified in the animal studies. An RfC based on one of the human studies would have resulted in an inhalation value that would be similar to the RfC value that has been derived from the NTP (1986) mouse study. However, the chronic RfC was not determined from the human occupational data because these studies failed to establish a definitive threshold for tetrachloroethylene toxicity and the quality of the data was considered unreliable due to several limitations. These studies had small sample sizes, based exposure concentrations on monitoring data that did not represent the entire exposure period, and most lacked correlations between the estimates of exposure (duration or intensity) and the measured parameter(s).

The 103-week mouse study by NTP (1986) was chosen as the principal study because it demonstrated significant hepatotoxicity (degeneration and necrosis) and renal toxicity (karyomegaly). The NTP (1986) study was a well-conducted chronic study, with both critical effects being concentration related and all the target organs (liver, kidney, brain) being examined. Supporting data were found in human occupational studies and other animal studies which also identified critical effects occurring in the kidneys and liver at similar concentrations. Several occupational studies reported renal and neurological effects at lower concentrations than that reported in the critical study, however, none of the human studies were not chosen as the principal study due to limitations in the data (discussed above). A 30-day study by Kjellstrand et al. (1984) reported histological changes in the liver of mice at low tetrachloroethylene concentrations, however, the incidence and severity data were not presented in the study, therefore, it could not be determined if the effect was concentration related.

A provisional chronic RfC of 0.4 mg/m^3 tetrachloroethylene was determined based on the LOAEL of 100 ppm from the chronic mouse NTP (1986) study. Derivation of a provisional RfC value is as follows:

a. LOAEL of 100 ppm (678 mg/m^3) is adjusted for intermediate exposure:

$$LOAEL_{ADJ} = 678 \text{ mg/m}^3 \times 6 \text{ hrs}/24 \text{ hrs} \times 5 \text{ days}/7 \text{ days} = 121 \text{ mg/m}^3$$

b. Derivation of the $LOAEL_{HEC}$:

$$LOAEL_{HEC} = LOAEL_{ADJ} \times L_A/L_H$$

where: L_A = animal blood:air partition coefficient and

L_H = human blood:air coefficient.

The HEC was calculated assuming periodicity was attained. Gargas et al. (1989) reported blood:air partition coefficients for tetrachloroethylene: humans (10.3), Fischer 344 rats (18.9), and B6C3F1 mice (17.5). Since the animal blood:air partition coefficients are greater than human value, the default value of 1.0 is used. Since no values were found for the other species and strains, the default value of 1.0 is used.

Therefore,

$LOAEL_{HEC}$ = 121 mg/m^3 x 1 (default value for animal:human blood:air partition coefficient ratio)

= 121 mg/m^3

RfC = $LOAEL_{HEC}$ / UF = 121 mg/m^3 / 300

where a total uncertainty factor of 300 was applied to account for interspecies (3) and intraspecies (10) variability and the use of a LOAEL (10).

= 0.4 mg/m^3

Confidence in the principal study is medium-high. The NTP study was well-conducted and was given medium-high confidence because the most sensitive animal species was identified, the critical effects were concentration related, all target organs were examined, and the appropriate number of animals and statistical analyses were used, although a NOAEL was not established for the study. Furthermore, the critical effects identified in the study were supported by several other inhalation studies (including occupational studies), as well as the oral RfD for tetrachloroethylene, which was based on hepatotoxicity in mice.

Confidence in the database is medium. There is a large amount of data on inhalation exposure to tetrachloroethylene in humans and animals. Chronic human data come from studies conducted on exposed workers in dry-cleaning shops (Cai et al., 1991; Ferroni et al., 1992; Franchini et al., 1983; Lauwerys et al., 1983; Mutti et al., 1992; Seeber, 1989; Vyskocil et al., 1990). However, these occupational studies have several limitations (inadequate monitoring data, lack of concentration-response trends, small sampling size). Therefore, there is a need for well-conducted chronic occupational studies to establish the thresholds for CNS and renal effects in humans. Case reports of acute human exposures to tetrachloroethylene are also available as supporting evidence (Carpenter, 1937; Freed and Kandel, 1988; Hake and Stewart, 1977; Rowe et al., 1952; Coler and Rossmiller, 1953; Stewart et al., 1970). There are numerous animal studies for chronic and subchronic durations that describe the effects of tetrachloroethylene exposure in rats, mice, and gerbils. The primary target organs found in these studies were the liver, kidney, and CNS. Hepatic and renal effects were assessed in rats and mice (Carpenter, 1937; Green et al., 1990; Kjellstrand et al., 1984; NTP, 1986;

Odum et al., 1988; Soni et al., 1990) and neurological changes were evaluated in rats and gerbils (Briving et al., 1986; Karlsson et al., 1987; Kyrklund et al., 1984, 1987, 1988, 1990; Rosengren et al., 1986; Wang et al., 1993). Also, several investigators (Eskenazi et al., 1991a, 1991b; Kyyronen et al., 1989; Olsen et al., 1990) have assessed the effect of tetrachloroethylene on the reproductive outcomes of exposed dry-cleaning workers and/or their wives, however, these studies lack exposure information and study bias may have occurred. The database contains some information on the developmental and reproductive toxicity of tetrachloroethylene in mice and rats (Carpenter, 1937; Hardin et al., 1981; Nelson et al., 1980; Schwetz et al., 1975), but a multigenerational reproductive study has not yet been performed. Based on the confidence in the database (medium) and in the principal study (medium-high), the overall confidence in the RfD is medium.

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Tetrachloroethylene RfC

Study	Species	Critical Effect(s)	Effect Level	HEC Concentration
CHRONIC HUMAN STUDIES				
(The RfC was not derived from these studies due to several limitations (insufficient sample size, inadequate monitoring data, failure to correlate estimates of exposure (duration and intensity) with measured parameters).				
Lauwerys et al. 1983	Occupational workers	CNS (psychomotor tests) Kidney (biochemical changes)	NOAEL = 20.8 ppm = 141 mg/m³	NOAEL = 34 mg/m³
Mutti et al. 1992	Occupational workers	Kidney (biochemical changes)	LOAEL = 14.8 ppm = 100 mg/m³	LOAEL = 24 mg/m³
Vyskocil et al. 1990	Occupational workers	Kidney (lysozymuria)	LOAEL = 157 mg/m³	LOAEL = 37 mg/m³
Franchini et al. 1983	Occupational workers	Kidney (increased β-glucuronidase; lysozymuria)	LOAEL = 10 ppm (estimated from urinary levels) = 68 mg/m³	LOAEL = 16 mg/m³
Cai et al. 1991	Occupational workers	CNS (subjective symptoms)	LOAEL = 100 ppm = 136 mg/m³	LOAEL = 32 mg/m³
Ferroni et al. 1992	Occupational workers	CNS (prolonged reaction time)	LOAEL = 15 ppm = 102 mg/m³	LOAEL = 24 mg/m³
Seeber 1989	Occupational workers	CNS (impaired perceptual function, attention, and intellectual function)	LOAEL = 205 mg/m³	LOAEL = 49 mg/m³

Study	Species	Critical Effect(s)	Effect Level	HEC Concentration
CHRONIC ANIMAL STUDIES				
NTP 1986 103-week study	Fischer 344 rats	Kidney (karyomegaly)	LOAEL = 200 ppm = 1356 mg/m³	LOAEL = 242 mg/m³
	B6C3F1 mice	Kidney (karyomegaly) Liver (degeneration and necrosis)	LOAEL = 100 ppm = 678 mg/m³	*LOAEL = 121 mg/m³
Briving et al. 1986 12-month study	Mongolian gerbils	CNS (decreased taurine in hippocampus and posterior part of cerebellar vermis; increased glutamine in hippocampus)	LOAEL = 120 ppm = 814 mg/m³	LOAEL = 814 mg/m³
Kyrklund et al. 1984 12-month study	Mongolian gerbils (males)	CNS (altered fatty acid composition of phosphatidylethanolamine in cerebral cortex and hippocampus)	LOEL = 120 ppm = 814 mg/m³	LOAEL = 814 mg/m³
SUBCHRONIC ANIMAL STUDIES				
NTP 1986 13-week study	Fischer 344 rats	Liver (congestion)	NOAEL = 200 ppm = 1356 mg/m³ LOAEL = 400 ppm = 2713 mg/m³	NOAEL = 242 mg/m³ LOAEL = 484 mg/m³
	B6C3F1 mice	Liver (mitotic alterations) Kidney (karyomegaly)	LOAEL = 200 ppm = 1356 mg/m³	LOAEL = 242 mg/m³
Kjellstrand et al. 1984 30-day study	NMRI mice	Liver (increased absolute weight, enlarged and yellowish color appearance)	LOAEL = 9 ppm = 61 mg/m³	LOAEL = 61 mg/m³

Perchloroethylene RfC (Continued)

Study	Species	Critical Effect(s)	Effect Level	HEC Concentration
Kylin et al. 1965 1–8-week study	female mice (strain not specified)	Liver (fatty degeneration)	LOAEL = 200 ppm = 1356 mg/m³	LOAEL = 194 mg/m³
Odum et al. 1988 14–28-day study	Fischer 344 rats	Liver (centrilobular hypertrophy; increased palmitoyl CoA oxidase activity)	LOAEL = 200 ppm = 300 mg/m³	LOAEL = 300 mg/m³
	B6C3F1 mice	Liver (lipid accumulation; peroxisomal proliferation; increased relative organ weight)	LOAEL = 200 ppm = 300 mg/m³	LOAEL = 300 mg/m³
Green et al. 1990 28-day study	Fischer 344 rats	Kidney (no effects)	NOAEL = 400 ppm = 2713 mg/m³	NOAEL = 678 mg/m³
	B6C3F1 mice	Kidney (no effects)	NOAEL = 400 ppm = 2713 mg/m³	NOAEL = 678 mg/m³
Soni et al. 1990 4-week study	Sprague-Dawley rats (males)	Liver (biochemical alterations)	LOAEL = 50 ppm = 339 mg/m³	LOAEL = 339 mg/m³
Carpenter 1937 7-month study	rats (strain not specified)	Liver (decreased glycogen) Kidney (secretion; cloudy swelling; desquamation) Spleen (congestion; increased hemosiderin deposits)	NOAEL = 70 ppm = 475 mg/m³ LOAEL = 230 ppm = 1560 mg/m³	NOAEL = 113 mg/m³ LOAEL = 371 mg/m³
Karlsson et al. 1987 3-month study	Mongolian gerbils	CNS (decreased DNA in frontal cerebral cortex)	LOAEL = 60 ppm = 407 mg/m³	LOAEL = 407 mg/m³
Rosengren et al. 1986 3-month study	Mongolian gerbils	CNS (decreased DNA in frontal cerebral cortex)	LOAEL = 60 ppm = 407 mg/m³	LOAEL = 407 mg/m³
Kyrklund et al. 1987 3-month study	Mongolian gerbils (sex not reported)	CNS (altered fatty acid pattern in cerebral cortex and hippocampus)	LOAEL = 320 ppm = 217 mg/m³	LOAEL = 2170 mg/m³

Perchloroethylene RfC (Continued)

Study	Species	Critical Effect(s)	Effect Level	HEC Concentration
Kyrklund et al. 1990 90-day study	Sprague-Dawley rats (males)	CNS (altered fatty acid pattern in cerebral cortex)	LOAEL = 320 ppm = 2170 mg/m³	LOAEL = 2170 mg/m³
Kyrklund et al. 1988 30-day study	Sprague-Dawley rats (males)	CNS (altered fatty acid pattern in cerebral cortex)	LOAEL = 320 ppm = 2170 mg/m³	LOAEL = 2170 mg/m³
Wang et al. 1993 12-week study	Sprague-Dawley rats (males)	CNS (decreased brain weight, decreased DNA and protein in cerebral cortex and brain stem; decreased glial fibrillary acid protein and S-100 protein)	LOAEL = 600 ppm = 4069 mg/m³	LOAEL = 4069 mg/m³
REPRODUCTIVE/DEVELOPMENTAL STUDIES				
Schwetz et al. 1985 reproductive study	Sprague-Dawley rats	Reproductive (increased fetal resorption)	LOAEL = 300 ppm = 2035 mg/m³	LOAEL = 2035 mg/m³
	Swiss Webster mice	Developmental (decreased fetal body weight; delayed ossification)	LOAEL = 300 ppm = 2035 mg/m³	LOAEL = 2035 mg/m³
Nelson et al. 1980 neurodevelopmental study	Sprague-Dawley rats	Developmental (behavioral and neurochemical changes)	NOAEL = 100 ppm = 678 mg/m³ LOAEL = 900 ppm = 6104 mg/m³	NOAEL = 678 mg/m³ LOAEL = 6104 mg/m³

The critical effect.

Risk Assessment Issue Paper for:
Derivation of a Provisional RfC for
Trimethylbenzene (1,2,4 and 1,3,5)
(CASRN 108-67-8 and 95-63-6)

A TOXLINE literature search (1981-1991, oral RfD and cancer strategies) was conducted in November 1991. In addition, literature searches of TOXLINE (1991-1993, CAS number and chemical name, all cites), MEDLINE (1990-1993), CANCERLINE (mid-70's-1993, all cites), HSDB, RTECS, and TSCATS were performed in May 1993. Documents on the CARA lists (U.S. EPA, 1991, 1994c) are a Health Effects Assessment document (U.S. EPA, 1987a) and a Health Advisory (U.S. EPA, 1987b), which were consulted. Other sources of information that were consulted were IRIS (U.S. EPA, 1995a), the RfD/RfC Monthly Status Report (U.S. EPA, 1995b), the Drinking Water Regulations and Health Advisories list (U.S. EPA, 1994a), the HEAST (U.S. EPA, 1994b), and NTP Status Reports (NTP, 1993a). Trimethylbenzene has not been the subject of an ATSDR toxicological profile.

Summary of Relevant Toxicity Data for Trimethylbenzene

Data regarding the inhalation toxicity of trimethylbenzene are limited to one occupational study (Battig et al., 1958) and several acute and subchronic studies in rodents (Battig et al., 1958; Bernshtein, 1972; Cameron et al., 1938; Lazarew, 1929; Wiglusz et al., 1975a,b). Of all these studies, only Wiglusz et al. (1975a) was available for review; details of the others were obtained from secondary sources. Based on the available data, U.S. EPA (1987a) determined that the data were inadequate for derivation of a subchronic or chronic inhalation RfC.

The U.S. EPA (1987b,c) reported the results of the occupational study of Battig et al. (1958). In that study, an increase in toxic symptoms was found in 27 workers exposed for several years to "Fleet-X-DV-99", as compared to 10 unexposed controls. Fleet-X-DV-99 is a solvent containing 97.5% aromatic hydrocarbons (>30% 1,3,5-trimethylbenzene and >50% 1,2,4-trimethylbenzene) and 2.5% of paraffinic and naphthenic hydrocarbons. Rough quantitation of the exposure levels reported concentrations of hydrocarbons vapor ranging from 10 to 60 ppm (49-295 mg/m^3). Clinical findings in the workers included central nervous system effects (vertigo, headaches, drowsiness), chronic asthma-like bronchitis (classification criteria not reported), hyperchromic anemia (<4.5 million erythrocytes/mm^3) and disturbances in blood clotting.

A 4-hour inhalation LC_{50} of 24 g/m^3 was reported for rats (RTECS, 1993). U.S. EPA (1987b) summarized some acute data in rats and mice. Concentrations of 35-40 mg/L of 1,3,5-trimethylbenzene in the air (34,480-44,332 mg/m^3) caused loss of reflexes and prostration in mice, but the duration of the exposures was not reported (Lazarew, 1929). Rats exposed to 2240 ppm (11,034 mg/m^3) 1,3,5-trimethylbenzene for 24 hours slowly developed narcosis and 4/16 died from respiratory failure (Cameron et al., 1938). Congestion of the lungs was the only remarkable pathology. No adverse effects were observed in rats and mice exposed

to 560 ppm (2758 mg/m^3) for 24 hours or to the same concentration 8 hours/day for 14 days (Cameron et al., 1938). Wiglusz et al. (1975a,b) reported an increase in the proportion of segmented neutrophilic granulocytes, and a decrease in the proportion of lymphocytes in rats exposed to 6.0 mg/L (6000 mg/m^3) 1,3,5-trimethylbenzene for 6 hours. Serum alkaline phosphatase was increase at 3.0 mg/L and no adverse effects were noticed at 1.5 mg/L (1500 mg/m^3).

U.S. EPA (1987a,b,c) reported the results of several subchronic inhalation studies in rats. Battig et al. (1958) exposed rats 5 days/week, 8 hours/day to 1700 ppm (8357 mg/m^3) (8 rats) Fleet-X-DV-99 solvent for 4 months or to 500 ppm (2458 mg/m^3) (unspecified number of rats) for 70 days. Four of the 8 rats exposed to 1700 ppm died within 2 weeks of exposure, while none of the animals in the 500 ppm group died. Histological changes in the animals that died (only these were examined) included cloudy swelling and fatty infiltration in the kidneys, peripheral fatty infiltration in the liver, an increase in secondary nodules in the spleen, and marked congestion of the pulmonary capillaries with alveolar wall thickening. Alterations in differential WBC counts (increase in the percentage of segmented neutrophilic granulocytes and a decrease in the percentage of lymphocytes) were reported at ≥500 ppm. Similar alterations in differential WBC counts as well as a significant elevation of SGOT levels were found in rats (n=6) exposed to 3.0 mg/L (610 ppm) 1,3,5-trimethylbenzene 6 hours/day, 6 days/week for 5 weeks (Wiglusz et al., 1975a,b). No exposure-related effects on hemoglobin levels and erythrocyte or leukocyte counts, or on the activity of SGPT, glutamate dehydrogenase or ornithine carbamyl transferase were found in the Wiglusz et al. (1975a,b) studies. Bernshtein (1972) exposed rats to 1 mg/L (200 ppm) of a mixture of trimethylbenzenes 4 hours/day for 6 months. An inhibition of phagocytic activity of the leukocytes was reported. This study was summarized by Sandmeyer (1981) and further experimental details were not provided.

ACGIH (1986, 1992) has adopted a TLV-TWA of 25 ppm (123 mg/m^3) for trimethylbenzenes based on the occupational study by Battig et al. (1958). OSHA (1992) lists an 8-hour TWA of 25 ppm for trimethylbenzenes to prevent adverse respiratory and blood effects in exposed workers (OSHA, 1989). NIOSH (1992) lists a REL TWA of 25 ppm to protect workers from skin irritation, CNS depression, and respiratory failure.

Derivation of a Provisional RfC for Trimethylbenzene

A provisional inhalation RfC can be derived for an isomeric mixture of trimethylbenzene based on the occupational study by Battig et al. (1958). Adverse respiratory, neurological, and hematological effects were reported in that study in workers exposed to a solvent containing >80% trimethylbenzenes. The lowest reported exposure concentration was 10 ppm (49 mg/m^3, assuming the solvent content to be exclusively trimethylbenzenes) and constitutes a LOAEL. The RfC can be calculated as follows:

$$\text{RfC} = 49\ \text{mg/m}^3 \times (10\ \text{m}^3/20\ \text{m}^3) \times (5\ \text{day}/7\ \text{day})/3000$$

$$= 17.5\ \text{mg/m}^3/3000$$

where: 49 mg/m^3 = LOAEL

10 m^3/20 m^3 = adjustment assuming that occupationally exposed humans inhale 10 m^3/workday and 10 m^3 during the remainder of the day.

5 day/7 day = adjustment for a 5-day work week.

3000 = uncertainty factor (10 for the use of a LOAEL, 10 to extrapolate from subchronic to chronic exposure, 10 to protect sensitive individuals, and 3 for incomplete database).

RfC = 6E-3 mg/m^3

Confidence in the key study is low because a small number of subjects were examined, and the workers were exposed to other chemicals in the solvent mixture. Low confidence in the database reflects the lack of chronic, developmental or reproduction studies in animals by any route of exposure, and the poor quality of the available subchronic inhalation studies. Low confidence in the provisional inhalation RfC follows.

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Attachment I

(93-17c/3-12-93)

Risk Assessment Issue Paper for:
Derivation of a Subchronic RfC for Naphthalene (CASRN 91-20-3)

Review documents available for naphthalene include U.S. EPA (1980, 1987a, 1990) and ATSDR (1990). An update search of TOXLINE (1992-1993) was conducted to identify new studies pertinent to derivation of a subchronic inhalation RfC. No pertinent new studies were located.

ACGIH (1992, 1986) has adopted an TLV-TWA of 10 ppm and a STEL of 15 ppm for naphthalene to prevent ocular effects, acknowledging that these levels may not prevent blood changes in susceptible individuals. The TLV values are based on the following statement, which occurs at the end of an analytical methods paper: "...distinct irritation of the eyes is noticeable when concentrations exceed 15 ppm" (Robbins, 1951). The statement refers to exposure in the work place; additional detail was not provided. The OSHA (1989, 1992) PELs and NIOSH (1992) RELs are identical to the ACGIH TWA and STEL values for this chemical.

HUMAN EXPOSURE

Very little information on the health effects of inhalation exposure to naphthalene is available. In humans, ocular effects including cataract formation have been associated with occupational exposure to naphthalene. Eight of 21 workers exposed to naphthalene fumes or dust for up to 5 years in a plant manufacturing a dye intermediate developed cataracts (Ghetti and Mariani, 1956). Seven of the affected workers were less than 50 years old and therefore would not have been likely to have developed cataracts spontaneously; the remaining affected worker was less than 60. Exposure was not quantitated. A 44-year-old man who worked with powdered naphthalene developed cataracts and a retinal hemorrhage and a co-worker had chorioretinitis in one eye (Van der Hoeve, 1906).

Hemolytic anemia has been observed in humans exposed by inhalation, combined inhalation and dermal exposure or combined inhalation and oral exposure. Case reports have described acute hemolytic anemia resulting from inhalation of naphthalene vapor from a naphthalene-containing medication by neonates (Hanssler, 1964; Irle, 1964), inhalation of naphthalene vapor from excessive numbers of mothballs in the home by a child and adults exposed for several years (Linick, 1983), and inhalation of naphthalene vapor or combined inhalation and dermal absorption by neonates (Cock, 1957; Dawson et al., 1958; Grigor et al., 1966; Naiman and Kosoy, 1964; Schafer, 1951; Valaes et al., 1963) and adults (Younis et al., 1957) from clothing and bedding that had been stored in mothballs. In some of the neonatal cases, clinical signs of neurological effects (lethargy, decreased crying) were mentioned; these signs may have been secondary to the decreased oxygen carrying capacity of the blood. In severe cases, the hemolytic anemia was accompanied by jaundice, high serum levels of bilirubin, cyanosis, and kernicterus with pronounced neurological signs. In addition, acute hemolytic anemia was diagnosed in an infant whose only exposure was during gestation; his

mother had inhaled and ingested naphthalene from moth balls during pregnancy, particularly during the last trimester (Zinkham and Childs, 1958). Neonates and persons with erythrocytes deficient in glucose-6-phosphate dehydrogenase or readily depleted of glutathione are especially sensitive to naphthalene exposure by any route (U.S. EPA, 1987a).

The only one of the above case reports that attempted to quantitate exposure was that by Linick (1983). Linick reported that a 26-year old woman and her 4-year-old daughter, and 7 relatives living in two other households, had anemia, jaundice, headache, confusion, nausea and vomiting. In addition, visitors to the woman's apartment became ill with headache, nausea and vomiting. Excessive numbers of mothballs (used for years to curb odors and control insects) were found in all three households. Analysis of air samples collected on charcoal in the woman's apartment revealed exposure levels of 20 ppb naphthalene, but the author pointed out that levels may have been much higher when fresh supplies of mothballs were introduced. When mothball use was discontinued, the above signs and symptoms disappeared.

Additional data are available from studies of oral exposure to naphthalene. These data include case reports of cataracts and acute hemolytic anemia in humans who ingested naphthalene. In humans, ingestion of naphthalene led to optic atrophy and bilateral cataracts in a 36-year-old man who ingested 5 g of naphthalene in oil (approximately 71 mg/kg assuming a body weight of 70 kg) (Lezenius, 1902). Acute hemolytic anemia, elevated serum bilirubin and jaundice have been reported in children and adults following ingestion of naphthalene from mothballs or deodorant cakes as a single incident, for up to 3 months or for unspecified periods (U.S. EPA, 1987a). Some cases also involved neurological signs such as lethargy (possibly secondary to the hemolytic anemia) and gastrointestinal irritation. In addition, a case of hemolytic anemia was reported in an infant exposed only during gestation when his mother ingested naphthalene from mothballs during the last trimester (Anziulewicz et al., 1959). Dose levels were not available for many of these cases of hemolytic anemia, but can be estimated at 85.7 and 142.8 mg/kg in two instances of unsuccessful suicide attempts [ingestion of 6 g by a 16-year-old girl and 10 g by a person of unspecified sex and age as reported by Gidron and Leurer (1956); body weights assumed to be 70 kg]; these cases recovered with treatment. As reviewed by Gerarde (1960), ingestion of 2 g naphthalene over a 2-day period caused the death of a 6-year-old child (approximately 48 mg/kg/day assuming 21 kg body weight (U.S. EPA, 1987b). Hence acute doses of naphthalene that caused cataracts, acute hemolytic anemia and death in humans are within the ranges of doses tested in oral subchronic and chronic studies in rodents (reviewed below) with no ocular effects, no toxicologically significant effects on erythrocyte parameters and no treatment-related deaths. The lack of ocular effects may be related to the relative insensitivity of nonpigmented strains.

ANIMAL EXPOSURE

B6C3F1 mice (75/sex/group) were exposed to naphthalene (scintillation grade, >99% purity) at target concentrations of 0, 10, 30 and 30 ppm (0, 52.4, 157 and 157 mg/cu.m; 2 high-exposure groups in separate chambers) for 6 hours/day, 5 days/week for 104 weeks (NTP, 1992). The duration-adjusted exposure levels were 0, 9.36, 28.08 and 28.08 mg/cu.m. The naphthalene vapor was generated by direct sublimation; the high exposure level was approximately half of the saturating

concentration for naphthalene at 20C. Overall mean analytical concentrations were not reported. Weekly mean concentrations were within 20% of the target concentration except for one week in which the mean for the 10 ppm group was 5.5 ppm. The mice were monitored twice a day for signs of toxicity, weighed at intervals throughout the study and 5 mice/sex/exposure level were examined ophthalmologically at 6-month intervals. Hematological evaluations were performed on 5 mice/sex/group at 14 days but the remaining scheduled interim evaluations were cancelled because of high mortality in the male controls. All the mice were necropsied; complete histopathologic examinations were performed on all control and high-exposure mice and on all mice that died or were killed moribund prior to 21 months. In addition, the nasal cavities and lungs of all low-exposure mice were examined histopathologically.

Because survival, body weight and tumor incidence data for male and female mice of the two high-exposure groups were similar to each other, data for the two high-exposure groups were combined. Mean body weights of both sexes of mice exposed to both levels of naphthalene were slightly lower than but within 10% of those of controls; the depressions in body weight tended to be greater at the higher exposure level and occurred throughout the study in the female groups and during the first 18 months in the male groups. During the periods of exposure to naphthalene, the mice tended to huddle in the cage corners, which resulted in less frequent fighting among the exposed males than among control males (the mice were housed 5/cage). This behavior probably was a response to the irritating qualities of the naphthalene vapor, but the possibility of a nervous system effect cannot be ruled out. Survival was statistically significantly decreased in the control males relative to the exposed males; the decreased survival was due to wounds and secondary lesions resulting from the higher frequency of fighting in the control males. Survival in the females was not affected. According to the authors, no biologically significant, treatment-related changes in hematological values were seen at 14 days of exposure. There were small but statistically significant alterations in hematological values (decreased hematocrit and mean cell volume and increased leukocyte counts) in females at the high-exposure level as compared with controls. No treatment-related ocular lesions were seen during the ophthalmological examinations.

Nonneoplastic nasal lesions occurred in nearly all of the exposed mice and in virtually none of the controls. These lesions included inflammation, olfactory epithelial metaplasia and respiratory epithelial hyperplasia. They were focal or multifocal, were found primarily in the posterior nasal cavity and were of minimal to moderate severity. The nasal lesions were thought to be consistent with a generalized inflammatory and regenerative process. Incidences of these nasal lesions, followed by average severity grade in parentheses (1=minimal, 2=mild, 3=moderate and 4=marked), were as follows, in order of control through high exposure: inflammation - 0/70 (0), 67/69 (2.2) and 133/135 (2.6) for males and 1/69 (2.0), 65/65 (2.3) and 135/135 (2.4) for females; olfactory epithelial metaplasia - 0/70 (0), 66/69 (2.5) and 134/135 (2.6) for males and 0/69 (0), 65/65 (2.5) and 135/135 (2.4) for females; respiratory epithelial hyperplasia - 0/70 (0), 66/69 (2.6) and 134/135 (2.8) for males and 0/69 (0), 65/65 (2.5) and 135/135 (2.7) for females. Tests for trend were statistically significant for each type of lesion in each sex and pairwise comparisons with controls were statistically significant for each type of lesion in each sex at each exposure level. For nasal effects, the LOAEL is 10 ppm (52.4 mg/cu.m), 6 hours/day, 5 days/week; the LOAEL(ADJ) is 9.36 mg/cu.m and the LOAEL(HEC) is 1.3 mg/cu.m.

Nonneoplastic pulmonary lesions also occurred in the exposed mice. The pulmonary lesions were categorized as inflammation or as the similar but more advanced lesion granulomatous inflammation. According to the authors, usually only one of these terms was applied to any given mouse. Incidences of these lesions followed by average severity grade in parentheses (1=minimal, 2=mild, 3=moderate and 4=marked) were as follows, in order of control through high exposure: inflammation - 0/70 (0), 21/69 (2.3) and 56/135 (2.1) for males and 3/69 (1.7), 13/65 (2.2) and 52/135 (2.1) for females; granulomatous inflammation - 0/70 (0), 19/69 (2.2) and 15/135 (2.6) for males and 0/69 (0), 38/65 (2.1) and 42/135 (2.3) for females. Tests for trend were statistically significant for each type of lesion in each sex and pairwise comparisons with controls were statistically significant for each type of lesion in each sex at each exposure level. A rough estimate of the total combined incidence of pulmonary inflammation and granulomatous inflammation can be obtained by adding the number of animals with each condition; this estimate may slightly overestimate the combined incidence. Again in order of controls through high exposure, the total incidence of pulmonary inflammation or granulomatous inflammation combined is roughly estimated at 0/70 (0%), 40/69 (67%) and 71/135 (53%) for males and 3/69 (4%), 51/65 (78%) and 94/135 (70%) in females. Additional pulmonary findings that were statistically significantly increased in pairwise comparison with controls were the incidences of histiocyte infiltration in males at both exposure levels, incidences of lymphocyte infiltration in females at both exposure levels and the incidence of bronchial submucosal gland inflammation at the high exposure level in females. The trachea was not affected in either sex. Neoplastic changes were also seen in the lungs. The incidences of alveolar/bronchiolar adenoma were statistically significantly increased in the high-exposure females and 1 alveolar/bronchiolar carcinoma occurred in a high-exposure female. Incidences of alveolar/bronchiolar adenoma or carcinoma were marginally but not significantly increased in males at both naphthalene exposure levels; the marginally increased incidences were attributed to the greater survival in the exposed males as compared with control males. For nonneoplastic lung effects, the LOAEL is 10 ppm (52.4 mg/cu.m) 6 hours/day, 5 days/week; the LOAEL(ADJ) is 9.4 mg/cu.m and the LOAEL(HEC) calculated for a gas:respiratory effect in the pulmonary region (Sa = 291 sq.cm. and Sh = 635,545 sq.cm.) is 46 mg/cu.m.

The RfC is based on the LOAEL(HEC) for nasal effects because the dose-response curve for the nasal effects appears to be steeper and the effects were more severe than in the lung, suggesting that the nasal cavity may be more sensitive. In addition, use of the nasal LOAEL(HEC) yields a more conservative RfC.

The only subchronic or chronic inhalation study of naphthalene in experimental animals is the chronic study in mice by the NTP (1992), chosen as the principal study. No reproductive or developmental toxicity studies of inhaled naphthalene were located. A few acute inhalation studies are available, and are described below.

In an unpublished study, Fait and Nachreiner (1985) exposed 5 male and 5 female Wistar rats to a mean analytical concentration of 77.7 ppm naphthalene for 4 hours in a 24C exposure chamber; no control animals were used. The naphthalene vapor was generated by heating naphthalene (nominal purity 100%) to 101C. This was the highest concentration that could be generated; large amounts of naphthalene crystallized on the walls of the exposure chamber. Following exposure, the rats were held for a 14 day observation period. No deaths occurred. Clinical signs during exposure included closed

eyes, lacrimation and mouth breathing. No signs of toxicity were seen during the observation period. "Complete" gross necropsy (including nasal cavity, trachea, bronchi and lungs) at the end of 14 days revealed no lesions attributable to treatment. Whether the eyes were examined was not stated.

In an unpublished study performed for Union Carbide (1949), 6 rats were exposed for 8 hours to a "substantially saturated" vapor of naphthalene generated at room temperature and were then observed for 14 days. Body weight gain was normal in 5 of the rats and static in the sixth. Another group of 6 rats was exposed to a mist generated by aerating naphthalene at 170C. One of the rats died but the remaining 5 survived and "gained weight normally". No additional details regarding this study are available. An additional unpublished study by Union Carbide (1968) reported that the LC50 for 8-hour inhalation exposure was 100 ppm in rats; details were not available. This value is higher than the saturation concentration at 24C reported by Fait and Nachreiner (1985).

Buckpitt (1982, 1992) studied pulmonary effects in groups of 5 male Swiss Webster mice subjected to nose-only inhalation exposure to naphthalene at analytical concentrations of 93, 179 or 376 ug/L (93, 179 or 376 mg/cu.m) for 4 hours. The incidences of mild lung damage (swelling of some cells in major or in terminal airways) at the two lower exposure levels were 2/5 and 2/5. At the highest exposure, the incidence of mild lung damage was 2/5 and of moderate lung damage (sloughing of some cells into major or terminal airway lumens) was 3/5. Lung damage was not seen in controls exposed to filtered air.

Studies associating inhalation exposure with the development of cataracts or hemolytic anemia in experimental animals were not located. These effects were not seen in the only available chronic inhalation study (NTP, 1992), although slight decreases in hematocrit and in mean cell volume were reported in the female mice exposed to 30 ppm for 14 days (the only time interval for which hematological analyses were performed). Based on oral studies of naphthalene cataractogenesis (Van Heyningen, 1979; Van Heyningen and Pirie, 1967, 1976), the nonpigmented strain of mice used in the NTP (1992) study would not be expected to be sensitive to the ocular toxicity of naphthalene. The acute inhalation studies may not have examined endpoints relevant to these effects.

Additional data are available from studies of oral exposure to naphthalene. These data include orally administered naphthalene in experimental animals. In experimental animals, subchronic and chronic oral studies of naphthalene toxicity reported effects that were relatively nonspecific. These effects included decreased body weight gain in rats in a subchronic gavage study (BCL, 1980a), transient clinical signs possibly indicative of neurological effects (lethargy and decreased food consumption) (BCL, 1980b) and altered organ weights (Shopp et al., 1984) in mice in subchronic gavage studies, and no toxic effects in a limited chronic dietary study in rats (Schmahl, 1955). Cataracts and hematological effects were seen only in acute, high-dose oral studies (Van Heyningen and Pirie, 1967; Fitzhugh and Buschke, 1949; Koch et al., 1976; Zuelzer and Apt, 1949).

In a subchronic oral study in Fischer 344 rats, naphthalene (>99% pure) in corn oil was administered by gavage to groups of 10 males and 10 females at dose levels of 0, 25, 50, 100, 200 or 400 mg/kg, 5 days/week for 13 weeks (BCL, 1980a). At the highest dose level, two males died during the last week of treatment, and rats of both sexes displayed diarrhea, lethargy, hunched posture and rough coats at intermittent intervals throughout the study (BCL, 1980a). Food consumption was

not affected by exposure, but average body weight gains (over the 13-week period) in several groups of exposed rats were depressed by more than 10% of the weight gain displayed by the controls (24% and 60% depression in 200- and 400-mg/kg males compared with controls, and 13%, 15% and 69% depression in 100-, 200- and 400-mg/kg females). Differences between mean values of hematological parameters in exposed groups and those in control groups were <10% of control values, except for a 94% increase in numbers of mature neutrophils and a 25.1% decrease in numbers of lymphocytes in male 400-mg/kg rats and a 37.2% increase in mature neutrophils in 400-mg/kg females. Histological examinations revealed small incidences of lesions in kidneys of males and thymuses of females of the 200 and 400 mg/kg groups; no lesions were observed in kidneys or thymuses of respective controls. Focal cortical lymphocytic infiltration or focal tubular regeneration were observed in kidneys of 2/10 200-mg/kg male rats, and diffuse renal tubular degeneration occurred in 1/10 400-mg/kg male rats. Lymphoid depletion of the thymus occurred in 2/10 400-mg/kg females, but not in any 200-mg/kg females. No other compound-related lesions were detected in other tissues. Decreased body weight gain was the most sensitive effect noted in this study; a NOAEL of 50 mg/kg and a of LOAEL 100 mg/kg (35.7 and 71.4 mg/kg/day, respectively) were identified.

In a subchronic oral study in B6C3F1 mice, groups of 10 males and 10 females were administered gavage doses of naphthalene in corn oil at levels of 0, 12.5, 25, 50, 100 or 200 mg/kg 5 days/week for 13 weeks (BCL, 1980b). Seven mice (3 males and 2 females of the 200-mg/kg group, one female of the 25-mg/kg group and one control male) died during the 2nd, 3rd and 4th weeks from gavage trauma or accident. Transient signs of toxicity (lethargy, rough hair coats and decreased food consumption) occurred between weeks 3 and 5 in the 200-mg/kg groups. All exposed male mice gained more weight during the study than did control males (weight gains expressed as a percentage of control weight gain were 154.3, 116.0, 125.9, 122.2 and 107.4% for the 12.5-200 mg/kg groups, respectively). In contrast, exposed female mice displayed decreased weight gain compared with controls (weight gains expressed as a percentage of control weight gain were 97.5, 81.5, 81.5, 77.8 and 76.5% for the 12.5-200 mg/kg groups, respectively). All mice were necropsied and 27 organs (including the eyes, thymus and lungs) from the mice in the control and high-dose groups were examined histologically. No exposure-related lesions were observed in any organs. The highest incidences of lesions observed were for minimal to mild, focal or multifocal, subacute pneumonia in both controls (4/10 males and 2/10 females) and high-dose mice (4/10 males and 5/10 females). Organ weight data were not reported. Hematological analyses were performed on all groups. Exposed groups displayed mean values that were within 10% of the control means for the following parameters: hemoglobin, hematocrit, total white blood cells and total red blood cells. An increase in lymphocytes (18% increase) and a decrease in segmented neutrophils (38.8% decrease) in high-dose males were not considered biologically significant by the authors. The authors considered the highest dose level to be a NOAEL. The authors expressed the belief that the differences in weight gain did not conclusively indicate a toxic effect, because other consistent signs of toxicity were not observed and "such a marked indication of sex difference in (body weight) response" was observed. Adopting the authors' interpretation that the effects on female body weight gain in this study were equivocal, but not their interpretation of the absence of other signs of toxicity, the highest dose level (200 mg/kg; 142.9 mg/kg/day) in the study is judged to be a LOAEL for transient clinical signs of toxicity. These observations (especially lethargy) are similar to clinical observations in the NTP (1991) rat developmental study (reviewed below) and may be indicaative of neurological effects.

In a subchronic oral study in albino CD-1 mice, both sexes (approximately 6-weeks old at the start) were administered gavage doses of 0, 5.3, 53 or 133 mg/kg naphthalene (99.3% pure) in corn oil for 90 consecutive days (Shopp et al., 1984). A naive control group and the low- and mid-dose groups each contained 76 male mice and 40 female mice. The vehicle control group contained 112 male mice and 76 female mice. The high-dose group contained 96 male mice and 60 female mice. Significant chemical-related decreases in terminal body weights or survival were not observed in either sex. No significant alterations in absolute or relative organ weights occurred in exposed male mice, but significant decreases in absolute weights of brain, liver and spleen and relative weight of spleen occurred in high-dose females. Histopathological examination of organs was not conducted, but the authors noted that cataracts were not formed in exposed mice. Examination of hematological parameters (including numbers of leukocytes, erythrocytes and platelets and determination of hematocrit and hemoglobin) at termination revealed only slight, but statistically significant, increases in hemoglobin in high-dose females only; however the hematological data were not shown in the available report. Chemical analysis of serum showed statistically significant decreased blood urea nitrogen in all exposed female groups, and increased serum globulin and protein in the two highest female dose groups. No exposure-related responses were found in a battery of immunological assays (humoral immune response, lymphocyte responsiveness, delayed-type hypersensitivity response, popliteal lymph node response, and bone marrow function); immunotoxic responses were observed in positive controls given intraperitoneal injections of 50 mg/kg cyclophosphamide on days 87, 88, 89, and 90. The study identified a LOAEL of 133 mg/kg/day and a NOAEL of 53 mg/kg/day for altered organ weights in female mice; the toxicological significance of the statistically significant alterations in hematological and serum chemical parameters is not clear.

In the only chronic oral study, a group of 28 BDI and BDIII rats (100 days-old at the start, sex not specified) were fed naphthalene in an oily solution in normal food at daily dosage levels of 10-20 mg/rat, 6 times per week until a total dose of 10 g/rat was reached on the 700th experimental day (Schmahl, 1955). Hence the dose was approximately 35 mg/kg/day (10,000 mg/rat x 1/700 days x 1/0.35 kg x 6 days/7 days = 35 mg/kg/day, assuming a reference body weight of 0.35 kg). The test material was tested for purity by absorption spectroscopy, but a percent purity was not reported. The animals were allowed to live until spontaneous death; in some cases death occurred after more than 1000 experimental days. Body weight data were not reported. The lifespan of the orally treated rats was reported to be 800 days and corresponded to the lifespan of control rats; however, information on control rats (numbers of animals, actual lifespan, etc.) was not provided. Rats were autopsied at death and those organs that appeared unusual were examined histologically. Reported results from the autopsy and histological examinations were restricted to the statement that no toxic effects were seen, including eye damage and tumors. Inadequacies in experimental design (e.g. only one dose level was administered, the histopathological examination was not complete, hematological endpoints were not evaluated and some rats lived as long as 300 days beyond exposure before being examined) and inadequacies in reporting of experimental details and results limit the conclusions that can be drawn from this study.

None of the available subchronic or chronic oral studies of naphthalene report cataracts or hemolytic anemia in rats or mice. Cataract formation and retinal damage have been described in acute oral studies in rabbits at a dose of 1000 mg/kg/day for up to 28 days (van Heyningen and Pirie, 1967) and slight cataracts developed in weanling rats fed 20,000 ppm naphthalene in the diet for 2 months

[approximately 2000 mg/kg/day using an assumed body weight of 0.18 kg and an equation described by U.S. EPA (1987b) that relates the rate of food consumption to body weight in weanling rats] (Fitzhugh and Buschke, 1949). Koch et al. (1976) reported that when administered 1000 mg/kg of naphthalene on alternate days, all rats of pigmented strains developed cataracts within 16 to 28 days, whereas only some of the rats of the albino strains (Sprague-Dawley and Wistar) developed minor lens changes after a greater period of time. These dose/exposure levels are very high compared with those studied in the subchronic and chronic studies. The demonstration of naphthalene-induced hemolytic anemia in animals is restricted to an acute oral exposure study with dogs (Zuelzer and Apt, 1949). In this study, hemolytic anemia (decreased hemoglobin, Heinz body appearance, reticulocytosis) occurred in three dogs following oral administration of naphthalene incorporated into a meat diet; one dog (weighing 7.3-kg body weight) received a single dose of 3 g (410 mg/kg), the second 5.9-kg dog received a single 9 g dose (1.53 g/kg), and the third 6.8-kg dog received 7 consecutive daily doses ranging from 0.5 to 3.0 g (74-441 mg/kg) for a total dose of 12.5 g naphthalene.

A few developmental toxicity studies of oral exposure to naphthalene in experimental animals are available. As reviewed below, results of one of these studies (NTP, 1991) indicated that in mice, the dams are more sensitive than the fetuses; the critical effect in the dams was transient clinical signs (lethargy, slow breathing, prone body posture) indicative of CNS depression. The other studies were conducted at dose levels that either produced frank toxic effects or no effects in both dams and fetuses.

In an oral developmental study in Sprague-Dawley CD rats, naphthalene in corn oil was administered by gavage to groups of 25-26 pregnant females at dose levels of 0, 50, 150 or 450 mg/kg/day on gestational days 6-15 (NTP, 1991). Dams were examined daily for clinical signs of toxicity until sacrifice at gestation day 20. Fetuses were examined on gestation day 20 for gross, visceral and skeletal malformations. Fetal development was essentially unaffected, but significant maternal toxicity was observed. Two dams died in the low-dose group, but no deaths occurred in the higher dose groups. Exposed dams displayed lethargy, slow breathing, and prone body posture indicative of CNS depression during the first five days of dosing. This behavior was evident in 81% of the dams in the low-dose group and 96% of mid- and high-dose dams on the first day of dosing. By the third day of dosing, this behavior had disappeared in the low-dose group indicating that tolerance to these effects had developed. The mid-dose group no longer displayed signs of CNS depression on the sixth day of dosing. The incidence of naphthalene-induced lethargy and slow breathing in the high-dose group declined with continued exposure, but never dropped below 15%. Statistically significant decreased body weight and body weight gain were observed in dams of the two highest dose groups; the effects on body weight were associated with decreased food and water consumption. No significant differences were observed between exposed groups and controls in maternal liver weights and gravid uterine weights. No treatment related effects were noted in the number of corpora lutea per dam, the number of implantation sites per litter, the number of live fetuses per litter, the incidences of resorptions or the incidences of late fetal deaths. Significant tests for trends with increasing dose were demonstrated for adversely affected implants per litter and for average fetal body weight per litter, but an analysis of variance did not detect a significant overall effect of dose on these parameters. Unequivocal exposure-related effects on visceral and skeletal fetal development were not observed. The authors concluded that the highest dose (450 mg/kg) was a NOAEL for fetal developmental effects and that the lowest dose level in the study (50 mg/kg/day) was a LOAEL for maternal toxicity.

In a less adequate oral developmental toxicity study in CD-1 mice, pregnant females were given 8 consecutive daily gavage doses of 0 or 300 mg/kg naphthalene in corn oil starting on day 7 of gestation (Plasterer et al., 1985). The control and exposed groups contained 40 and 33 mice, respectively. Deaths occurred in 5/33 exposed dams during the dosing period; all control dams survived and delivered litters. Clinical observations for signs of toxicity were not reported. Natural deliveries occurred in 26/28 exposed dams that survived the treatment. Average weight gain during gestation was significantly decreased in exposed dams compared with controls. The average number of live pups per litter in the exposed group was significantly smaller than that in the control group, but average body weight of the pups was not affected by exposure. Examination of the pups for gross structural abnormalities revealed no exposure-related effects. The 300 mg/kg level appeared to produce frank maternal (deaths and decreased weight gain in survivors) and fetal (decreased number of live pups per litter) effects.

In an oral range-finding developmental toxicity study in New Zealand white rabbits, groups of 4 does were given gavage doses of 0, 50, 250, 630 or 1000 mg/kg/day naphthalene in 1% methylcellulose from gestation days 6 to 18 (Naismith and Matthews, 1985). All of the high-dose does died. Mortality and decreased weight gain occurred in 2/4 does at 630 mg/kg/day; survivors at this dose aborted their pregnancies. At the two lower dose levels, no exposure-related alterations were observed in maternal reproductive performance, fetal survival or gross fetal structural development.

In the subsequent developmental toxicity study in New Zealand white rabbits, groups of 18 does were given gavage doses of 0, 40, 200 or 400 mg/kg/day naphthalene in 1% methylcellulose from gestation day 6 to 18 (Naismith and Matthews, 1986). Cesarean sections were performed on does at gestational day 29. Maternal survival, body weights and body weight gains were not affected by exposure. Clinical observations were reported to reveal no signs of toxicity. Reproductive parameters (including number of corpora lutea, total number of implantations, viable or nonviable fetuses, pre-or post-implantation loss, fetal body weights and fetal sex distribution) were not affected by exposure. Examination of fetuses for visceral and skeletal abnormalities revealed no exposure-related effects on fetal development. The study identified 400 mg/kg/day as a NOEL for maternal and fetal effects in rabbits.

Developmental studies with parenterally administered naphthalene have provided conflicting results. Harris et al. (1979) reported that the incidence of retarded cranial ossification and heart growth in offspring of Sprague-Dawley rats given intraperitoneal doses of 395 mg/kg naphthalene in corn oil on gestational days 1-15 was 2 times higher than the control incidences. In contrast, Hardin et al. (1981) reported that intraperitoneal administration of the same dose of naphthalene in corn oil on gestation days 1-15 was not maternotoxic, fetotoxic or teratogenic to Sprague-Dawley rats.

Although the above subchronic and developmental toxicity studies of naphthalene indicate that rats may be slightly more sensitive than mice to orally administered naphthalene, oral LD50 values (U.S. EPA, 1987a) and acute intraperitoneal studies of pulmonary effects (reviewed below) provide evidence that mice are more sensitive to the acute lethality and pulmonary toxicity of naphthalene than are rats and that rats may be somewhat more sensitive to acute nasal toxicity. The relevance of this information to inhalation exposure is uncertain. Intraperitoneal administration of naphthalene, at single doses ranging from approximately 50-350 mg/kg, produced necrosis of pulmonary bronchiolar

epithelial cells in C57B/6J, Swiss Webster and Swiss T.O. mice; the non-ciliated cells (Clara cells) were more sensitive (Mahvi et al., 1977; O'Brien et al., 1985, 1989; Plopper et al., 1992; Reid et al., 1973; Tong et al., 1981). Single intraperitoneal injections of naphthalene at 64-1600 mg/kg did not produce pulmonary necrosis in Sprague-Dawley and Wistar-derived rats, suggesting that rats are not as susceptible to the acute pulmonary toxicity of parenterally administered naphthalene (Dinsdale and Verschoyle, 1987; O'Brien et al., 1985; Plopper et al., 1992). Hamsters were intermediate in sensitivity (Plopper et al., 1992). When naphthalene was administered to mice daily for seven days at intraperitoneal dose levels of 50, 100 or 200 mg/kg, significant alterations in lung morphology were not observed (O'Brien et al., 1989). These results suggest that a tolerance to the pulmonary effects of parenterally administered naphthalene develops in mice, and may explain why lung effects were not observed in subchronic gavage tests with naphthalene in mice (see BCL, 1980b). In the inhalation study of naphthalene in B6C3F1 mice (NTP, 1992), however, chronic exposure resulted in lung effects, primarily in the alveoli, as discussed previously. In the single-dose intraperitoneal studies by Plopper et al. (1992), necrosis of the olfactory region of the nasal mucosa was seen in mice at 400 mg/kg, in rats at 200 mg/kg and in hamsters at 400 mg/kg; hence rats appeared to be more sensitive to the nasal toxicity than were mice or hamsters. In the chronic inhalation study of naphthalene in B6C3F1 mice (NTP, 1992), nasal effects occurred not only in the olfactory epithelium, but also in the respiratory epithelium, as discussed previously.

The oxidative metabolism of naphthalene to reactive intermediates has been proposed to be involved in the production of the observed acute lung effects in mice (Buckpitt and Franklin, 1989). Evidence from single-dose intraperitoneal studies of naphthalene's effects on potential target tissues in mice, rats, and hamsters, correlated with evidence from in vitro metabolism studies using microsomes from the same tissues and species, indicates that an epoxide, 1R,2S-naphthalene oxide, may be the toxic intermediate responsible for the pulmonary effects and also for effects on the nasal olfactory epithelium (Buckpitt et al., 1992; Plopper et al., 1992). Studies in which isolated mouse lung was perfused with naphthalene or selected metabolites demonstrated that pulmonary damage (Clara cell necrosis) does not require extrapulmonary metabolism but can be mediated entirely through pulmonary metabolism of naphthalene to reactive intermediates (Kanekal et al., 1990) and that circulating naphthalene oxide could cause similar pulmonary damage; other metabolites were ineffective or much less effective (Kanekal et al., 1991). As reviewed by Kanekal et al. (1991), naphthalene oxide has been detected in the blood of mice injected intraperitoneally with a high dose (600 mg/kg) of naphthalene; the blood concentration was higher than the concentration used in the lung perfusion experiment. No information was located regarding naphthalene oxide in blood following inhalation (or oral) exposure to naphthalene.

SUMMARY

One of the single-exposure studies (Buckpitt, 1982, 1992) and the chronic exposure study (NTP, 1992) identify the respiratory tract as a target for inhalation exposure. The single-exposure study in mice (Buckpitt, 1982, 1992) is not of adequate duration or quality to serve as the basis for a subchronic RfC, but does provide evidence that a 4-hour nose-only single inhalation exposure of mice to 93, 179 or 376 mg/m^3 produced lung damage, the severity and incidence of which increased with increasing exposure level. Other portions of the respiratory tract, such as the nasal cavity, were not

examined.

The chronic inhalation study identified nasal lesions (inflammation, metaplasia and hyperplasia) as the critical effect of inhalation exposure to naphthalene in mice (NTP, 1992). These lesions, which occurred at both exposure levels [10 and 30 ppm (52.4 and 157 mg/m^3), 6 hr/day, 5 days/week] were thought to be consistent with a generalized inflammatory and regenerative process. Lesions were also seen in the lungs, but these occurred at incidences and severities that were lower than for the nasal lesions. Accordingly, the LOAEL for this study is 52.4 mg/m^3 ($LOAEL_{ADJ}$ = 9.36 mg/m^3; $LOAEL_{HEC}$ = 1.3 mg/m^3).

Because respiratory effects have been demonstrated in both an acute and a chronic study at similar exposure levels, it is reasonable to assume that subchronic exposure would also produce these effects at similar exposure levels. Accordingly, it is recommended that the proposed chronic RfC of 4E-4 mg/m^3, based on the LOAEL for nasal effects in chronically exposed mice (NTP, 1992), be adopted as the provisional subchronic RfC. The proposed chronic RfC was calculated by applying an uncertainty factor of 3000 to the $LOAEL_{HEC}$ of 1.3 mg/m^3. The uncertainty factor reflects 10 for the use of a LOAEL, 3 for interspecies conversion with dosimetric adjustments, 10 for variation in sensitivity among humans and 10 for the lack of adequate chronic or subchronic inhalation studies of pertinent extrarespiratory endpoints [ocular effects in a sensitive (pigmented) strain and hematological and neurological effects] and the lack of a 2-generation reproductive study. This uncertainty factor is appropriate for subchronic as well as chronic exposure.

The principal study was fairly well-conducted, with adequate numbers of animals exposed by inhalation to two different concentrations and suitable statistical analysis, but had several deficiencies. Failure to cage the male mice individually resulted in high, fighting-related mortality among control males, hematological endpoints were not investigated adequately, and the number and spacing of exposure levels were not adequate to define a NOAEL or to explore potential differences in sensitivity to respiratory and extrarespiratory toxicity. Accordingly, confidence in the study is medium. The lack of adequate chronic or subchronic inhalation data for ocular effects in an appropriate (pigmented) strain and for hematological and neurological effects, and the lack of a 2-generation reproductive study limits confidence in the data base to low. Developmental toxicity data and subchronic/chronic toxicity data in several animal species are available for the oral route. Reflecting medium confidence in the principal study and low confidence in the data base, confidence in the RfC is low.

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APPENDIX G

EMISSION ESTIMATES BY SOURCE CATEGORY

PHOENIX STUDY AREA

Distribution of HAP Emissions for Phoenix (%)

Description	1,4-Dichlorobenzene 106-46-7	Dibromoethane 106-93-4	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Acrylonitrile 107-13-1
Fuel Combustion - Agricultural					
Fuel Combustion - Oil and Gas Production					
Fuel Combustion - Petroleum Refining					
Fuel Combustion - Other Manufacturing/Industrial					
Fuel Combustion - Electric Utilities				<1	
Fuel Combustion - Other Services and Commerce			<1	<1	
Fuel Combustion - Residential					
Fuel Combustion - Other			<1		
Waste Burning - Agricultural Debris					
Waste Burning - Range Management					
Waste Burning - Forest Management					
Waste Burning - Incineration					
Waste Burning - Other					
Solvent Use - Dry Cleaning			<1		
Solvent Use - Degreasing					
Solvent Use - Architectural Coating					
Solvent Use - Other Surface Coating					
Solvent Use - Asphalt Paving					
Solvent Use - Printing					
Solvent Use - Domestic	71				
Solvent Use - Industrial	29				
Solvent Use - Other					
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction					
Petroleum Processes, Storage & Transfer - Petroleum Refining					
Petroleum Processes, Storage & Transfer - Petroleum Marketing					
Petroleum Processes, Storage & Transfer - Other					
Industrial Processes - Chemical				<1	4

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	1,4-Dichlorobenzene 106-46-7	Dibromoethane 106-93-4	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Acrylonitrile 107-13-1
Industrial Processes - Food and Agricultural					
Industrial Processes - Mineral Processes					
Industrial Processes - Metal Processes					
Industrial Processes - Wood and Paper					
Industrial Processes - Other					
Misc Processes - Pesticide Application					
Misc Processes - Farming Operations					
Misc Processes - Construction and Demolition					
Misc Processes - Entrained Road Dust - Paved					
Misc Processes - Entrained Road Dust - Unpaved					
Misc Processes - Unplanned Fires			<1		
Misc Processes - Waste Disposal					47
Misc Processes - Natural Sources					
Misc Processes - Other		100		3	45
On Road Vehicles			37	63	
Other Mobile - Off Road Vehicles			2	<1	
Other Mobile - Trains					
Other Mobile - Ships			<1	<1	
Other Mobile - Aircraft - Government			<1	2	
Other Mobile - Aircraft - Other			3	28	
Other Mobile - Mobile Equipment			3		
Other Mobile - Utility Equipment			53	2	
Other Misc Sources			<1	<1	4
Total	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Perchloroethylene 127-18-4	Hexavalent Chromium 18540-29-9	Trimethylbenzene 25551-13-7	Formaldehyde 50-00-0
Fuel Combustion - Agricultural				
Fuel Combustion - Oil and Gas Production				
Fuel Combustion - Petroleum Refining				
Fuel Combustion - Other Manufacturing/Industrial				
Fuel Combustion - Electric Utilities			<1	<1
Fuel Combustion - Other Services and Commerce		<1		<1
Fuel Combustion - Residential			<1	<1
Fuel Combustion - Other				1
Waste Burning - Agricultural Debris				<1
Waste Burning - Range Management				
Waste Burning - Forest Management				
Waste Burning - Incineration				
Waste Burning - Other				
Solvent Use - Dry Cleaning	69			
Solvent Use - Degreasing	24		<1	
Solvent Use - Architectural Coating			<1	
Solvent Use - Other Surface Coating				
Solvent Use - Asphalt Paving			27	
Solvent Use - Printing			63	
Solvent Use - Domestic			7	<1
Solvent Use - Industrial				
Solvent Use - Other				
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				
Petroleum Processes, Storage & Transfer - Petroleum Refining				
Petroleum Processes, Storage & Transfer - Petroleum Marketing				
Petroleum Processes, Storage & Transfer - Other				
Industrial Processes - Chemical				
Industrial Processes - Food and Agricultural				
Industrial Processes - Mineral Processes			<1	

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Perchloroethylene 127-18-4	Hexavalent Chromium 18540-29-9	Trimethylbenzene 25551-13-7	Formaldehyde 50-00-0
Industrial Processes - Metal Processes				
Industrial Processes - Wood and Paper				
Industrial Processes - Other				
Misc Processes - Pesticide Application				
Misc Processes - Farming Operations				
Misc Processes - Construction and Demolition				
Misc Processes - Entrained Road Dust - Paved				
Misc Processes - Entrained Road Dust - Unpaved				
Misc Processes - Unplanned Fires				
Misc Processes - Waste Disposal	<1			
Misc Processes - Natural Sources				
Misc Processes - Other	5	25		<1
On Road Vehicles				59
Other Mobile - Off Road Vehicles				<1
Other Mobile - Trains				2
Other Mobile - Ships				<1
Other Mobile - Aircraft - Government				<1
Other Mobile - Aircraft - Other				7
Other Mobile - Mobile Equipment				17
Other Mobile - Utility Equipment				12
Other Misc Sources	2	75	3	<1
Total	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Benzo(a)pyrene 50-32-8	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5	Mercury 7439-97-6
Fuel Combustion - Agricultural						
Fuel Combustion - Oil and Gas Production						
Fuel Combustion - Petroleum Refining						
Fuel Combustion - Other Manufacturing/Industrial	1		<1	<1	<1	
Fuel Combustion - Electric Utilities	49		<1	<1	<1	<1
Fuel Combustion - Other Services and Commerce			<1	<1	<1	
Fuel Combustion - Residential	46		<1	<1	<1	<1
Fuel Combustion - Other			<1	<1	<1	
Waste Burning - Agricultural Debris						
Waste Burning - Range Management						
Waste Burning - Forest Management						
Waste Burning - Incineration			<1	4	<1	55
Waste Burning - Other	2			<1	<1	
Solvent Use - Dry Cleaning						
Solvent Use - Degreasing			<1			
Solvent Use - Architectural Coating			<1			
Solvent Use - Other Surface Coating			<1			
Solvent Use - Asphalt Paving						
Solvent Use - Printing			<1			
Solvent Use - Domestic						
Solvent Use - Industrial						
Solvent Use - Other						
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction			<1			
Petroleum Processes, Storage & Transfer - Petroleum Refining				<1	<1	<1
Petroleum Processes, Storage & Transfer - Petroleum Marketing			<1			
Petroleum Processes, Storage & Transfer - Other			<1	<1	<1	<1
Industrial Processes - Chemical			<1			
Industrial Processes - Food and Agricultural						

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Benzo(a)pyrene 50-32-8	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5	Mercury 7439-97-6
Industrial Processes - Mineral Processes			<1	<1	<1	<1
Industrial Processes - Metal Processes						
Industrial Processes - Wood and Paper						
Industrial Processes - Other						
Misc Processes - Pesticide Application						
Misc Processes - Farming Operations			3	<1	2	<1
Misc Processes - Construction and Demolition						
Misc Processes - Entrained Road Dust - Paved				40	22	11
Misc Processes - Entrained Road Dust - Unpaved				2	2	2
Misc Processes - Unplanned Fires				<1	<1	
Misc Processes - Waste Disposal		<1	<1			
Misc Processes - Natural Sources				<1	<1	<1
Misc Processes - Other	2	2	<1	<1	<1	1
On Road Vehicles			55	3	<1	1
Other Mobile - Off Road Vehicles			<1	12	<1	<1
Other Mobile - Trains				7	<1	
Other Mobile - Ships			<1			
Other Mobile - Aircraft - Government			<1			
Other Mobile - Aircraft - Other			<1			
Other Mobile - Mobile Equipment			3			
Other Mobile - Utility Equipment			36			
Other Misc Sources		98	<1	30	71	27
Total	100	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Nickel 7440-02-0	Arsenic 7440-38-2	Barium 7440-39-3	Cadmium 7440-43-9	Vinyl Chloride 75-01-4
Fuel Combustion - Agricultural					
Fuel Combustion - Oil and Gas Production					
Fuel Combustion - Petroleum Refining					
Fuel Combustion - Other Manufacturing/Industrial	<1	<1	<1	<1	
Fuel Combustion - Electric Utilities	<1	<1		<1	
Fuel Combustion - Other Services and Commerce	<1	<1	<1	<1	
Fuel Combustion - Residential		<1	<1		
Fuel Combustion - Other	<1				
Waste Burning - Agricultural Debris					
Waste Burning - Range Management					
Waste Burning - Forest Management					
Waste Burning - Incineration	<1	<1	<1	1	
Waste Burning - Other	<1			3	
Solvent Use - Dry Cleaning					
Solvent Use - Degreasing					
Solvent Use - Architectural Coating					
Solvent Use - Other Surface Coating					
Solvent Use - Asphalt Paving					
Solvent Use - Printing					
Solvent Use - Domestic					
Solvent Use - Industrial					
Solvent Use - Other					
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction					
Petroleum Processes, Storage & Transfer - Petroleum Refining	<1	<1	<1	<1	
Petroleum Processes, Storage & Transfer - Petroleum Marketing					
Petroleum Processes, Storage & Transfer - Other	<1	<1	<1	<1	
Industrial Processes - Chemical					11
Industrial Processes - Food and Agricultural					

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Nickel 7440-02-0	Arsenic 7440-38-2	Barium 7440-39-3	Cadmium 7440-43-9	Vinyl Chloride 75-01-4
Industrial Processes - Mineral Processes	<1	2	<1	2	
Industrial Processes - Metal Processes					
Industrial Processes - Wood and Paper					
Industrial Processes - Other					
Misc Processes - Pesticide Application					
Misc Processes - Farming Operations	1	2	1	3	
Misc Processes - Construction and Demolition					
Misc Processes - Entrained Road Dust - Paved	28	15	25	31	
Misc Processes - Entrained Road Dust - Unpaved	2	7	2	2	
Misc Processes - Unplanned Fires					
Misc Processes - Waste Disposal					80
Misc Processes - Natural Sources	<1	<1	<1	<1	
Misc Processes - Other	3	<1	<1	6	<1
On Road Vehicles	1	3	3	10	
Other Mobile - Off Road Vehicles	<1	<1	<1	1	
Other Mobile - Trains	<1		<1		
Other Mobile - Ships					
Other Mobile - Aircraft - Government					
Other Mobile - Aircraft - Other					
Other Mobile - Mobile Equipment					
Other Mobile - Utility Equipment					
Other Misc Sources	63	68	67	41	9
Total	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Acetaldehyde 75-07-0	Dichloromethane 75-09-2	Ethylene Oxide 75-21-8	Trichloroethylene 79-01-6
Fuel Combustion - Agricultural				
Fuel Combustion - Oil and Gas Production				
Fuel Combustion - Petroleum Refining				
Fuel Combustion - Other Manufacturing/Industrial	<1			
Fuel Combustion - Electric Utilities	<1			
Fuel Combustion - Other Services and Commerce	<1			
Fuel Combustion - Residential	<1			
Fuel Combustion - Other				
Waste Burning - Agricultural Debris				
Waste Burning - Range Management				
Waste Burning - Forest Management				
Waste Burning - Incineration				
Waste Burning - Other			45	
Solvent Use - Dry Cleaning				
Solvent Use - Degreasing				
Solvent Use - Architectural Coating		29		90
Solvent Use - Other Surface Coating		6		
Solvent Use - Asphalt Paving				
Solvent Use - Printing				
Solvent Use - Domestic				
Solvent Use - Industrial		9		
Solvent Use - Other		56		10
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				
Petroleum Processes, Storage & Transfer - Petroleum Refining				
Petroleum Processes, Storage & Transfer - Petroleum Marketing				
Petroleum Processes, Storage & Transfer - Other				
Industrial Processes - Chemical				
Industrial Processes - Food and Agricultural			<1	
Industrial Processes - Mineral Processes				

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HABS research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emission for Phoenix (%) (Continued)

Description	Acetaldehyde 75-07-0	Dichloromethane 75-09-2	Ethylene Oxide 75-21-8	Trichloroethylene 79-01-6
Industrial Processes - Metal Processes				
Industrial Processes - Wood and Paper				
Industrial Processes - Other				
Misc Processes - Pesticide Application				
Misc Processes - Farming Operations				
Misc Processes - Construction and Demolition				
Misc Processes - Entrained Road Dust - Paved				
Misc Processes - Entrained Road Dust - Unpaved				
Misc Processes - Unplanned Fires				
Misc Processes - Waste Disposal		<1		<1
Misc Processes - Natural Sources				
Misc Processes - Other	<1	<1	<1	<1
On Road Vehicles	75			
Other Mobile - Off Road Vehicles	<1			
Other Mobile - Trains	2			
Other Mobile - Ships	<1			
Other Mobile - Aircraft - Government	<1			
Other Mobile - Aircraft - Other	8			
Other Mobile - Mobile Equipment	6			
Other Mobile - Utility Equipment	7			
Other Misc Sources	<1		54	
Total	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPS research program report shows the emissions magnitude in tons per year for each compound of interest.

TUCSON STUDY AREA

Distribution of HAP Emissions for Tucson (%)

Description	1,4-Dichlorobenzene 106-46-7	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Toluene 108-88-3	Chlorobenzene 108-90-7	Hexane 110-54-3
Fuel Combustion - Agricultural						
Fuel Combustion - Oil and Gas Production						
Fuel Combustion - Petroleum Refining						
Fuel Combustion - Other Manufacturing/Industrial				<1	<1	<1
Fuel Combustion - Electric Utilities						
Fuel Combustion - Other Services and Commerce				<1	<1	<1
Fuel Combustion - Residential		1		<1	<1	
Fuel Combustion - Other						
Waste Burning - Agricultural Debris						
Waste Burning - Range Management						
Waste Burning - Forest Management						
Waste Burning - Incineration						4
Waste Burning - Other		<1				<1
Solvent Use - Dry Cleaning				<1	<1	
Solvent Use - Degreasing						
Solvent Use - Architectural Coating				<1		7
Solvent Use - Other Surface Coating				21		
Solvent Use - Asphalt Paving				1		
Solvent Use - Printing				1		
Solvent Use - Domestic	83			3		
Solvent Use - Industrial	17				100	
Solvent Use - Other						
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction						
Petroleum Processes, Storage & Transfer - Petroleum Refining						
Petroleum Processes, Storage & Transfer - Petroleum Marketing				<1		3
Petroleum Processes, Storage & Transfer - Other				<1		2
Industrial Processes - Chemical						

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	1,4-Dichlorobenzene 106-46-7	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Toluene 108-88-3	Chlorobenzene 108-90-7	Hexane 110-54-3
Industrial Processes - Food and Agricultural						
Industrial Processes - Mineral Processes						
Industrial Processes - Metal Processes						
Industrial Processes - Wood and Paper						
Industrial Processes - Other						
Misc Processes - Pesticide Application						
Misc Processes - Farming Operations				<1		<1
Misc Processes - Construction and Demolition						
Misc Processes - Entrained Road Dust - Paved						
Misc Processes - Entrained Road Dust - Unpaved						
Misc Processes - Unplanned Fires		<1				
Misc Processes - Waste Disposal						
Misc Processes - Natural Sources						
Misc Processes - Other			<1			<1
On Road Vehicles		43	78	49		73
Other Mobile - Off Road Vehicles		<1	<1	<1		<1
Other Mobile - Trains						
Other Mobile - Ships		<1	<1	<1		<1
Other Mobile - Aircraft - Government		2	19	<1		
Other Mobile - Aircraft - Other		<1	<1	<1		
Other Mobile - Mobile Equipment		3		1		2
Other Mobile - Utility Equipment		51	2	15		8
Other Misc Sources				8		
Total	100	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Perchloroethylene 127-18-4	Hexavalent Chromium 18540-29-9	Trimethylbenzene 25551-13-7
Fuel Combustion - Agricultural			
Fuel Combustion - Oil and Gas Production			
Fuel Combustion - Petroleum Refining			
Fuel Combustion - Other Manufacturing/Industrial			
Fuel Combustion - Electric Utilities			<1
Fuel Combustion - Other Services and Commerce			
Fuel Combustion - Residential			<1
Fuel Combustion - Other			<1
Waste Burning - Agricultural Debris			
Waste Burning - Range Management			
Waste Burning - Forest Management			
Waste Burning - Incineration			
Waste Burning - Other			
Solvent Use - Dry Cleaning			
Solvent Use - Degreasing	83		<1
Solvent Use - Architectural Coating	17		
Solvent Use - Other Surface Coating			
Solvent Use - Asphalt Paving			5
Solvent Use - Printing			80
Solvent Use - Domestic			13
Solvent Use - Industrial			
Solvent Use - Other			
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction			
Petroleum Processes, Storage & Transfer - Petroleum Refining			
Petroleum Processes, Storage & Transfer - Petroleum Marketing			
Petroleum Processes, Storage & Transfer - Other			
Industrial Processes - Chemical			
Industrial Processes - Food and Agricultural			
Industrial Processes - Mineral Processes			

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Perchloroethylene 127-18-4	Hexavalent Chromium 18540-29-9	Trimethylbenzene 25551-13-7
Industrial Processes - Metal Processes			
Industrial Processes - Wood and Paper			
Industrial Processes - Other			
Misc Processes - Pesticide Application			
Misc Processes - Farming Operations			
Misc Processes - Construction and Demolition			
Misc Processes - Entrained Road Dust - Paved			
Misc Processes - Entrained Road Dust - Unpaved			
Misc Processes - Unplanned Fires			
Misc Processes - Waste Disposal			
Misc Processes - Natural Sources			
Misc Processes - Other			
On Road Vehicles			
Other Mobile - Off Road Vehicles			
Other Mobile - Trains			
Other Mobile - Ships			
Other Mobile - Aircraft - Government			
Other Mobile - Aircraft - Other			
Other Mobile - Mobile Equipment			
Other Mobile - Utility Equipment			
Other Misc Sources		100	2
Total	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Formaldehyde 50-00-0	Benzo(a)pyrene 50-32-8	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5
Fuel Combustion - Agricultural						
Fuel Combustion - Oil and Gas Production						
Fuel Combustion - Petroleum Refining						
Fuel Combustion - Other Manufacturing/Industrial	<1			<1	<1	<1
Fuel Combustion - Electric Utilities						
Fuel Combustion - Other Services and Commerce	<1			<1	<1	<1
Fuel Combustion - Residential	2	100		1	<1	<1
Fuel Combustion - Other						
Waste Burning - Agricultural Debris						
Waste Burning - Range Management						
Waste Burning - Forest Management						
Waste Burning - Incineration				<1	4	<1
Waste Burning - Other		<1			<1	<1
Solvent Use - Dry Cleaning						
Solvent Use - Degreasing						
Solvent Use - Architectural Coating				<1		
Solvent Use - Other Surface Coating						
Solvent Use - Asphalt Paving						
Solvent Use - Printing						
Solvent Use - Domestic						
Solvent Use - Industrial						
Solvent Use - Other						
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				<1		
Petroleum Processes, Storage & Transfer - Petroleum Refining						
Petroleum Processes, Storage & Transfer - Petroleum Marketing				1		
Petroleum Processes, Storage & Transfer - Other				<1		
Industrial Processes - Chemical						
Industrial Processes - Food and Agricultural						

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Formaldehyde 50-00-0	Benzo(a)pyrene 50-32-8	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5
Industrial Processes - Mineral Processes						
Industrial Processes - Metal Processes						
Industrial Processes - Wood and Paper						
Industrial Processes - Other						
Misc Processes - Pesticide Application						
Misc Processes - Farming Operations				<1	<1	<1
Misc Processes - Construction and Demolition						
Misc Processes - Entrained Road Dust - Paved					71	45
Misc Processes - Entrained Road Dust - Unpaved					<1	<1
Misc Processes - Unplanned Fires					<1	<1
Misc Processes - Waste Disposal						
Misc Processes - Natural Sources					<1	<1
Misc Processes - Other	<1		1	<1	1	
On Road Vehicles	67			61	2	<1
Other Mobile - Off Road Vehicles	<1			<1	<1	<1
Other Mobile - Trains	<1				<1	<1
Other Mobile - Ships	<1			<1		
Other Mobile - Aircraft - Government	4			<1		
Other Mobile - Aircraft - Other	<1			<1		
Other Mobile - Mobile Equipment	14			3		
Other Mobile - Utility Equipment	12			33		
Other Misc Sources			99		19	52
Total	100	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Nickel 7440-02-0	Arsenic 7440-38-2	Barium 7440-39-3	Cadmium 7440-43-9	Acetaldehyde 75-07-0
Fuel Combustion - Agricultural					
Fuel Combustion - Oil and Gas Production					
Fuel Combustion - Petroleum Refining					
Fuel Combustion - Other Manufacturing/Industrial					
Fuel Combustion - Electric Utilities	6	37	<1	2	
Fuel Combustion - Other Services and Commerce					
Fuel Combustion - Residential	2	<1	<1	<1	
Fuel Combustion - Other		1	<1		<1
Waste Burning - Agricultural Debris					
Waste Burning - Range Management					
Waste Burning - Forest Management					
Waste Burning - Incineration					
Waste Burning - Other	<1	<1	<1	1	
Solvent Use - Dry Cleaning	<1			<1	
Solvent Use - Degreasing					
Solvent Use - Architectural Coating					
Solvent Use - Other Surface Coating					
Solvent Use - Asphalt Paving					
Solvent Use - Printing					
Solvent Use - Domestic					
Solvent Use - Industrial					
Solvent Use - Other					
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction					
Petroleum Processes, Storage & Transfer - Petroleum Refining					
Petroleum Processes, Storage & Transfer - Petroleum Marketing					
Petroleum Processes, Storage & Transfer - Other					
Industrial Processes - Chemical					
Industrial Processes - Food and Agricultural					
Industrial Processes - Mineral Processes					

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Nickel 7440-02-0	Arsenic 7440-38-2	Barium 7440-39-3	Cadmium 7440-43-9	Acetaldehyde 75-07-0
Industrial Processes - Metal Processes					
Industrial Processes - Wood and Paper					
Industrial Processes - Other					
Misc Processes - Pesticide Application					
Misc Processes - Farming Operations	<1	<1	<1	<1	
Misc Processes - Construction and Demolition					
Misc Processes - Entrained Road Dust - Paved	48	26	48	59	
Misc Processes - Entrained Road Dust - Unpaved	<1	1	<1	<1	
Misc Processes - Unplanned Fires					
Misc Processes - Waste Disposal					
Misc Processes - Natural Sources	<1	<1	<1	<1	
Misc Processes - Other	3				<1
On Road Vehicles	<1	3	3	9	84
Other Mobile - Off Road Vehicles	<1	<1	<1	<1	<1
Other Mobile - Trains	<1		<1		<1
Other Mobile - Ships					<1
Other Mobile - Aircraft - Government					5
Other Mobile - Aircraft - Other					<1
Other Mobile - Mobile Equipment					4
Other Mobile - Utility Equipment					7
Other Misc Sources	39	31	47	27	
Total	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Dichloromethane 75-09-2	Ethylene Oxide 75-21-8	Trichloroethylene 79-01-6
Fuel Combustion - Agricultural			
Fuel Combustion - Oil and Gas Production			
Fuel Combustion - Petroleum Refining			
Fuel Combustion - Other Manufacturing/Industrial			
Fuel Combustion - Electric Utilities			
Fuel Combustion - Other Services and Commerce			
Fuel Combustion - Residential			
Fuel Combustion - Other			
Waste Burning - Agricultural Debris			
Waste Burning - Range Management			
Waste Burning - Forest Management			
Waste Burning - Incineration			
Waste Burning - Other		100	
Solvent Use - Dry Cleaning			
Solvent Use - Degreasing			
Solvent Use - Architectural Coating	24		89
Solvent Use - Other Surface Coating	8		
Solvent Use - Asphalt Paving			
Solvent Use - Printing			
Solvent Use - Domestic	<1		
Solvent Use - Industrial	11		
Solvent Use - Other	53		11
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction			
Petroleum Processes, Storage & Transfer - Petroleum Refining			
Petroleum Processes, Storage & Transfer - Petroleum Marketing			
Petroleum Processes, Storage & Transfer - Other			
Industrial Processes - Chemical			
Industrial Processes - Food and Agricultural			
Industrial Processes - Mineral Processes			

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Tucson (%) (Continued)

Description	Dichloromethane 75-09-2	Ethylene Oxide 75-21-8	Trichloroethylene 79-01-6
Industrial Processes - Metal Processes			
Industrial Processes - Wood and Paper			
Industrial Processes - Other			
Misc Processes - Pesticide Application			
Misc Processes - Farming Operations			
Misc Processes - Construction and Demolition			
Misc Processes - Entrained Road Dust - Paved			
Misc Processes - Entrained Road Dust - Unpaved			
Misc Processes - Unplanned Fires			
Misc Processes - Waste Disposal			
Misc Processes - Natural Sources			
Misc Processes - Other			
On Road Vehicles			
Other Mobile - Off Road Vehicles			
Other Mobile - Trains			
Other Mobile - Ships			
Other Mobile - Aircraft - Government			
Other Mobile - Aircraft - Other			
Other Mobile - Mobile Equipment			
Other Mobile - Utility Equipment			
Other Misc Sources	3		
Total	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

CASA GRANDE STUDY AREA

Distribution of HAP Emissions for Casa Grande (%)

Description	1,4-Dichlorobenzene 106-46-7	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Toluene 108-88-3
Fuel Combustion - Agricultural				
Fuel Combustion - Oil and Gas Production				
Fuel Combustion - Petroleum Refining				
Fuel Combustion - Other Manufacturing/Industrial			<1	<1
Fuel Combustion - Electric Utilities				<1
Fuel Combustion - Other Services and Commerce				
Fuel Combustion - Residential				<1
Fuel Combustion - Other		1		
Waste Burning - Agricultural Debris				
Waste Burning - Range Management				
Waste Burning - Forest Management				
Waste Burning - Incineration				
Waste Burning - Other			2	<1
Solvent Use - Dry Cleaning		<1		
Solvent Use - Degreasing				<1
Solvent Use - Architectural Coating				
Solvent Use - Other Surface Coating				<1
Solvent Use - Asphalt Paving				9
Solvent Use - Printing				<1
Solvent Use - Domestic				<1
Solvent Use - Industrial	31			1
Solvent Use - Other	69			
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				
Petroleum Processes, Storage & Transfer - Petroleum Refining				
Petroleum Processes, Storage & Transfer - Petroleum Marketing				<1
Petroleum Processes, Storage & Transfer - Other				<1
Industrial Processes - Chemical				<1
Industrial Processes - Food and Agricultural				

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	1,4-Dichlorobenzene 106-46-7	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Toluene 108-88-3
Industrial Processes - Mineral Processes				
Industrial Processes - Metal Processes				
Industrial Processes - Wood and Paper				
Industrial Processes - Other				
Misc Processes - Pesticide Application				
Misc Processes - Farming Operations				<1
Misc Processes - Construction and Demolition				
Misc Processes - Entrained Road Dust - Paved				
Misc Processes - Entrained Road Dust - Unpaved				
Misc Processes - Unplanned Fires		<1		
Misc Processes - Waste Disposal				
Misc Processes - Natural Sources				
Misc Processes - Other				35
On Road Vehicles		60	89	45
Other Mobile - Off Road Vehicles		1	<1	<1
Other Mobile - Trains				
Other Mobile - Ships		<1	<1	<1
Other Mobile - Aircraft - Government				
Other Mobile - Aircraft - Other		<1	6	<1
Other Mobile - Mobile Equipment		3		<1
Other Mobile - Utility Equipment		34	2	6
Other Misc Sources				
Total	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Hexavalent Chromium 18540-29-9	Trimethylbenzene 25551-13-7	Formaldehyde 50-00-0	Benzo(a)pyrene 50-32-8
Fuel Combustion - Agricultural				
Fuel Combustion - Oil and Gas Production				
Fuel Combustion - Petroleum Refining				
Fuel Combustion - Other Manufacturing/Industrial			<1	10
Fuel Combustion - Electric Utilities		<1	2	
Fuel Combustion - Other Services and Commerce				
Fuel Combustion - Residential		<1	<1	
Fuel Combustion - Other			1	9
Waste Burning - Agricultural Debris				
Waste Burning - Range Management				
Waste Burning - Forest Management				
Waste Burning - Incineration				
Waste Burning - Other			4	81
Solvent Use - Dry Cleaning				<1
Solvent Use - Degreasing		4		
Solvent Use - Architectural Coating				
Solvent Use - Other Surface Coating				
Solvent Use - Asphalt Paving		5		
Solvent Use - Printing		80		
Solvent Use - Domestic		10		
Solvent Use - Industrial				
Solvent Use - Other				
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				
Petroleum Processes, Storage & Transfer - Petroleum Refining				
Petroleum Processes, Storage & Transfer - Petroleum Marketing				
Petroleum Processes, Storage & Transfer - Other				
Industrial Processes - Chemical				
Industrial Processes - Food and Agricultural				
Industrial Processes - Mineral Processes				

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Hexavalent Chromium 18540-29-9	Trimethylbenzene 25551-13-7	Formaldehyde 50-00-0	Benzo(a)pyrene 50-32-8
Industrial Processes - Metal Processes				
Industrial Processes - Wood and Paper				
Industrial Processes - Other				
Misc Processes - Pesticide Application				
Misc Processes - Farming Operations				
Misc Processes - Construction and Demolition				
Misc Processes - Entrained Road Dust - Paved				
Misc Processes - Entrained Road Dust - Unpaved				
Misc Processes - Unplanned Fires				
Misc Processes - Waste Disposal				
Misc Processes - Natural Sources				
Misc Processes - Other	10			
On Road Vehicles			74	
Other Mobile - Off Road Vehicles			<1	
Other Mobile - Trains			<1	
Other Mobile - Ships			<1	
Other Mobile - Aircraft - Government				
Other Mobile - Aircraft - Other			1	
Other Mobile - Mobile Equipment			11	
Other Mobile - Utility Equipment			5	
Other Misc Sources	90			
Total	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5	Nickel 7440-02-0	Arsenic 7440-38-2	Barium 7440-39-3
Fuel Combustion - Agricultural							
Fuel Combustion - Oil and Gas Production							
Fuel Combustion - Petroleum Refining		<1					
Fuel Combustion - Other Manufacturing/Industrial		<1	<1	<1	8	31	<1
Fuel Combustion - Electric Utilities							
Fuel Combustion - Other Services and Commerce		<1	<1	<1	<1	<1	<1
Fuel Combustion - Residential		<1	<1	<1		<1	<1
Fuel Combustion - Other							
Waste Burning - Agricultural Debris							
Waste Burning - Range Management							
Waste Burning - Forest Management							
Waste Burning - Incineration		<1	2	<1	<1	<1	<1
Waste Burning - Other			<1	2	<1		
Solvent Use - Dry Cleaning							
Solvent Use - Degreasing							
Solvent Use - Architectural Coating		<1					
Solvent Use - Other Surface Coating							
Solvent Use - Asphalt Paving							
Solvent Use - Printing							
Solvent Use - Domestic							
Solvent Use - Industrial							
Solvent Use - Other							
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction							
Petroleum Processes, Storage & Transfer - Petroleum Refining		<1					
Petroleum Processes, Storage & Transfer - Petroleum Marketing		<1					
Petroleum Processes, Storage & Transfer - Other		<1					
Industrial Processes - Chemical							
Industrial Processes - Food and Agricultural							
Industrial Processes - Mineral Processes							

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5	Nickel 7440-02-0	Arsenic 7440-38-2	Barium 7440-39-3
Industrial Processes - Metal Processes							
Industrial Processes - Wood and Paper							
Industrial Processes - Other							
Misc Processes - Pesticide Application							
Misc Processes - Farming Operations		5	2	14	8	10	10
Misc Processes - Construction and Demolition							
Misc Processes - Entrained Road Dust - Paved			65	58	60	20	63
Misc Processes - Entrained Road Dust - Unpaved			9	12	9	22	13
Misc Processes - Unplanned Fires			<1	<1			
Misc Processes - Waste Disposal							
Misc Processes - Natural Sources			<1	1	<1	<1	1
Misc Processes - Other	2	<1					
On Road Vehicles		71	16	8	8	10	8
Other Mobile - Off Road Vehicles		<1	2	<1	<1	<1	<1
Other Mobile - Trains			1	<1	<1		<1
Other Mobile - Ships		<1					
Other Mobile - Aircraft - Government							
Other Mobile - Aircraft - Other		<1					
Other Mobile - Mobile Equipment		3					
Other Mobile - Utility Equipment		17					
Other Misc Sources	98	<1	2	4	5	4	4
Total	100	100	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Cadmium 7440-43-9	Acetaldehyde 75-07-0	Dichloromethane 75-09-2	Trichloroethylene 79-01-6	Trifluralin 1582-09-8
Fuel Combustion - Agricultural					
Fuel Combustion - Oil and Gas Production					
Fuel Combustion - Petroleum Refining					
Fuel Combustion - Other Manufacturing/Industrial		<1			
Fuel Combustion - Electric Utilities	1				
Fuel Combustion - Other Services and Commerce					
Fuel Combustion - Residential	<1				
Fuel Combustion - Other		<1			
Waste Burning - Agricultural Debris					
Waste Burning - Range Management					
Waste Burning - Forest Management					
Waste Burning - Incineration					
Waste Burning - Other	<1	2			
Solvent Use - Dry Cleaning	3				
Solvent Use - Degreasing					
Solvent Use - Architectural Coating			29	90	
Solvent Use - Other Surface Coating			7		
Solvent Use - Asphalt Paving					
Solvent Use - Printing					
Solvent Use - Domestic					
Solvent Use - Industrial			9		
Solvent Use - Other			55	10	
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction					
Petroleum Processes, Storage & Transfer - Petroleum Refining					
Petroleum Processes, Storage & Transfer - Petroleum Marketing					
Petroleum Processes, Storage & Transfer - Other					
Industrial Processes - Chemical					
Industrial Processes - Food and Agricultural					

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Cadmium 7440-43-9	Acetaldehyde 75-07-0	Dichloromethane 75-09-2	Trichloroethylene 79-01-6	Trifluralin 1582-09-8
Industrial Processes - Mineral Processes					
Industrial Processes - Metal Processes					
Industrial Processes - Wood and Paper					
Industrial Processes - Other					
Misc Processes - Pesticide Application					
Misc Processes - Farming Operations	13				100
Misc Processes - Construction and Demolition					
Misc Processes - Entrained Road Dust - Paved	46				
Misc Processes - Entrained Road Dust - Unpaved	8				
Misc Processes - Unplanned Fires					
Misc Processes - Waste Disposal					
Misc Processes - Natural Sources	<1				
Misc Processes - Other					
On Road Vehicles	25	89			
Other Mobile - Off Road Vehicles	<1	<1			
Other Mobile - Trains		<1			
Other Mobile - Ships		<1			
Other Mobile - Aircraft - Government					
Other Mobile - Aircraft - Other		1			
Other Mobile - Mobile Equipment		3			
Other Mobile - Utility Equipment		3			
Other Misc Sources	3				
Total	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Chlorpyrifos 2921-88-2	Methyl Parathion 298-00-0	Disulfoton 298-04-4	Ammonia 7664-41-7
Fuel Combustion - Agricultural				
Fuel Combustion - Oil and Gas Production				
Fuel Combustion - Petroleum Refining				
Fuel Combustion - Other Manufacturing/Industrial				
Fuel Combustion - Electric Utilities				
Fuel Combustion - Other Services and Commerce				
Fuel Combustion - Residential				
Fuel Combustion - Other				
Waste Burning - Agricultural Debris				
Waste Burning - Range Management				
Waste Burning - Forest Management				
Waste Burning - Incineration				
Waste Burning - Other				
Solvent Use - Dry Cleaning				
Solvent Use - Degreasing				
Solvent Use - Architectural Coating				
Solvent Use - Other Surface Coating				
Solvent Use - Asphalt Paving				
Solvent Use - Printing				
Solvent Use - Domestic				
Solvent Use - Industrial				
Solvent Use - Other				
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				
Petroleum Processes, Storage & Transfer - Petroleum Refining				
Petroleum Processes, Storage & Transfer - Petroleum Marketing				
Petroleum Processes, Storage & Transfer - Other				
Industrial Processes - Chemical				
Industrial Processes - Food and Agricultural				
Industrial Processes - Mineral Processes				

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Casa Grande (%) (Continued)

Description	Chlorpyrifos 2921-88-2	Methyl Parathion 298-00-0	Disulfoton 298-04-4	Ammonia 7664-41-7
Industrial Processes - Metal Processes				
Industrial Processes - Wood and Paper				
Industrial Processes - Other				
Misc Processes - Pesticide Application				
Misc Processes - Farming Operations	100	100	100	
Misc Processes - Construction and Demolition				
Misc Processes - Entrained Road Dust - Paved				
Misc Processes - Entrained Road Dust - Unpaved				
Misc Processes - Unplanned Fires				
Misc Processes - Waste Disposal				
Misc Processes - Natural Sources				
Misc Processes - Other				100
On Road Vehicles				
Other Mobile - Off Road Vehicles				
Other Mobile - Trains				
Other Mobile - Ships				
Other Mobile - Aircraft - Government				
Other Mobile - Aircraft - Other				
Other Mobile - Mobile Equipment				
Other Mobile - Utility Equipment				
Other Misc Sources				
Total	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research report shows the emissions magnitude in tons per year for each compound of interest.

PAYSON STUDY AREA

Distribution of HAP Emissions for Payson (%)

Description	1,4-Dichlorobenzene 106-46-7	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Toluene 108-88-3	Hexane 110-54-3
Fuel Combustion - Agricultural					
Fuel Combustion - Oil and Gas Production					
Fuel Combustion - Petroleum Refining					
Fuel Combustion - Other Manufacturing/Industrial					
Fuel Combustion - Electric Utilities				<1	<1
Fuel Combustion - Other Services and Commerce					
Fuel Combustion - Residential				<1	<1
Fuel Combustion - Other		50			
Waste Burning - Agricultural Debris					
Waste Burning - Range Management					
Waste Burning - Forest Management					
Waste Burning - Incineration					
Waste Burning - Other					2
Solvent Use - Dry Cleaning		3			3
Solvent Use - Degreasing				<1	
Solvent Use - Architectural Coating					
Solvent Use - Other Surface Coating				<1	3
Solvent Use - Asphalt Paving				5	
Solvent Use - Printing				<1	
Solvent Use - Domestic				<1	
Solvent Use - Industrial	100			1	
Solvent Use - Other					
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction					
Petroleum Processes, Storage & Transfer - Petroleum Refining					
Petroleum Processes, Storage & Transfer - Petroleum Marketing					
Petroleum Processes, Storage & Transfer - Other				<1	1
Industrial Processes - Chemical				<1	<1

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	1,4-Dichlorobenzene 106-46-7	1,3-Butadiene 106-99-0	Acrolein 107-02-8	Toluene 108-88-3	Hexane 110-54-3
Industrial Processes - Food and Agricultural					
Industrial Processes - Mineral Processes					
Industrial Processes - Metal Processes					
Industrial Processes - Wood and Paper					
Industrial Processes - Other					
Misc Processes - Pesticide Application					
Misc Processes - Farming Operations				<1	<1
Misc Processes - Construction and Demolition					
Misc Processes - Entrained Road Dust - Paved					
Misc Processes - Entrained Road Dust - Unpaved					
Misc Processes - Unplanned Fires		1			
Misc Processes - Waste Disposal					
Misc Processes - Natural Sources					
Misc Processes - Other					
On Road Vehicles		32	97	83	86
Other Mobile - Off Road Vehicles		<1	<1	<1	<1
Other Mobile - Trains					
Other Mobile - Ships		<1	<1	<1	<1
Other Mobile - Aircraft - Government					
Other Mobile - Aircraft - Other		<1	1	<1	
Other Mobile - Mobile Equipment		<1		<1	<1
Other Mobile - Utility Equipment		13	1	9	4
Other Misc Sources				<1	<1
Total	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Perchloroethylene 127-18-4	Hexavalent Chromium 18540-29-9	Chrysene 218-01-9	Trimethylbenzene 25551-13-7
Fuel Combustion - Agricultural				
Fuel Combustion - Oil and Gas Production				
Fuel Combustion - Petroleum Refining				
Fuel Combustion - Other Manufacturing/Industrial				
Fuel Combustion - Electric Utilities				<1
Fuel Combustion - Other Services and Commerce				
Fuel Combustion - Residential				
Fuel Combustion - Other			98	
Waste Burning - Agricultural Debris				
Waste Burning - Range Management				
Waste Burning - Forest Management				
Waste Burning - Incineration				
Waste Burning - Other				
Solvent Use - Dry Cleaning			2	
Solvent Use - Degreasing	78			8
Solvent Use - Architectural Coating	22			
Solvent Use - Other Surface Coating				
Solvent Use - Asphalt Paving				2
Solvent Use - Printing				86
Solvent Use - Domestic				4
Solvent Use - Industrial				
Solvent Use - Other				
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				
Petroleum Processes, Storage & Transfer - Petroleum Refining				
Petroleum Processes, Storage & Transfer - Petroleum Marketing				
Petroleum Processes, Storage & Transfer - Other				
Industrial Processes - Chemical				
Industrial Processes - Food and Agricultural				
Industrial Processes - Mineral Processes				

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Perchloroethylene 127-18-4	Hexavalent Chromium 18540-29-9	Chrysene 218-01-9	Trimethylbenzene 25551-13-7
Industrial Processes - Metal Processes				
Industrial Processes - Wood and Paper				
Industrial Processes - Other				
Misc Processes - Pesticide Application				
Misc Processes - Farming Operations				
Misc Processes - Construction and Demolition				
Misc Processes - Entrained Road Dust - Paved				
Misc Processes - Entrained Road Dust - Unpaved				
Misc Processes - Unplanned Fires				
Misc Processes - Waste Disposal				
Misc Processes - Natural Sources				
Misc Processes - Other				
On Road Vehicles				
Other Mobile - Off Road Vehicles				
Other Mobile - Trains				
Other Mobile - Ships				
Other Mobile - Aircraft - Government				
Other Mobile - Aircraft - Other				
Other Mobile - Mobile Equipment				
Other Mobile - Utility Equipment				
Other Misc Sources	<1	100		
Total	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Formaldehyde 50-00-0	Benzo(a)pyrene 50-32-8	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5	Nickel 7440-02-0
Fuel Combustion - Agricultural							
Fuel Combustion - Oil and Gas Production							
Fuel Combustion - Petroleum Refining							
Fuel Combustion - Other Manufacturing/Industrial	<1			<1	<1	<1	3
Fuel Combustion - Electric Utilities							
Fuel Combustion - Other Services and Commerce	<1			<1	<1	<1	<1
Fuel Combustion - Residential	55	96		48	<1	<1	
Fuel Combustion - Other							
Waste Burning - Agricultural Debris							
Waste Burning - Range Management							
Waste Burning - Forest Management							
Waste Burning - Incineration				<1	<1	<1	<1
Waste Burning - Other		4			<1	2	1
Solvent Use - Dry Cleaning							
Solvent Use - Degreasing							
Solvent Use - Architectural Coating				<1			
Solvent Use - Other Surface Coating							
Solvent Use - Asphalt Paving							
Solvent Use - Printing							
Solvent Use - Domestic							
Solvent Use - Industrial							
Solvent Use - Other							
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction							
Petroleum Processes, Storage & Transfer - Petroleum Refining							
Petroleum Processes, Storage & Transfer - Petroleum Marketing				<1			
Petroleum Processes, Storage & Transfer - Other				<1			
Industrial Processes - Chemical							

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Formaldehyde 50-00-0	Benzo(a)pyrene 50-32-8	Chloroform 67-66-3	Benzene 71-43-2	Lead 7439-92-1	Manganese 7439-96-5	Nickel 7440-02-0
Industrial Processes - Food and Agricultural							
Industrial Processes - Mineral Processes							
Industrial Processes - Metal Processes							
Industrial Processes - Wood and Paper							
Industrial Processes - Other							
Misc Processes - Pesticide Application							
Misc Processes - Farming Operations				<1			
Misc Processes - Construction and Demolition							
Misc Processes - Entrained Road Dust - Paved					81	79	83
Misc Processes - Entrained Road Dust - Unpaved					4	6	5
Misc Processes - Unplanned Fires					<1	3	
Misc Processes - Waste Disposal							
Misc Processes - Natural Sources					<1	<1	<1
Misc Processes - Other							
On Road Vehicles	39			43	9	5	5
Other Mobile - Off Road Vehicles	<1			<1	<1	<1	<1
Other Mobile - Trains	<1				<1	<1	<1
Other Mobile - Ships	<1			<1			
Other Mobile - Aircraft - Government							
Other Mobile - Aircraft - Other	<1			<1			
Other Mobile - Mobile Equipment	3			<1			
Other Mobile - Utility Equipment	2			8			
Other Misc Sources			100	<1	2	4	3
Total	100	100	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Arsenic 7440-38-2	Barium 7440-39-3	Cadmium 7440-43-9	Acetaldehyde 75-07-0	Dichloromethane 75-09-2
Fuel Combustion - Agricultural					
Fuel Combustion - Oil and Gas Production					
Fuel Combustion - Petroleum Refining					
Fuel Combustion - Other Manufacturing/Industrial	15	<1	<1		
Fuel Combustion - Electric Utilities					
Fuel Combustion - Other Services and Commerce	<1	<1	<1		
Fuel Combustion - Residential	13	<2		26	
Fuel Combustion - Other					
Waste Burning - Agricultural Debris					
Waste Burning - Range Management					
Waste Burning - Forest Management					
Waste Burning - Incineration	<1	<1	<1		
Waste Burning - Other			38		
Solvent Use - Dry Cleaning					
Solvent Use - Degreasing					24
Solvent Use - Architectural Coating					9
Solvent Use - Other Surface Coating					
Solvent Use - Asphalt Paving					
Solvent Use - Printing					
Solvent Use - Domestic					12
Solvent Use - Industrial					55
Solvent Use - Other					
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction					
Petroleum Processes, Storage & Transfer - Petroleum Refining					
Petroleum Processes, Storage & Transfer - Petroleum Marketing					

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Arsenic 7440-38-2	Barium 7440-39-3	Cadmium 7440-43-9	Acetaldehyde 75-07-0	Dichloromethane 75-09-2
Petroleum Processes, Storage & Transfer - Other					
Industrial Processes - Chemical					
Industrial Processes - Food and Agricultural					
Industrial Processes - Mineral Processes					
Industrial Processes - Metal Processes					
Industrial Processes - Wood and Paper					
Industrial Processes - Other					
Misc Processes - Pesticide Application					
Misc Processes - Farming Operations					
Misc Processes - Construction and Demolition					
Misc Processes - Entrained Road Dust - Paved	42	82	46		
Misc Processes - Entrained Road Dust - Unpaved	18	6	3		
Misc Processes - Unplanned Fires					
Misc Processes - Waste Disposal					
Misc Processes - Natural Sources	<1	<1	<1		
Misc Processes - Other					
On Road Vehicles	10	5	12	70	
Other Mobile - Off Road Vehicles	<1	<1	<1	<1	
Other Mobile - Trains		<1		<1	
Other Mobile - Ships				<1	
Other Mobile - Aircraft - Government					
Other Mobile - Aircraft - Other				<1	
Other Mobile - Mobile Equipment				1	
Other Mobile - Utility Equipment				2	
Other Misc Sources	3	2	1		
Total	100	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Ethylene Oxide 75-21-8	Trichloroethylene 79-01-6	Naphthalene 91-20-3	Polycyclic Organic Matter (POM)
Fuel Combustion - Agricultural				
Fuel Combustion - Oil and Gas Production				
Fuel Combustion - Petroleum Refining				
Fuel Combustion - Other Manufacturing/Industrial			<1	
Fuel Combustion - Electric Utilities				
Fuel Combustion - Other Services and Commerce				
Fuel Combustion - Residential			95	94
Fuel Combustion - Other				
Waste Burning - Agricultural Debris				
Waste Burning - Range Management				
Waste Burning - Forest Management				
Waste Burning - Incineration	100			
Waste Burning - Other				
Solvent Use - Dry Cleaning			<1	
Solvent Use - Degreasing		89		
Solvent Use - Architectural Coating				
Solvent Use - Other Surface Coating			<1	
Solvent Use - Asphalt Paving				
Solvent Use - Printing				
Solvent Use - Domestic				
Solvent Use - Industrial		11		
Solvent Use - Other				
Petroleum Processes, Storage & Transfer - Oil and Gas Extraction				
Petroleum Processes, Storage & Transfer - Petroleum Refining				
Petroleum Processes, Storage & Transfer - Petroleum Marketing			<1	
Petroleum Processes, Storage & Transfer - Other			<1	
Industrial Processes - Chemical				
Industrial Processes - Food and Agricultural				

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

Distribution of HAP Emissions for Payson (%) (Continued)

Description	Ethylene Oxide 75-21-8	Trichloroethylene 79-01-6	Naphthalene 91-20-3	Polycyclic Organic Matter (POM)
Industrial Processes - Mineral Processes				
Industrial Processes - Metal Processes				
Industrial Processes - Wood and Paper				
Industrial Processes - Other				
Misc Processes - Pesticide Application				
Misc Processes - Farming Operations				
Misc Processes - Construction and Demolition				
Misc Processes - Entrained Road Dust - Paved				
Misc Processes - Entrained Road Dust - Unpaved				
Misc Processes - Unplanned Fires				
Misc Processes - Waste Disposal				
Misc Processes - Natural Sources				
Misc Processes - Other				
On Road Vehicles				
Other Mobile - Off Road Vehicles			5	6
Other Mobile - Trains				
Other Mobile - Ships				
Other Mobile - Aircraft - Government				
Other Mobile - Aircraft - Other				
Other Mobile - Mobile Equipment			<1	
Other Mobile - Utility Equipment			<1	<1
Other Misc Sources			<1	<1
Total	100	100	100	100

This table illustrates on a percentage basis the distribution of emissions by source type, which does not necessarily correspond to human health risk. Table 3-5 in this volume of the HAPs research program report shows the emissions magnitude in tons per year for each compound of interest.

APPENDIX H

APPLICATION OF THE CHEMICAL MASS BALANCE RECEPTOR MODEL TO APPORTIONMENT OF HAPS

INTRODUCTION

As part of the HAPS research program, a statewide survey is being conducted by ADEQ and the Desert Research Institute (DRI) to determine the concentrations of HAPs in the ambient air in Arizona. This appendix applies the Chemical Mass Balance (CMB) receptor model to these data to estimate the contribution of major sources to ambient concentrations of HAP species. This present analysis is intended to provide a qualitative evaluation of the emissions inventory data.

Monitoring for ambient concentrations of HAPs was conducted over a one-year period at a residential site in central Phoenix. Samples were collected every sixth day, in parallel with the PM10 sampling schedule, from April 1994 to March 1995. In addition to Phoenix, residential neighborhood measurements were also initiated at Payson in January 1995, and in Casa Grande and Tucson in April 1995. Payson was specifically selected in order to examine the contribution of woodsmoke-related HAPS. Sampling at these three additional sites will be continue for a full year. Further details about the sampling locations and site characteristics were provided in Volume 1 of this report. Samples were also collected every twelve days at a background site on a mountain top at 1400 m elevation, near the town of Hillside, which is located about 80 miles north of Phoenix.

Ambient samples were also collected in order to characterize the chemical composition of three of the major sources of HAPs. An existing monitoring site near the intersection of Indian School Road and 33th Avenue was used to characterize motor-vehicle emission. Samples were collected at this site during both summer and winter in order to develop seasonal-specific profiles. Diesel exhaust profiles were developed from samples collected at a truck stop just west of the town of Buckeye, and woodsmoke profiles were derived from ambient measurements at Payson.

The Research HAPS List contains approximately 600 compounds. These HAPs are remarkably diverse, consisting of industrial chemicals and intermediates, pesticides, chlorinated and hydrocarbon solvents, metals, combustion byproducts, etc. Some of the HAPs are not single compounds but rather complex mixtures or chemical groups spanning a broad range of chemical and physical properties, such as, for example, polycyclic aromatic hydrocarbons (PAH). Some of the HAPs are common air pollutants that were assigned to the list based on their recognized toxicity in the workplace environment. Some HAPs are unlikely to exist in ambient air because of their chemical reactivities. Clearly, it was neither possible nor desirable to monitor all compounds on the Arizona HAPs list. Based on our previous experience, the HAPs that are most likely to be present in ambient air in Arizona were selected for monitoring. Table 1 lists these compounds classified according to compound type (hydrocarbons, halogenated hydrocarbons, carbonyl compounds and PAH), and listed in order of decreasing volatility.

Table 1
Source Profiles for Arizona HAP Research Program

Species	Diesel Truck wt%	Auto Exhaust Summer wt%	Auto Exhaust Winter wt%	Wood Smoke wt%
		Hydrocarbon Compounds		
C2CMPD	9.5113 ± 3.5478	6.9164 ± 1.5612	7.7645 ± 0.8351	5.9547 ± 7.6109
PROPE	2.2769 ± 0.8663	0.9724 ± 0.1574	1.3926 ± 0.0944	0.0000 ± 0.3822
N_PROP	0.0000 ± 0.0004	0.0000 ± 0.0002	0.0000 ± 0.0001	0.0041 ± 3.9877
I_BUTA	1.3172 ± 0.4874	0.7058 ± 0.1257	1.0749 ± 0.0727	7.7510 ± 3.1333
BEABYL	1.2561 ± 0.5469	0.6818 ± 0.0871	1.1554 ± 0.0648	0.3210 ± 0.4927
BUDI13	0.4587 ± 0.2399	0.2375 ± 0.0361	0.3692 ± 0.0232	0.5139 ± 0.3868
N_BUTA	2.7122 ± 1.4424	2.5485 ± 0.3784	3.1293 ± 0.1875	5.1522 ± 4.6474
T2BUTE	0.1457 ± 0.0958	0.2154 ± 0.0964	0.3718 ± 0.0223	0.2641 ± 0.3839
C2BUTE	0.0935 ± 0.0638	0.2278 ± 0.0266	0.2539 ± 0.0094	0.1649 ± 0.3176
B1E3ME	0.0614 ± 0.0455	0.1036 ± 0.0465	0.1125 ± 0.0059	0.0998 ± 0.1120
IPENTA	3.0482 ± 1.5296	7.1333 ± 0.8819	6.9577 ± 0.3578	0.0000 ± 1.1271
PENTE1	1.1525 ± 0.9647	0.1431 ± 0.0689	0.1612 ± 0.0525	0.4935 ± 0.1623
N_PENT	1.3425 ± 0.5787	3.8021 ± 0.4161	3.6525 ± 0.2679	0.0000 ± 0.9045
I_PREN	0.4816 ± 0.2462	0.0000 ± 0.0163	0.1422 ± 0.0277	1.5589 ± 0.5202
T2PENE	0.0815 ± 0.0766	0.5507 ± 0.1760	0.6321 ± 0.0590	0.7437 ± 0.6009
C2PENE	0.0866 ± 0.0511	0.2848 ± 0.0423	0.2891 ± 0.0199	0.0000 ± 0.1193
B2E2M	0.3771 ± 0.3794	0.5709 ± 0.1022	0.7189 ± 0.0458	0.0000 ± 0.1982
BU22DM	0.1093 ± 0.0893	0.2433 ± 0.0442	0.2410 ± 0.0223	0.0000 ± 0.1058
CPENTE	0.1518 ± 0.2335	0.0881 ± 0.0410	0.1300 ± 0.0131	0.0000 ± 0.0744
BU23DM	0.3606 ± 0.2743	0.6701 ± 0.0892	0.8118 ± 0.0652	0.0000 ± 0.2391
PENA2M	1.2474 ± 0.5771	2.8436 ± 0.4479	2.7589 ± 0.1894	0.0000 ± 0.6194
PENA3M	1.6733 ± 0.6357	1.5978 ± 0.2224	1.6927 ± 0.0794	0.5203 ± 0.4176
P1E2ME	0.0364 ± 0.0792	0.1057 ± 0.0484	0.1306 ± 0.0106	0.0000 ± 0.0725
N_HEX	1.2212 ± 0.7027	4.3132 ± 1.4695	2.3858 ± 0.7858	0.0000 ± 2.0482
T2HEXE	0.0494 ± 0.1131	0.1037 ± 0.0490	0.1649 ± 0.0182	0.1862 ± 0.1076
C2HEXE	0.0777 ± 0.1593	0.0431 ± 0.0372	0.0822 ± 0.0093	0.0448 ± 0.0729
MCYPNA	0.6761 ± 0.2734	1.6212 ± 0.2728	1.6392 ± 0.0723	0.0000 ± 0.2548
PEN24M	0.2702 ± 0.2107	0.6509 ± 0.0910	0.8425 ± 0.0720	0.4758 ± 0.3378
BENZE	2.1308 ± 0.8480	2.7197 ± 0.3454	3.2135 ± 0.1908	3.5348 ± 1.1992
CYHEXA	0.5020 ± 0.1851	0.3574 ± 0.0393	0.5096 ± 0.0377	0.5794 ± 0.4091
HEXA2M	0.3974 ± 0.1336	1.1085 ± 0.0813	1.2696 ± 0.0578	0.0000 ± 0.2412
PEN23M	0.4528 ± 0.1254	1.1648 ± 0.1374	1.4487 ± 0.1018	0.4002 ± 0.3373
HEXA3M	1.2486 ± 0.6730	1.2350 ± 0.0932	1.3457 ± 0.0646	0.0499 ± 0.3257
PA224M	0.7860 ± 0.2449	1.5974 ± 0.1602	2.1195 ± 0.1738	1.5590 ± 0.5550
N_HEPT	0.3897 ± 0.2079	1.0919 ± 0.3553	1.1531 ± 0.0768	0.1328 ± 0.2971
MECYHX	0.8868 ± 0.3627	1.0244 ± 0.4818	0.9190 ± 0.1058	0.2632 ± 0.5727

Table 1
Source Profiles for Arizona HAP Research Program

Species	Diesel Truck wt%	Auto Exhaust Summer wt%	Auto Exhaust Winter wt%	Wood Smoke wt%
PA234M	0.1638 ± 0.1414	0.5192 ± 0.0795	0.7526 ± 0.0773	0.0120 ± 0.2640
TOLUE	4.0922 ± 1.1696	9.8873 ± 3.0518	9.8304 ± 0.3855	0.0000 ± 1.1673
HEP2ME	0.2085 ± 0.1235	0.3576 ± 0.1896	0.4457 ± 0.0150	0.0000 ± 0.1172
HEP3ME	0.2000 ± 0.0765	0.4856 ± 0.0746	0.5761 ± 0.0595	0.0405 ± 0.1897
N_OCT	0.5535 ± 0.2516	0.4597 ± 0.2227	0.4165 ± 0.0263	0.1438 ± 0.3215
ETBZ	0.6014 ± 0.1232	1.4123 ± 0.2196	1.6390 ± 0.0737	0.0791 ± 0.2539
MP_XYL	2.2426 ± 0.3064	5.5214 ± 1.0088	5.9526 ± 0.2223	0.2220 ± 0.6898
STYR	1.7961 ± 0.3562	0.2366 ± 0.1123	0.2639 ± 0.0657	1.3608 ± 0.2174
O_XYL	1.1329 ± 0.1723	1.9107 ± 0.2136	2.0893 ± 0.1246	0.1018 ± 0.3980
N_NON	0.5453 ± 0.1983	0.2266 ± 0.1350	0.2442 ± 0.0404	0.0501 ± 0.1526
IPRBZ	0.3555 ± 0.1478	0.0673 ± 0.0369	0.0478 ± 0.0375	0.4445 ± 0.2065
APINEN	0.0102 ± 0.0704	0.0000 ± 0.0148	0.0072 ± 0.0093	0.0746 ± 0.1356
N_PRBZ	0.3272 ± 0.1493	0.2809 ± 0.0576	0.3588 ± 0.0139	0.0302 ± 0.1167
BZ135M	0.3791 ± 0.0592	0.5822 ± 0.0659	0.8114 ± 0.0250	0.0000 ± 0.1456
BPINEN	0.4401 ± 0.4672	0.0268 ± 0.0393	0.0923 ± 0.1624	0.0000 ± 0.4418
BZ124M	1.8807 ± 0.6055	1.8490 ± 0.4025	1.8379 ± 0.8155	2.8651 ± 2.1534
N_DEC	0.9254 ± 0.2790	0.2293 ± 0.0894	0.2216 ± 0.1160	0.8566 ± 0.5965
N_UNDE	1.2759 ± 0.5269	0.2409 ± 0.1289	0.1053 ± 0.0862	1.3315 ± 0.8278
N_DODE	0.7507 ± 0.5982	0.1201 ± 0.0631	0.0230 ± 0.0344	0.5400 ± 0.1784
		Carbonyl Compounds		
FORMAL	-99	1.2437 ± 0.1866	1.2438 ± 0.1866	-99
ACETAL	-99	0.5653 ± 0.0848	0.5654 ± 0.0848	-99
ACETO	-99	0.5314 ± 0.0797	0.5315 ± 0.0797	-99
ACROLN	-99	0.0678 ± 0.0102	0.0678 ± 0.0102	-99
PROAL	-99	0.0678 ± 0.0102	0.0678 ± 0.0102	-99
CROTON	-99	0.0452 ± 0.0068	0.0452 ± 0.0068	-99
MEK	-99	0.1922 ± 0.0288	0.1922 ± 0.0288	-99
ACROLX	-99	-99	-99	-99
MACROL	-99	-99	-99	-99
BUTAL	-99	0.0791 ± 0.0119	0.0792 ± 0.0119	-99
BENZAL	-99	-99	-99	-99
VALAL	-99	-99	-99	-99
TOLUAL	-99	-99	-99	-99
HEXAL	-99	0.0113 ± 0.0017	0.0113 ± 0.0017	-99

Table 1
Source Profiles for Arizona HAP Research Program

Species	Diesel Truck wt%	Auto Exhaust Summer wt%	Auto Exhaust Winter wt%	Wood Smoke wt%
		PAH Compounds		
NAPHTH	0.2722 ± 0.1238	0.2251 ± 0.1718	0.1948 ± 0.0524	4.6761 ± 2.2922
MNAPH2	0.3401 ± 0.1823	0.2034 ± 0.1540	0.1143 ± 0.0362	2.4611 ± 1.2330
MNAPH1	0.1996 ± 0.1159	0.0632 ± 0.0493	0.0513 ± 0.0156	1.1442 ± 0.5417
DMN267	0.1833 ± 0.1215	0.0006 ± 0.0073	0.0064 ± 0.0066	0.2024 ± 0.1272
DM1367	0.3129 ± 0.2033	0.0000 ± 0.0122	0.0054 ± 0.0111	0.4319 ± 0.2378
D14523	0.1044 ± 0.0667	0.0000 ± 0.0040	0.0002 ± 0.0036	0.1478 ± 0.0572
DMN12	0.0449 ± 0.0328	0.0000 ± 0.0020	0.0000 ± 0.0018	0.0477 ± 0.0242
DMN18	0.0004 ± 0.0012	0.0015 ± 0.0035	0.0000 ± 0.0001	0.0042 ± 0.0066
BIPHEN	0.0920 ± 0.0546	0.0000 ± 0.0033	0.0000 ± 0.0030	0.1338 ± 0.0466
ACNAPY	0.0311 ± 0.0236	0.0000 ± 0.0014	0.0061 ± 0.0013	0.1661 ± 0.0632
ACNAPE	0.0105 ± 0.0073	0.0017 ± 0.0012	0.0010 ± 0.0004	0.0288 ± 0.0218
FLUORE	0.0613 ± 0.0392	0.0000 ± 0.0024	0.0000 ± 0.0021	0.1016 ± 0.0202
PHENAN	0.1150 ± 0.0785	0.0003 ± 0.0047	0.0000 ± 0.0043	0.1712 ± 0.0520
ANTHRA	0.0164 ± 0.0097	0.0001 ± 0.0006	0.0000 ± 0.0005	0.0287 ± 0.0080
FLUORA	0.0119 ± 0.0072	0.0036 ± 0.0018	0.0007 ± 0.0004	0.0537 ± 0.0180
PYRENE	0.0168 ± 0.0099	0.0009 ± 0.0006	0.0001 ± 0.0005	0.0537 ± 0.0185
RETENE	0.0016 ± 0.0029	0.0000 ± 0.0002	0.0000 ± 0.0002	0.0961 ± 0.0398
UNID	43.233 ± 9.1152	24.608 ± 1.8362	20.063 ± 2.3428	51.130 ± 19.917
TOTAL	99.991 ± 10.449	99.998 ± 4.5741	99.996 ± 2.8695	99.897 ± 22.970

METHODS AND APPROACH

Sampling and Analysis Methods. All samples were collected over 24-hour periods, starting at 10 a.m. Three different sampling methods were necessary due to the range of chemical and physical properties of HAPs selected for monitoring. Volatile hydrocarbons and halogenated hydrocarbons were collected using stainless steel SUMMA- canisters and analyzed by high resolution capillary gas chromatography with flame ionization detector and electron capture detector (Hewlett-Packard 5890 Series II).

Additional efforts were not made to resolve the C2 compounds since they are not HAPs species, and their mixing ratios are reported as the sum of C2 compounds. Carbonyl compounds were collected using C18 Sep-Pak cartridges impregnated with 2,4-dinitrophenylhydrazine (DNPH), and resulting hydrazones were eluted with HPLC grade acetonitrile and analyzed by HPLC (Waters, Inc.) with UV detection at 360 nm. PAHs were collected using Teflon-impregnated glass-fiber filters followed by PUF/XAD-4/PUF sandwich-type cartridges. Deuterated internal standards were added to each sample, and the filter-sorbent pairs were extracted together. The extracts were then analyzed by electron impact (EI) GC/MS technique, using a Hewlett-Packard 5890 GC equipped with a 7673A Automatic Sampler and interfaced to a 5970B Mass Selective Detector (MSD). Details of the above sampling and analysis methods and associated quality assurance procedures are given in Zielinska et al., (1995).

For this study, an uncertainty was estimated for each concentration value from the following formula

$$\sigma(c) = \sqrt{(2xmdl)^2 + (CVxc)^2}$$

where s(c) is the root mean square error for the concentration value (c), mdl is the minimum detection limit for the GC-FID method (0.2 ppbC), and CV is the coefficient of variation (± 10%). Ambient data near the detection limit are automatically given less weight in the CMB calculations because this formula assigns higher uncertainties to these values. The ambient data (originally reported in units of ppbC) were converted to μg/m3 prior to calculating the weight percentages using species-specific conversion factors. These uncertainties are explicit inputs to the CMB receptor model. They are propagated by the model mathematics to calculate the standard errors of source contribution estimates and to give lower influence to imprecise values in the least square estimation process.

Chemical Mass Balance Receptor Model. Model calculations were made using the U.S. EPA/DRI Version 7.0 of the CMB model (3). The CMB model consists of a least-squares solution to a set of linear equations which expresses each receptor concentration of a chemical species as a linear sum of products of source profile species and source contributions. The source profile species (the fractional amount of the species in the

NMHC emissions from each source type) and the receptor concentrations, each with realistic uncertainty estimates, serve as input data to the CMB model. The output consists of the contributions for each source type to the total ambient NMHC as well as to individual hydrocarbon concentrations. The model calculates values for contributions from each source and the uncertainties of those values. Input data uncertainties are used both to weight the relative importance of the input data to the model solution and to estimate uncertainties of the source contributions.

Source Profiles. The receptor modeling approach requires accurate and precise measurements of the chemical composition of emission sources and ambient receptor concentrations. Moreover, the source profiles must be representative of the study area during the period when the ambient data were collected. Three VOC profiles were specifically developed for this study, diesel exhaust, motor vehicle emissions and woodsmoke. Total VOC is the sum of C2-C11 hydrocarbons, C1-C7 carbonyls and semi-volatile PAH species up to Retene. Uncertainties are root mean squares of the analytical uncertainties or the standard deviation of values among samples in composite, whichever is higher. Table 1 lists the three profiles, expressed as weight percents.

A composite diesel truck exhaust profile was derived from the ambient measurements collected at the truck stop along Interstate 10 just west of Buckeye. Most of the samples had low concentrations because the sampling arrangement was less than ideal (i.e., sampler location was upwind of the trucks most of the time). Three of the eight samples with very low concentrations were removed from our data set. The remaining five samples were averaged and, as in the ambient data base, source profile data reported in units of ppbC were converted to μg/m3 prior to calculating the weight percentages using species-specific conversion factors. The species-specific standard deviations among the five samples were used to assign uncertainties. We also revised the values for total hydrocarbon by removing several peaks that appear to be oxygenated compounds. Weight fractions for compounds in the emissions profiles that were not identified in the ambient samples or reported as unidentified were combined into "unidentified".

Using the CMB model, we derived a summer and winter automotive emissions profile by fitting the composite diesel profile to the ambient samples from the Indian School road site, using only species that are concentrated in diesel exhaust as fitting species (all of the dimethyl naphthalene isomer except 1,8 dimethyl naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene fluoranthene, and pyrene). The diesel contributions ranged from 10 to 20 percent of the total NMHC. The automotive emissions profiles were derived from subtracting the species concentrations attributed to diesel exhaust. We did not include carbonyls in our derivation of the automotive emissions profiles from ambient data (i.e., using the receptor modeling approach) due to formation of carbonyl species in the atmosphere from photooxidation of hydrocarbons. The ambient measurements at the Indian School Road site are expected to represent primarily

automotive exhaust. However, the composite summer and winter profiles derived from the Indian School Road samples may also contain contributions of other source such as gasoline evaporation and nearby industrial and area sources.

With respect to the relative importance of vehicle exhaust versus liquid gasoline, we have shown in previous studies (Fujita et al., 1994) that their relative contributions are sensitive to ratios of acetylene and light olefins to NMHC in the exhaust composition profile, which vary with emission control technology, vehicle condition, and operating mode. Siegl et al. (1992) showed that unburned fuel represents most (>50 percent) of the hydrocarbon emission from a spark-ignited single-cylinder engine. In the CMB calculations, liquid gasoline represents the additional unburned gasoline (due to misfiring and other engine malfunctions) that is not included in the exhaust profile, plus evaporative emissions from gasoline spillage and hot soaks. One method to estimate the specific contribution of running emissions from on-road vehicles (hot exhaust plus running evaporative emission) to total emissions is to fit the tunnel profile to an early morning ambient sample (to avoid loss of reactive species) using species that are produced solely from the combustion process (e.g., ethylene, acetylene, propene, 1,3 butadiene, and perhaps the butenes and benzene). The analysis is repeated with addition of the whole gasoline profile and other fitting species appropriate to gasoline. The difference between the two analyses may provide an estimate of the evaporative emissions of whole gasoline in the ambient sample. In previous studies conducted for the Coastal Oxidant Assessment for Southeast Texas, we have shown that this ratio of the contributions of exhaust and liquid gasoline is typically 3 to 1 (Lu and Fujita, 1995).

The woodsmoke profile was derived from fitting the ISR automotive emissions, LPG and gasoline vapor (from the DRI Ft. McHenry Tunnel Study) profiles to the Payson data collected during January and February 1995. These three profiles account for nearly all of the VOC mass. Because the residual mass (presumed to be from woodsmoke) is small, the corresponding uncertainties are higher, particularly for the light hydrocarbon species. These higher uncertainties are reflected in the propagated uncertainties for these species and in the source contribution estimates from the CMB modeling.

Fitting Species. A prerequisite for using receptor models is that the relative proportions of chemical species change little between source and receptor. For the majority of organic compounds emitted from anthropogenic and biogenic sources, reaction with the hydroxyl radical is the sole chemical sink process, and typical atmospheric residence times can be estimated from the rate constants [summarized by Atkinson (1989)] for the reaction of hydroxyl radical with various organic compounds at typical hydroxyl radical concentrations. Summertime average lifetimes for highly reactive atmospheres are 5 days for acetylene, 5 hours to 4 days for alkanes, 4 days for benzene, and 9 hours for toluene. These lifetimes are comparable to or exceed the typical summer residence time of air masses in urban areas. These species were used as fitting species in Chemical Mass Balance (CMB) receptor

model calculations. Species with short lifetimes, such as 2-4 hours for xylenes and less than 2 hours for most of the alkenes (e.g., 0.8 hours for 1,3-butadiene), will have substantially changed in proportion to the other species after the air mass containing them has aged for a few hours.

The default volatile hydrocarbon fitting species used in this study are the same as those used by Fujita et al. (1994). These species are major constituents in all samples, and have atmospheric lifetimes equal to or greater than toluene. More reactive species were not used to calculate source contribution estimates, but were retained in the CMB calculations as "floating species". The comparison of calculated and measured values for floating species is part of the model validation process. PAH fitting species included naphthalene, 1-methylnaphthalene, 2-methylnaphthalene, biphenyl and retene.

RESULTS AND CONCLUSIONS

The CMB model was applied in this study to the 24-hour combined hydrocarbon, carbonyl and volatile and semi-volatile PAH data set only, and the weight fractions for individual species in the source composition profiles were normalized to sum of the above species. CMB source apportionments were individually performed on 120 samples (61 from the Phoenix Super Site, 33 from Payson, and 13 each from Casa Grande and Tucson).

Model Outputs and Performance Measures. Source contribution estimates (SCE) are the main output of the CMB model. The monthly average source contributions by site and HAPs species are summarized in Attachment H-1.

Results of the CMB analyses show that, in general, mobile sources (diesel and automotive, including evaporative emission) account for nearly all of the ambient VOC. Mobile sources also account for nearly all of the ambient concentrations for most individual HAPs. HAPs species that are associated with evaporation of gasoline and combustion of gasolines in spark-ignition engines, such as benzene, toluene, ethylbenzene and xylenes are assigned almost exclusively to these sources (Figures 1 and 2). The same conclusion apply to other HAPs species that are emitted from automotive exhaust, such as 1,3-butadiene. A small amount of the benzene was assigned to woodsmoke during the winter months (less than 5% at Phoenix and as much as 10-15 percent at Payson).

As shown in Figures 3 and 4, the source contributions of polycyclic aromatics hydrocarbons vary considerably with specific PAH. Retene is specific to woodsmoke, the dimethyl napthalene isomers are highly concentrated in diesel exhaust, and naphthalene is produced both by automotive and diesel exhaust. The contribution of automotive exhaust to ambient

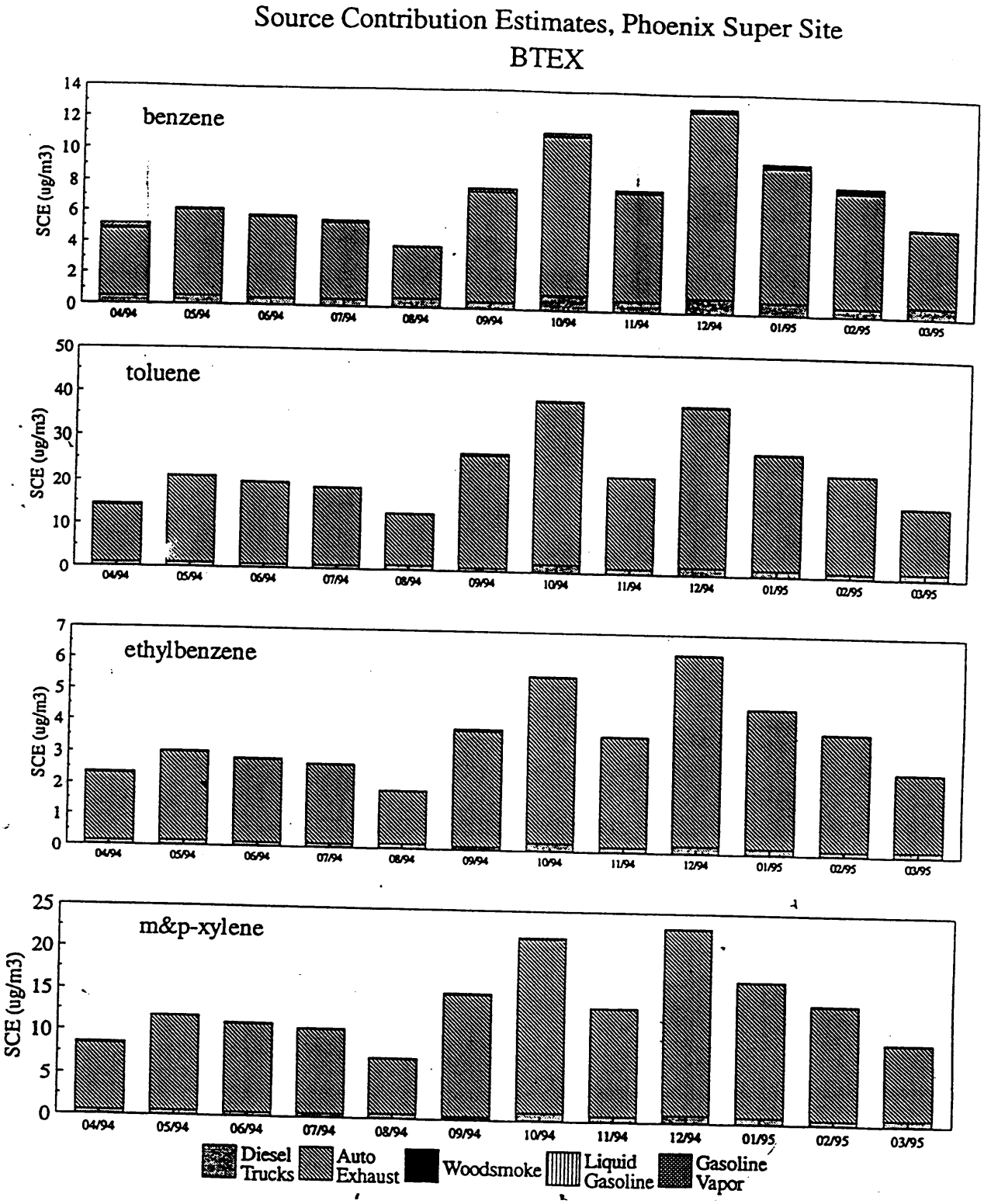

Figure 1 Source contribution estimates of benzene, toluene, ethylbenzene, and m&p-xylene at the Phoenix Super Site (April 1994 to March 1995)

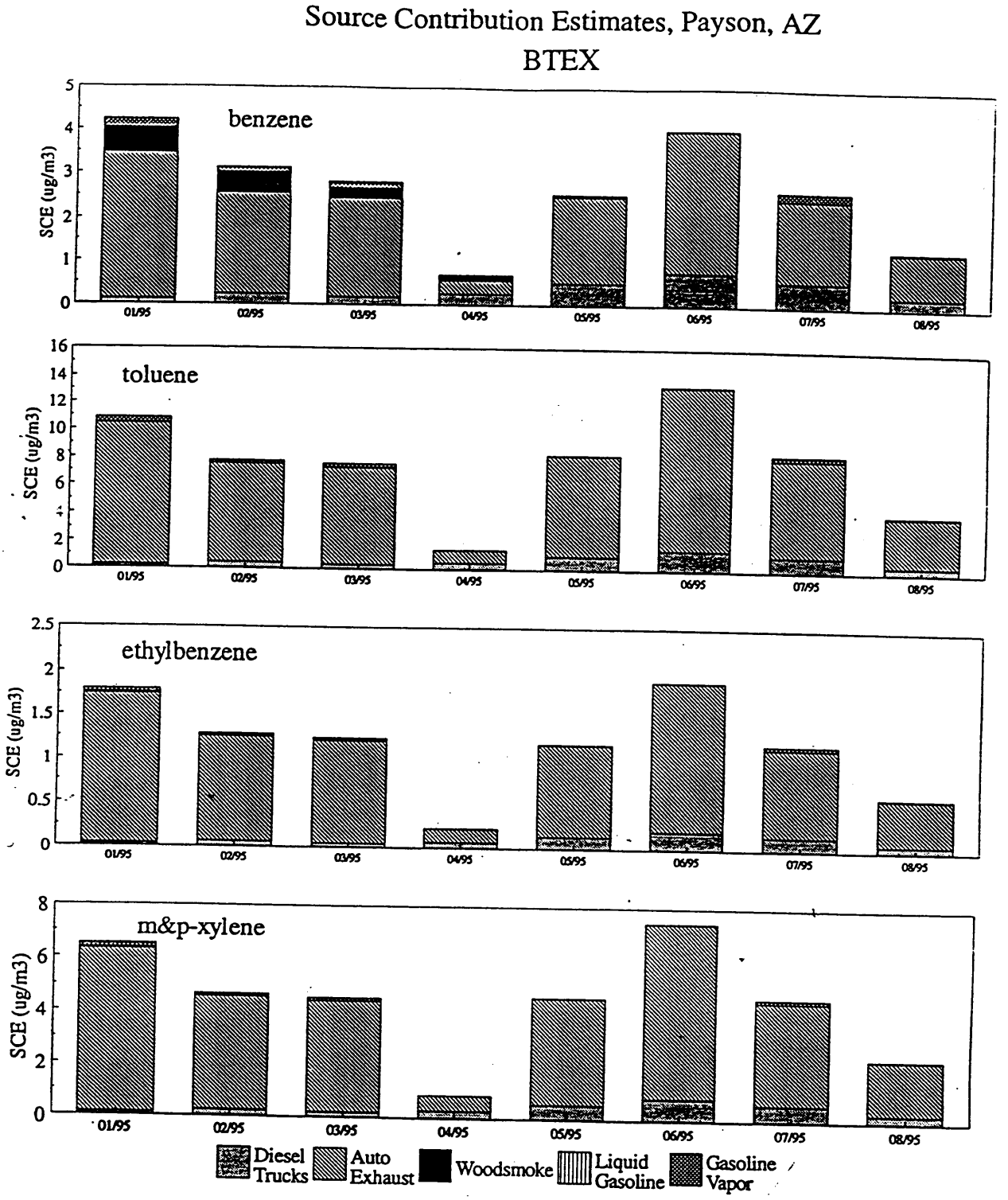

Figure 2 Source contribution estimates of benzene, toluene, ethylbenzene, and m&p-xylene at the Payson (January 1995 to August 1995)

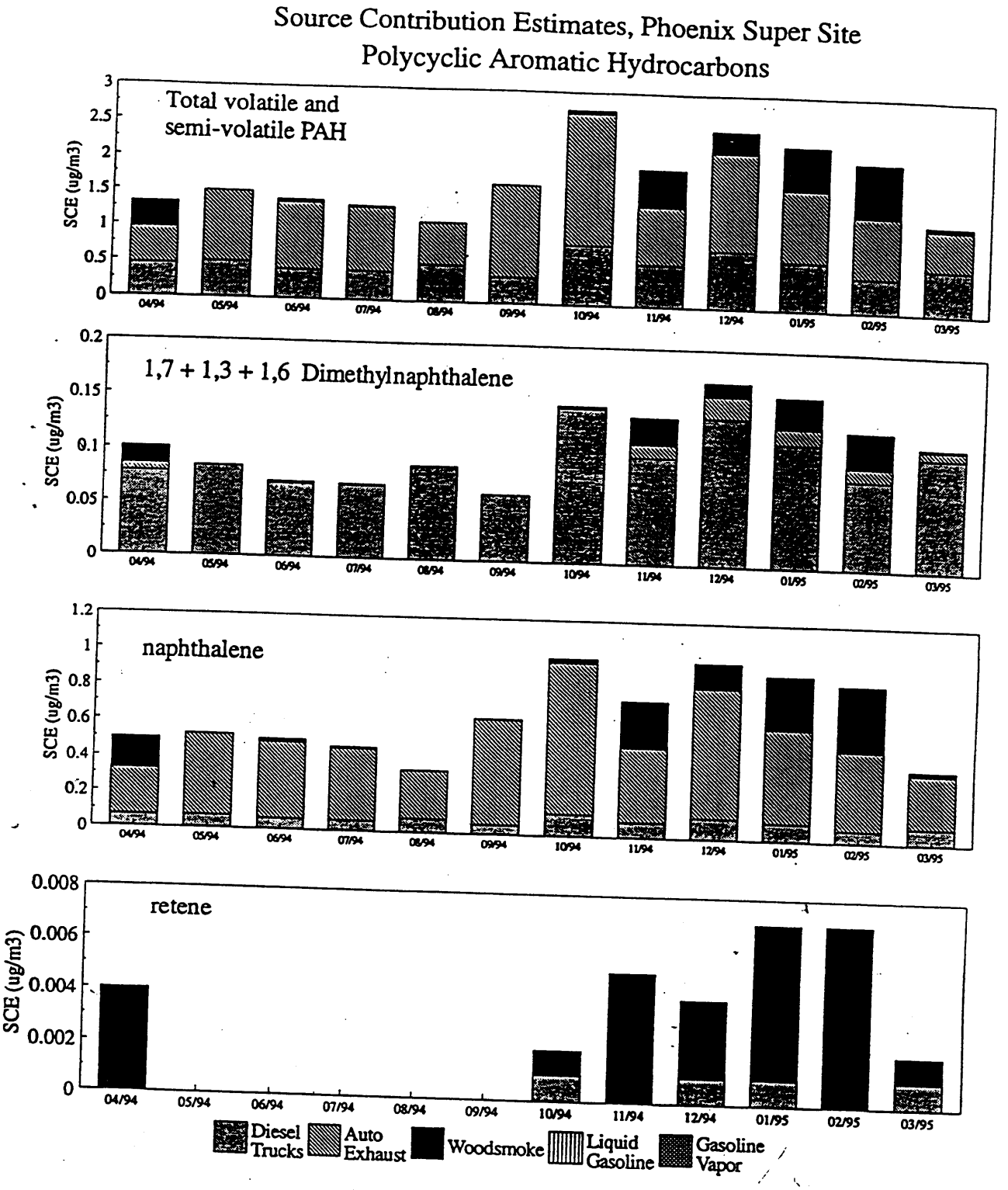

Figure 3. Source contribution estimates of polycyclic aromatics at the Phoenix Super Site (April 1994 to March 1995)

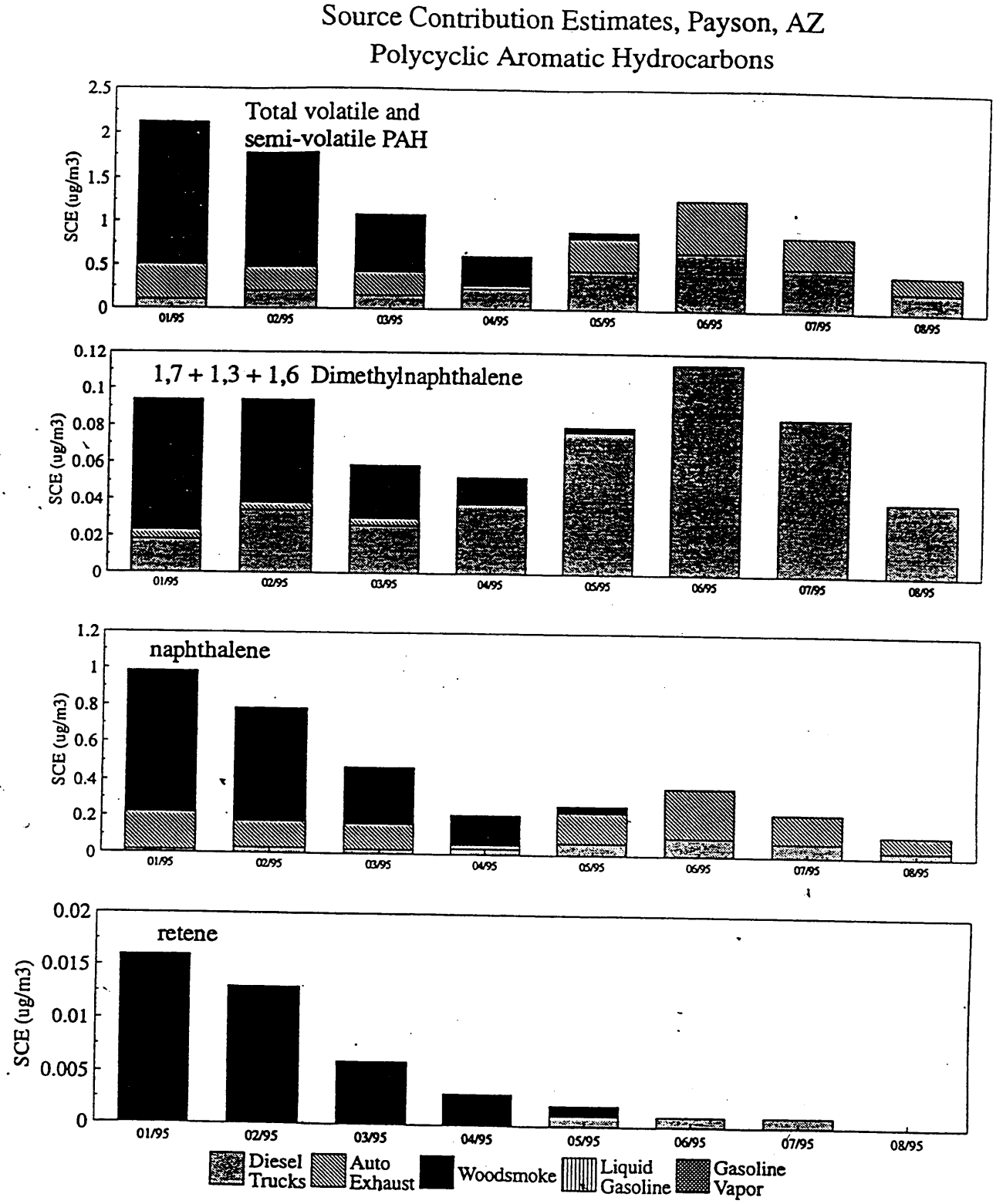

Figure 4 Source contribution estimates of polycyclic aromatics at the Payson (January 1995 to August 1995)

naphthalene is typically much higher than diesel exhaust because relative emissions of total hydrocarbon from automotive exhaust is typically much higher in urban areas. Woodsmoke is the predominant source (about 75%) of PAHs in Payson during the winter months. At other times of the year, automotive and diesel emissions contribute roughly equal amounts. Automotive emissions are the largest source of total PAHs at all times of the year at the Phoenix Super Site. Woodsmoke is also a significant contributor during the winter (as much as 25%), but substantially less on a fractional basis than is observed at Payson.

Species-by-species comparisons of calculated versus measured mass show that the reactive species account for most of the excess predicted mass. The over-prediction of mass increases with increasing photochemical reactivity because predicted concentrations for reactive species are calculated from source contribution estimates obtained by fitting only the less reactive species. For example, predicted mass for 1,3-butadiene is as much as 75% higher than observed mass (over a 24-hour period) during the summer months owing to its extreme reactivity (lifetime during the daylight hours is less than 1 hour during the summer). During the winter months, when photochemical reactions are not as significant, predicted and measured mass for 1,3-butadiene are nearly equal. In contrast, benzene which has a lifetime of 45 hours show equal predicted and observed mass at all times of the year. Reactive species were retained in the CMB calculations as floating species, and have no influence on the apportionment (i.e., relative source apportionments are valid despite reactivity). Excess predicted mass for reactive species also may be related to uncertainties in the source profiles, especially for species found in low concentrations.

Reconciliation with Emissions Estimates. One of the objectives of this study was to compare SCEs obtained by CMB modeling with corresponding emission inventory estimates. Two specific examples of this comparison raise issues that are common to other HAPS emissions as well.

As documented in the main text of this report, 1,3-butadiene is one of the HAP species found to pose significant risk in Arizona. Based on the emission inventory estimates, small utility engines such as lawnmowers contribute about 40% of the 1,3-butadiene. Mobile sources account for the balance of the emissions. In contrast, our CMB results assigns most of the ambient 1,3-butadiene to mobile sources.

The CMB analysis did not explicitly include small internal combustion engines, such as those used in lawn and garden equipment, because adequate data about the chemical composition of emissions from these engines were not available. Therefore, if the chemical composition of emissions from small gasoline engines is similar to the composition of emissions from spark-ignition vehicles, the receptor modeling attribution to spark-ignition motor vehicles includes both of these source categories. In fact, the CMB apportionment of the explained fraction of 1,3-butadiene and three other species to gasoline-fueled engines agrees within ten percent of their relative emission rates.

In the second example, PAHs are assigned almost exclusively to woodsmoke in the emission inventory, while CMB analyses show significant contributions from automotive emissions, particularly in Phoenix. The second example may be of little significance to the overall risk assessment, since the health risk posed by ambient PAHs relatively low. Nevertheless, this example is illustrative of contemporary emission inventory issues which have implications for ozone and particulate matter attainment.

Representativeness of ambient measurements relative to total regional emissions is a major uncertainty that is common to the interpretation of all source apportionment analysis. These uncertainties can be evaluated by a well-designed saturation monitoring program with supplemental measurements in specific source regions, and an understanding of the important atmospheric processes that affect ambient pollutant concentrations. Since the present monitoring program was not designed to meet fully these requirements, our CMB analyses provide source attributions for one location and only a qualitative evaluation of the emission inventory. Ambient VOC composition generally reflects sources that are uniformly distributed within the region, such as mobile, and local sources, especially during the calm, stable meteorological conditions. Given the preponderance of vehicle traffic at the Phoenix Super Site, the incremental contribution of utility engines to the ambient VOCs would be small. The larger calculated contributions of vehicle emissions are consistent with recent studies that suggest that the motor vehicle hydrocarbon emission inventories for motor vehicles have been substantially underestimated (Ingalls et al., 1989; Pierson et al., 1990; NRC, 1991; Chico et al., 1993; Wagner et al., 1991; Fujita et al., 1992; Harley et al., 1993).

In order to determine the relative contributions of woodsmoke, and gasoline- and diesel-fueled vehicles, it is necessary to characterize the variations in composition of particulate emissions with emission rates. Hildemann et al. (1991) examined seven 1977-1983 catalyst-equipped in-use vehicles with an average odometer reading of 77,000 miles and found that their exhaust emission rate of fine particles averaged 28.5 mg/mile. On the other hand, the Unocal SCRAP program measured PM_{10} emission from 31 vehicles (average age of 22 years in 1990) with an average emission rate of 1,510 mg/mile, with one vehicle emitting at a rate of 16,800 mg/mi. Sagebiel et al. (1995) measured particulate and gaseous emissions from 23 vehicles that were identified as high emitters by remote sensing (based on measurements of excessive CO and HC) and roadside inspections. The average IM240 particle emission rate for 17 non-smoking vehicles was 50.8 mg/mi. Six vehicles which emitted visible smoke averaged 558 mg/mi, with a high of 1342 mg/mi. In contrast, a "clean" vehicle typically emits 5 mg/mi and the fleet average particulate emission rates for Phoenix of 13 mg/mi was used for this study. DRI's preliminary examination of the PAH data shows that particle emissions from smokers contain higher relative amounts of particulate PAH relative to volatile and semi-volatile PAH. These recent data suggest that particulate emissions for gasoline vehicles may be substantially underestimated in the present inventory.

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ATTACHMENT H-1

SOURCE CONTRIBUTION BY SPECIES

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SOURCE CONTRIBUTION BY SPECIES

Source Contribution by Species

Monthly average source contribution estimates (SCE) from the CMB model by site and HAPs species, are presented in this attachment.

The uncertainties associated with these averages are the root-mean squared (RMS) errors of the source contribution estimates for individual samples rather than the standard error of the averages. The sum of these concentrations approximates the total mass concentrations. Negative SCE are not physically meaningful, but can occur when a source profile is collinear with another profile or when the source contribution is close to zero. Collinearity is usually identified in the similarity/uncertainty cluster display. When the SCE is less than its standard error, the source contribution is undetectable. Two or three times the standard error may be taken as the upper limit of the SCE in this case.

The chi square (c^2), R^2, and percent mass are goodness of fit measures for the least square calculation. The c^2 is the weighted sum of squares of the differences between calculated and measured fitting species concentrations. The weighting is inversely proportional to the squares of the precision in the source profiles and ambient data for each species. Ideally, there would be no difference between calculated and measured species concentrations and c^2 would be zero. A value of less than one indicates a very good fit to the data, while values between 1 and 2 are acceptable. c^2 values greater than 4 indicate that one or more of the fitting species concentrations are not well-explained by the source contribution estimates.

R^2 is determined by the linear regression of the measured versus model-calculated values for the fitting species. R^2 ranges from 0 to 1. The closer the value is to 1.0, the better the SCEs explain the measured concentrations. When R^2 is less than 0.8, the SECs do not explain the observations very well with the given source profiles.

Percent mass is the percent ratio of the sum of model-calculated SCEs to the measured mass concentration. This ratio should equal 100%, though values ranging from 80 to 120% are acceptable.

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
ACETAL	Phoenix SS	04/94	5	0.98	1.95	2.75 ± 0.25	27.1	0.000 ± 0.000	0.746 ± 0.136	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	05/94	5	0.98	1.15	4.56 ± 0.35	24.9	0.000 ± 0.001	1.137 ± 0.196	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	06/94	5	0.97	1.24	3.63 ± 0.27	29.5	0.000 ± 0.000	1.071 ± 0.186	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	07/94	6	0.99	0.71	6.42 ± 0.54	15.8	0.000 ± 0.000	1.016 ± 0.173	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	08/94	5	0.97	1.42	4.04 ± 0.33	16.7	0.000 ± 0.001	0.676 ± 0.114	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	09/94	4	0.98	0.90	6.76 ± 0.51	21.8	0.000 ± 0.001	1.473 ± 0.276	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
ACETAL	Phoenix SS	10/94	5	0.98	1.24	8.03 ± 0.59	26.3	0.000 ± 0.001	2.111 ± 0.350	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	11/94	5	0.99	1.25	6.11 ± 0.47	19.8	0.000 ± 0.001	1.212 ± 0.222	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	12/94	5	1.00	0.81	6.13 ± 0.48	34.2	0.000 ± 0.001	2.095 ± 0.336	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	01/95	5	0.99	1.15	5.03 ± 0.40	30.2	0.000 ± 0.001	1.521 ± 0.244	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	02/95	5	0.99	1.62	3.72 ± 0.29	34.5	0.000 ± 0.000	1.284 ± 0.217	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Phoenix SS	03/95	4	0.99	1.23	2.78 ± 0.22	30.6	0.000 ± 0.001	0.852 ± 0.134	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	04/94	5	0.98	1.95	0.47 ± 0.64	149.4	0.000 ± 0.000	0.702 ± 0.128	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	05/94	5	0.98	1.15	1.03 ± 0.74	103.8	0.000 ± 0.001	1.069 ± 0.184	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	06/94	5	0.97	1.24	1.58 ± 0.75	63.7	0.000 ± 0.000	1.007 ± 0.175	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	07/94	6	0.99	0.71	1.25 ± 0.75	76.4	0.000 ± 0.000	0.955 ± 0.163	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	08/94	5	0.97	1.42	0.39 ± 0.67	163.1	0.000 ± 0.001	0.636 ± 0.107	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	09/94	4	0.98	0.90	1.76 ± 0.79	78.7	0.000 ± 0.001	1.385 ± 0.259	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
ACETO	Phoenix SS	10/94	5	0.98	1.24	2.50 ± 0.77	79.4	0.000 ± 0.001	1.985 ± 0.329	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	11/94	5	0.99	1.25	3.15 ± 0.82	36.2	0.000 ± 0.001	1.140 ± 0.209	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	12/94	5	1.00	0.81	2.59 ± 0.81	76.1	0.000 ± 0.001	1.970 ± 0.316	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	01/95	5	0.99	1.15	0.99 ± 0.75	144.4	0.000 ± 0.001	1.430 ± 0.229	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	02/95	5	0.99	1.62	0.81 ± 0.74	149.0	0.000 ± 0.000	1.207 ± 0.204	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Phoenix SS	03/95	4	0.99	1.23	1.17 ± 0.73	68.5	0.000 ± 0.001	0.801 ± 0.126	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	04/94	5	0.98	1.95	0.01 ± 0.00	50.0	0.003 ± 0.002	0.001 ± 0.001	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	05/94	5	0.98	1.15	0.01 ± 0.00	60.0	0.003 ± 0.002	0.003 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	06/94	5	0.97	1.24	0.01 ± 0.00	50.0	0.002 ± 0.002	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	07/94	6	0.99	0.71	0.01 ± 0.00	50.0	0.002 ± 0.002	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	08/94	5	0.97	1.42	0.01 ± 0.00	50.0	0.003 ± 0.002	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	09/94	4	0.98	0.90	0.01 ± 0.00	70.0	0.002 ± 0.002	0.005 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
ACNAPE	Phoenix SS	10/94	5	0.98	1.24	0.01 ± 0.00	110.0	0.005 ± 0.004	0.006 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	11/94	5	0.99	1.25	0.01 ± 0.00	80.0	0.004 ± 0.003	0.002 ± 0.001	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	12/94	5	1.00	0.81	0.01 ± 0.00	100.0	0.005 ± 0.004	0.004 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	01/95	5	0.99	1.15	0.01 ± 0.00	90.0	0.004 ± 0.003	0.003 ± 0.001	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	02/95	5	0.99	1.62	0.01 ± 0.00	80.0	0.003 ± 0.002	0.002 ± 0.001	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Phoenix SS	03/95	4	0.99	1.23	0.00 ± 0.00		0.004 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	04/94	5	0.98	1.95	0.00 ± 0.00		0.008 ± 0.006	0.008 ± 0.002	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.00		0.008 ± 0.007	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	06/94	5	0.97	1.24	0.00 ± 0.00		0.007 ± 0.006	0.000 ± 0.003	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.00		0.007 ± 0.005	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.00		0.009 ± 0.007	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	09/94	4	0.98	0.90	0.01 ± 0.00	70.0	0.007 ± 0.006	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
ACNAPY	Phoenix SS	10/94	5	0.98	1.24	0.01 ± 0.00	150.0	0.014 ± 0.013	0.000 ± 0.006	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	11/94	5	0.99	1.25	0.03 ± 0.01	106.7	0.010 ± 0.009	0.013 ± 0.004	0.009 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	12/94	5	1.00	0.81	0.04 ± 0.01	105.0	0.014 ± 0.011	0.023 ± 0.005	0.005 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	01/95	5	0.99	1.15	0.03 ± 0.00	130.0	0.012 ± 0.010	0.016 ± 0.004	0.011 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	02/95	5	0.99	1.62	0.03 ± 0.01	113.3	0.008 ± 0.007	0.014 ± 0.003	0.012 ± 0.006	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Phoenix SS	03/95	4	0.99	1.23	0.01 ± 0.00	210.0	0.011 ± 0.009	0.009 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	04/94	5	0.98	1.95	0.00 ± 0.02		0.000 ± 0.000	0.090 ± 0.016	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
ACROLN	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.03		0.000 ± 0.001	0.136 ± 0.024	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	06/94	5	0.97	1.24	0.00 ± 0.03		0.000 ± 0.000	0.128 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.03		0.000 ± 0.000	0.122 ± 0.021	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.03		0.000 ± 0.001	0.081 ± 0.014	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.03		0.000 ± 0.001	0.177 ± 0.033	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
ACROLN	Phoenix SS	10/94	5	0.98	1.24	0.00 ± 0.03		0.000 ± 0.001	0.253 ± 0.042	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	11/94	5	0.99	1.25	0.00 ± 0.03		0.000 ± 0.001	0.145 ± 0.027	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	12/94	5	1.00	0.81	0.00 ± 0.03		0.000 ± 0.001	0.251 ± 0.040	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	01/95	5	0.99	1.15	0.00 ± 0.03		0.000 ± 0.001	0.182 ± 0.029	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	02/95	5	0.99	1.62	0.00 ± 0.03		0.000 ± 0.000	0.154 ± 0.026	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Phoenix SS	03/95	4	0.99	1.23	0.07 ± 0.03	145.7	0.000 ± 0.001	0.102 ± 0.016	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	04/94	5	0.98	1.95	0.00 ± 0.00		0.004 ± 0.002	0.000 ± 0.002	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.00		0.005 ± 0.003	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	06/94	5	0.97	1.24	0.00 ± 0.00		0.004 ± 0.002	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.00		0.004 ± 0.002	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.00		0.005 ± 0.003	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.00		0.003 ± 0.003	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	10/94	5	0.98	1.24	0.00 ± 0.00		0.007 ± 0.005	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
ANTHRA	Phoenix SS	11/94	5	0.99	1.25	0.00 ± 0.00		0.005 ± 0.004	0.000 ± 0.004	0.002 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	12/94	5	1.00	0.81	0.00 ± 0.00		0.007 ± 0.005	0.000 ± 0.006	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	01/95	5	0.99	1.15	0.00 ± 0.00		0.006 ± 0.004	0.000 ± 0.004	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	02/95	5	0.99	1.62	0.00 ± 0.00		0.005 ± 0.003	0.000 ± 0.004	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Phoenix SS	03/95	4	0.99	1.23	0.00 ± 0.00		0.006 ± 0.003	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BENZE	Phoenix SS	04/94	5	0.98	1.95	5.83 ± 0.18	88.4	0.530 ± 0.227	4.243 ± 0.306	0.132 ± 0.067	0.000 ± 0.000	0.246 ± 0.035
BENZE	Phoenix SS	05/94	5	0.98	1.15	6.69 ± 0.19	91.9	0.572 ± 0.244	5.470 ± 0.799	0.000 ± 0.001	0.000 ± 0.000	0.107 ± 0.015
BENZE	Phoenix SS	06/94	5	0.97	1.24	5.71 ± 0.16	100.8	0.465 ± 0.209	5.151 ± 0.761	0.013 ± 0.007	0.000 ± 0.000	0.125 ± 0.017
BENZE	Phoenix SS	07/94	6	0.99	0.71	5.70 ± 0.16	96.4	0.464 ± 0.193	4.888 ± 0.704	0.007 ± 0.004	0.000 ± 0.000	0.134 ± 0.017
BENZE	Phoenix SS	08/94	5	0.97	1.42	3.79 ± 0.11	103.5	0.593 ± 0.258	3.254 ± 0.463	0.001 ± 0.004	0.000 ± 0.000	0.075 ± 0.011
BENZE	Phoenix SS	09/94	4	0.98	0.90	8.48 ± 0.25	91.8	0.424 ± 0.226	7.087 ± 1.122	0.002 ± 0.001	0.000 ± 0.000	0.270 ± 0.041
BENZE	Phoenix SS	10/94	5	0.98	1.24	11.76 ± 0.30	96.6	0.976 ± 0.456	10.159 ± 1.424	0.018 ± 0.008	0.000 ± 0.000	0.203 ± 0.026
BENZE	Phoenix SS	11/94	5	0.99	1.25	7.90 ± 0.24	98.7	0.681 ± 0.319	6.890 ± 0.500	0.193 ± 0.071	0.000 ± 0.000	0.034 ± 0.005
BENZE	Phoenix SS	12/94	5	1.00	0.81	13.56 ± 0.34	96.5	0.934 ± 0.407	11.907 ± 0.756	0.108 ± 0.040	0.000 ± 0.000	0.137 ± 0.016
BENZE	Phoenix SS	01/95	5	0.99	1.15	9.46 ± 0.24	103.0	0.790 ± 0.356	8.646 ± 0.548	0.224 ± 0.080	0.000 ± 0.000	0.086 ± 0.011
BENZE	Phoenix SS	02/95	5	0.99	1.62	7.97 ± 0.22	103.7	0.566 ± 0.252	7.295 ± 0.489	0.271 ± 0.102	0.000 ± 0.000	0.131 ± 0.017
BENZE	Phoenix SS	03/95	4	0.99	1.23	5.75 ± 0.15	98.6	0.724 ± 0.308	4.843 ± 0.302	0.018 ± 0.007	0.000 ± 0.000	0.082 ± 0.011
BIPHEN	Phoenix SS	04/94	5	0.98	1.95	0.02 ± 0.00	140.0	0.023 ± 0.015	0.000 ± 0.002	0.005 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	05/94	5	0.98	1.15	0.02 ± 0.00	125.0	0.025 ± 0.016	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	06/94	5	0.97	1.24	0.02 ± 0.00	105.0	0.020 ± 0.013	0.000 ± 0.003	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	07/94	6	0.99	0.71	0.01 ± 0.00	200.0	0.020 ± 0.012	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	08/94	5	0.97	1.42	0.01 ± 0.00	260.0	0.026 ± 0.017	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	09/94	4	0.98	0.90	0.01 ± 0.00	180.0	0.018 ± 0.014	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
BIPHEN	Phoenix SS	10/94	5	0.98	1.24	0.02 ± 0.00	215.0	0.042 ± 0.029	0.000 ± 0.006	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	11/94	5	0.99	1.25	0.03 ± 0.00	120.0	0.029 ± 0.020	0.000 ± 0.004	0.007 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	12/94	5	1.00	0.81	0.03 ± 0.00	146.7	0.040 ± 0.026	0.000 ± 0.006	0.004 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	01/95	5	0.99	1.15	0.03 ± 0.00	140.0	0.034 ± 0.023	0.000 ± 0.004	0.008 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	02/95	5	0.99	1.62	0.02 ± 0.00	170.0	0.024 ± 0.016	0.000 ± 0.004	0.010 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Phoenix SS	03/95	4	0.99	1.23	0.01 ± 0.00	330.0	0.032 ± 0.020	0.000 ± 0.002	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	04/94	5	0.98	1.95	0.36 ± 0.03	172.5	0.114 ± 0.064	0.488 ± 0.037	0.019 ± 0.022	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	05/94	5	0.98	1.15	0.56 ± 0.03	107.3	0.123 ± 0.069	0.478 ± 0.084	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
BUDI13	Phoenix SS	06/94	5	0.97	1.24	0.36 ± 0.03	153.3	0.100 ± 0.059	0.450 ± 0.080	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	07/94	6	0.99	0.71	0.42 ± 0.02	125.7	0.100 ± 0.055	0.427 ± 0.074	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	08/94	5	0.97	1.42	0.23 ± 0.03	179.1	0.128 ± 0.073	0.284 ± 0.048	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	09/94	4	0.98	0.90	0.68 ± 0.04	104.6	0.092 ± 0.064	0.619 ± 0.117	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.001
BUDI13	Phoenix SS	10/94	5	0.98	1.24	1.07 ± 0.06	102.8	0.210 ± 0.129	0.887 ± 0.149	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	11/94	5	0.99	1.25	0.84 ± 0.06	114.9	0.146 ± 0.090	0.791 ± 0.061	0.028 ± 0.023	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	12/94	5	1.00	0.81	1.59 ± 0.09	99.7	0.201 ± 0.115	1.368 ± 0.092	0.016 ± 0.013	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	01/95	5	0.99	1.15	1.14 ± 0.06	104.8	0.170 ± 0.101	0.993 ± 0.067	0.032 ± 0.026	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	02/95	5	0.99	1.62	0.77 ± 0.04	129.9	0.122 ± 0.071	0.838 ± 0.060	0.040 ± 0.033	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Phoenix SS	03/95	4	0.99	1.23	0.47 ± 0.03	152.1	0.156 ± 0.087	0.556 ± 0.037	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
BZI24M	Phoenix SS	04/94	5	0.98	1.95	3.95 ± 0.26	77.6	0.467 ± 0.162	2.427 ± 1.310	0.107 ± 0.119	0.000 ± 0.000	0.066 ± 0.043
BZI24M	Phoenix SS	05/94	5	0.98	1.15	4.74 ± 0.28	89.7	0.505 ± 0.174	3.719 ± 0.931	0.000 ± 0.002	0.000 ± 0.000	0.029 ± 0.018
BZI24M	Phoenix SS	06/94	5	0.97	1.24	3.98 ± 0.26	99.4	0.410 ± 0.149	3.502 ± 0.886	0.010 ± 0.012	0.000 ± 0.000	0.033 ± 0.021
BZI24M	Phoenix SS	07/94	6	0.99	0.71	3.70 ± 0.23	102.0	0.410 ± 0.138	3.323 ± 0.820	0.006 ± 0.006	0.000 ± 0.000	0.036 ± 0.021
BZI24M	Phoenix SS	08/94	5	0.97	1.42	3.05 ± 0.19	90.4	0.524 ± 0.184	2.212 ± 0.540	0.001 ± 0.007	0.000 ± 0.000	0.020 ± 0.014
BZI24M	Phoenix SS	09/94	4	0.98	0.90	5.38 ± 0.37	97.9	0.374 ± 0.161	4.818 ± 1.307	0.002 ± 0.002	0.000 ± 0.000	0.072 ± 0.051
BZI24M	Phoenix SS	10/94	5	0.98	1.24	8.07 ± 0.49	97.1	0.861 ± 0.326	6.906 ± 1.659	0.015 ± 0.013	0.000 ± 0.000	0.054 ± 0.033
BZI24M	Phoenix SS	11/94	5	0.99	1.25	4.99 ± 0.35	94.3	0.601 ± 0.228	3.941 ± 2.137	0.157 ± 0.127	0.000 ± 0.000	0.009 ± 0.006
BZI24M	Phoenix SS	12/94	5	1.00	0.81	9.13 ± 0.55	85.0	0.824 ± 0.290	6.810 ± 3.230	0.087 ± 0.072	0.000 ± 0.000	0.037 ± 0.020
BZI24M	Phoenix SS	01/95	5	0.99	1.15	6.97 ± 0.42	83.9	0.697 ± 0.254	4.945 ± 2.342	0.181 ± 0.143	0.000 ± 0.000	0.023 ± 0.015
BZI24M	Phoenix SS	02/95	5	0.99	1.62	5.04 ± 0.33	97.8	0.500 ± 0.180	4.172 ± 2.090	0.220 ± 0.183	0.000 ± 0.000	0.035 ± 0.021
BZI24M	Phoenix SS	03/95	4	0.99	1.23	4.08 ± 0.25	84.5	0.639 ± 0.220	2.770 ± 1.289	0.015 ± 0.013	0.000 ± 0.000	0.022 ± 0.014
BZI35M	Phoenix SS	04/94	5	0.98	1.95	1.24 ± 0.10	95.8	0.094 ± 0.016	1.071 ± 0.040	0.000 ± 0.000	0.000 ± 0.000	0.023 ± 0.012
BZI35M	Phoenix SS	05/94	5	0.98	1.15	1.45 ± 0.11	88.5	0.102 ± 0.017	1.171 ± 0.153	0.000 ± 0.000	0.000 ± 0.000	0.010 ± 0.005
BZI35M	Phoenix SS	06/94	5	0.97	1.24	1.29 ± 0.09	92.9	0.083 ± 0.014	1.103 ± 0.145	0.000 ± 0.000	0.000 ± 0.000	0.012 ± 0.006
BZI35M	Phoenix SS	07/94	6	0.99	0.71	1.16 ± 0.09	98.4	0.083 ± 0.014	1.046 ± 0.134	0.000 ± 0.000	0.000 ± 0.000	0.012 ± 0.006
BZI35M	Phoenix SS	08/94	5	0.97	1.42	0.75 ± 0.06	107.9	0.105 ± 0.018	0.697 ± 0.088	0.000 ± 0.000	0.000 ± 0.000	0.007 ± 0.004
BZI35M	Phoenix SS	09/94	4	0.98	0.90	1.58 ± 0.12	102.4	0.076 ± 0.016	1.517 ± 0.214	0.000 ± 0.000	0.000 ± 0.000	0.025 ± 0.014
BZI35M	Phoenix SS	10/94	5	0.98	1.24	2.41 ± 0.16	98.3	0.174 ± 0.032	2.175 ± 0.272	0.000 ± 0.000	0.000 ± 0.000	0.019 ± 0.009
BZI35M	Phoenix SS	11/94	5	0.99	1.25	1.68 ± 0.12	111.0	0.121 ± 0.022	1.740 ± 0.066	0.000 ± 0.000	0.000 ± 0.000	0.003 ± 0.002
BZI35M	Phoenix SS	12/94	5	1.00	0.81	3.13 ± 0.20	101.8	0.166 ± 0.028	3.006 ± 0.099	0.000 ± 0.000	0.000 ± 0.000	0.013 ± 0.005
BZI35M	Phoenix SS	01/95	5	0.99	1.15	2.33 ± 0.14	100.1	0.141 ± 0.025	2.183 ± 0.072	0.000 ± 0.000	0.000 ± 0.000	0.008 ± 0.004
BZI35M	Phoenix SS	02/95	5	0.99	1.62	1.57 ± 0.12	124.5	0.101 ± 0.018	1.842 ± 0.064	0.000 ± 0.000	0.000 ± 0.000	0.012 ± 0.006
BZI35M	Phoenix SS	03/95	4	0.99	1.23	1.22 ± 0.10	111.5	0.129 ± 0.021	1.223 ± 0.040	0.000 ± 0.000	0.000 ± 0.000	0.008 ± 0.004
CROTON	Phoenix SS	04/94	5	0.98	1.95	0.00 ± 0.03		0.000 ± 0.000	0.060 ± 0.011	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.03		0.000 ± 0.001	0.091 ± 0.016	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	06/94	5	0.97	1.24	0.00 ± 0.03		0.000 ± 0.000	0.086 ± 0.015	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.03		0.000 ± 0.000	0.081 ± 0.014	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.03		0.000 ± 0.001	0.054 ± 0.009	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.03		0.000 ± 0.001	0.118 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
CROTON	Phoenix SS	10/94	5	0.98	1.24	0.00 ± 0.03		0.000 ± 0.001	0.169 ± 0.028	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	11/94	5	0.99	1.25	0.00 ± 0.03		0.000 ± 0.001	0.097 ± 0.018	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	12/94	5	1.00	0.81	0.00 ± 0.03		0.000 ± 0.001	0.167 ± 0.027	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	01/95	5	0.99	1.15	0.00 ± 0.03		0.000 ± 0.001	0.122 ± 0.019	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	02/95	5	0.99	1.62	0.00 ± 0.03		0.000 ± 0.000	0.102 ± 0.018	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Phoenix SS	03/95	4	0.99	1.23	0.03 ± 0.03	226.7	0.000 ± 0.001	0.068 ± 0.011	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	04/94	5	0.98	1.95	0.03 ± 0.00	106.7	0.026 ± 0.018	0.000 ± 0.006	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	05/94	5	0.98	1.15	0.02 ± 0.00	140.0	0.028 ± 0.019	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	06/94	5	0.97	1.24	0.02 ± 0.00	120.0	0.023 ± 0.017	0.000 ± 0.003	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
D14523	Phoenix SS	07/94	6	0.99	0.71	0.02 ± 0.00	115.0	0.023 ± 0.015	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	08/94	5	0.97	1.42	0.02 ± 0.00	145.0	0.029 ± 0.020	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	09/94	4	0.98	0.90	0.02 ± 0.00	105.0	0.021 ± 0.018	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
D14523	Phoenix SS	10/94	5	0.98	1.24	0.03 ± 0.00	163.3	0.048 ± 0.036	0.000 ± 0.006	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	11/94	5	0.99	1.25	0.04 ± 0.00	105.0	0.034 ± 0.025	0.000 ± 0.010	0.008 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	12/94	5	1.00	0.81	0.05 ± 0.00	100.0	0.046 ± 0.032	0.000 ± 0.014	0.004 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	01/95	5	0.99	1.15	0.04 ± 0.00	122.5	0.039 ± 0.028	0.001 ± 0.010	0.009 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	02/95	5	0.99	1.62	0.04 ± 0.00	97.5	0.028 ± 0.020	0.000 ± 0.010	0.011 ± 0.005	0.000 ± 0.000	0.000 ± 0.000
D14523	Phoenix SS	03/95	4	0.99	1.23	0.02 ± 0.00	185.0	0.036 ± 0.024	0.000 ± 0.006	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	04/94	5	0.98	1.95	0.05 ± 0.01	202.0	0.078 ± 0.054	0.007 ± 0.018	0.016 ± 0.013	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	05/94	5	0.98	1.15	0.07 ± 0.01	120.0	0.084 ± 0.058	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	06/94	5	0.97	1.24	0.08 ± 0.01	87.5	0.068 ± 0.050	0.000 ± 0.003	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	07/94	6	0.99	0.71	0.06 ± 0.01	115.0	0.068 ± 0.046	0.000 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	08/94	5	0.97	1.42	0.05 ± 0.01	174.0	0.087 ± 0.062	0.000 ± 0.002	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	09/94	4	0.98	0.90	0.05 ± 0.01	124.0	0.062 ± 0.054	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
DM1367	Phoenix SS	10/94	5	0.98	1.24	0.09 ± 0.01	161.1	0.143 ± 0.109	0.000 ± 0.006	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	11/94	5	0.99	1.25	0.11 ± 0.01	123.6	0.100 ± 0.076	0.012 ± 0.029	0.024 ± 0.014	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	12/94	5	1.00	0.81	0.17 ± 0.02	100.0	0.137 ± 0.097	0.020 ± 0.044	0.013 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	01/95	5	0.99	1.15	0.12 ± 0.01	131.7	0.116 ± 0.085	0.014 ± 0.032	0.028 ± 0.016	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	02/95	5	0.99	1.62	0.13 ± 0.01	98.5	0.083 ± 0.060	0.012 ± 0.028	0.033 ± 0.020	0.000 ± 0.000	0.000 ± 0.000
DM1367	Phoenix SS	03/95	4	0.99	1.23	0.07 ± 0.01	165.7	0.106 ± 0.074	0.008 ± 0.018	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	04/94	5	0.98	1.95	0.01 ± 0.00	130.0	0.011 ± 0.009	0.000 ± 0.003	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	05/94	5	0.98	1.15	0.01 ± 0.00	120.0	0.012 ± 0.009	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	06/94	5	0.97	1.24	0.01 ± 0.00	100.0	0.010 ± 0.008	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.00		0.010 ± 0.008	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.00		0.012 ± 0.010	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.00		0.009 ± 0.009	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
DMN12	Phoenix SS	10/94	5	0.98	1.24	0.01 ± 0.00	210.0	0.021 ± 0.017	0.000 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	11/94	5	0.99	1.25	0.01 ± 0.00	180.0	0.015 ± 0.012	0.000 ± 0.005	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	12/94	5	1.00	0.81	0.02 ± 0.00	110.0	0.020 ± 0.016	0.000 ± 0.007	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	01/95	5	0.99	1.15	0.01 ± 0.00	200.0	0.017 ± 0.014	0.000 ± 0.005	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	02/95	5	0.99	1.62	0.02 ± 0.00	80.0	0.012 ± 0.010	0.000 ± 0.004	0.004 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
DMN12	Phoenix SS	03/95	4	0.99	1.23	0.01 ± 0.00	160.0	0.016 ± 0.012	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	04/94	5	0.98	1.95	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.00		0.000 ± 0.000	0.003 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	06/94	5	0.97	1.24	0.00 ± 0.00		0.000 ± 0.000	0.003 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.00		0.000 ± 0.000	0.003 ± 0.007	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.00		0.000 ± 0.000	0.002 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.00		0.000 ± 0.001	0.004 ± 0.011	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
DMN18	Phoenix SS	10/94	5	0.98	1.24	0.00 ± 0.00		0.000 ± 0.001	0.006 ± 0.014	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	11/94	5	0.99	1.25	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	12/94	5	1.00	0.81	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	01/95	5	0.99	1.15	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.004	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	02/95	5	0.99	1.62	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.004	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN18	Phoenix SS	03/95	4	0.99	1.23	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	04/94	5	0.98	1.95	0.04 ± 0.00	152.5	0.045 ± 0.032	0.008 ± 0.011	0.008 ± 0.007	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	05/94	5	0.98	1.15	0.04 ± 0.00	125.0	0.049 ± 0.035	0.001 ± 0.017	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	06/94	5	0.97	1.24	0.03 ± 0.00	140.0	0.040 ± 0.030	0.001 ± 0.016	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	07/94	6	0.99	0.71	0.04 ± 0.00	102.5	0.040 ± 0.028	0.001 ± 0.015	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
DMN267	Phoenix SS	08/94	5	0.97	1.42	0.03 ± 0.00	170.0	0.051 ± 0.037	0.000 ± 0.010	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	09/94	4	0.98	0.90	0.04 ± 0.01	95.0	0.037 ± 0.032	0.001 ± 0.024	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
DMN267	Phoenix SS	10/94	5	0.98	1.24	0.06 ± 0.01	145.0	0.084 ± 0.065	0.002 ± 0.030	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	11/94	5	0.99	1.25	0.07 ± 0.01	120.0	0.059 ± 0.046	0.014 ± 0.017	0.011 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	12/94	5	1.00	0.81	0.11 ± 0.01	100.0	0.080 ± 0.058	0.024 ± 0.026	0.006 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	01/95	5	0.99	1.15	0.07 ± 0.01	140.0	0.068 ± 0.051	0.017 ± 0.019	0.013 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	02/95	5	0.99	1.62	0.08 ± 0.01	100.0	0.049 ± 0.036	0.015 ± 0.017	0.016 ± 0.011	0.000 ± 0.000	0.000 ± 0.000
DMN267	Phoenix SS	03/95	4	0.99	1.23	0.04 ± 0.00	182.5	0.062 ± 0.044	0.010 ± 0.010	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ETBZ	Phoenix SS	04/94	5	0.98	1.95	2.27 ± 0.11	104.4	0.149 ± 0.033	2.164 ± 0.118	0.003 ± 0.014	0.000 ± 0.000	0.054 ± 0.014
ETBZ	Phoenix SS	05/94	5	0.98	1.15	2.72 ± 0.11	111.3	0.161 ± 0.035	2.841 ± 0.508	0.000 ± 0.000	0.000 ± 0.000	0.024 ± 0.006
ETBZ	Phoenix SS	06/94	5	0.97	1.24	2.42 ± 0.11	117.1	0.131 ± 0.030	2.675 ± 0.483	0.000 ± 0.001	0.000 ± 0.000	0.027 ± 0.007
ETBZ	Phoenix SS	07/94	6	0.99	0.71	2.35 ± 0.10	114.9	0.131 ± 0.028	2.539 ± 0.448	0.000 ± 0.001	0.000 ± 0.000	0.029 ± 0.007
ETBZ	Phoenix SS	08/94	5	0.97	1.42	1.66 ± 0.08	112.9	0.168 ± 0.037	1.690 ± 0.294	0.000 ± 0.001	0.000 ± 0.000	0.016 ± 0.005
ETBZ	Phoenix SS	09/94	4	0.98	0.90	3.60 ± 0.15	107.2	0.120 ± 0.033	3.680 ± 0.713	0.000 ± 0.001	0.000 ± 0.000	0.059 ± 0.017
ETBZ	Phoenix SS	10/94	5	0.98	1.24	5.15 ± 0.19	108.7	0.275 ± 0.066	5.275 ± 0.905	0.001 ± 0.002	0.000 ± 0.000	0.045 ± 0.011
ETBZ	Phoenix SS	11/94	5	0.99	1.25	3.65 ± 0.15	101.8	0.192 ± 0.046	3.514 ± 0.193	0.004 ± 0.015	0.000 ± 0.000	0.007 ± 0.002
ETBZ	Phoenix SS	12/94	5	1.00	0.81	6.47 ± 0.24	98.4	0.263 ± 0.059	6.073 ± 0.292	0.002 ± 0.008	0.000 ± 0.000	0.030 ± 0.007
ETBZ	Phoenix SS	01/95	5	0.99	1.15	4.68 ± 0.16	99.5	0.223 ± 0.052	4.410 ± 0.212	0.005 ± 0.017	0.000 ± 0.000	0.019 ± 0.005
ETBZ	Phoenix SS	02/95	5	0.99	1.62	3.87 ± 0.16	101.2	0.160 ± 0.037	3.721 ± 0.189	0.006 ± 0.021	0.000 ± 0.000	0.029 ± 0.007
ETBZ	Phoenix SS	03/95	4	0.99	1.23	2.72 ± 0.10	99.0	0.204 ± 0.045	2.470 ± 0.116	0.000 ± 0.002	0.000 ± 0.000	0.018 ± 0.005
FLUORA	Phoenix SS	04/94	5	0.98	1.95	0.01 ± 0.00	60.0	0.003 ± 0.002	0.001 ± 0.001	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.00		0.003 ± 0.002	0.007 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	06/94	5	0.97	1.24	0.01 ± 0.00	100.0	0.003 ± 0.002	0.007 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	07/94	6	0.99	0.71	0.01 ± 0.00	100.0	0.003 ± 0.002	0.007 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	08/94	5	0.97	1.42	0.01 ± 0.00	80.0	0.004 ± 0.002	0.004 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.00		0.002 ± 0.002	0.010 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
FLUORA	Phoenix SS	10/94	5	0.98	1.24	0.00 ± 0.00		0.005 ± 0.004	0.013 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	11/94	5	0.99	1.25	0.01 ± 0.00	80.0	0.004 ± 0.002	0.001 ± 0.001	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	12/94	5	1.00	0.81	0.01 ± 0.00	100.0	0.005 ± 0.004	0.003 ± 0.002	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	01/95	5	0.99	1.15	0.01 ± 0.00	90.0	0.004 ± 0.003	0.002 ± 0.001	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	02/95	5	0.99	1.62	0.01 ± 0.00	80.0	0.003 ± 0.002	0.001 ± 0.001	0.004 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Phoenix SS	03/95	4	0.99	1.23	0.00 ± 0.00		0.004 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	04/94	5	0.98	1.95	0.02 ± 0.00	95.0	0.015 ± 0.010	0.000 ± 0.002	0.004 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	05/94	5	0.98	1.15	0.01 ± 0.00	160.0	0.016 ± 0.011	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	06/94	5	0.97	1.24	0.02 ± 0.00	65.0	0.013 ± 0.010	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	07/94	6	0.99	0.71	0.02 ± 0.00	65.0	0.013 ± 0.009	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	08/94	5	0.97	1.42	0.02 ± 0.00	85.0	0.017 ± 0.012	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	09/94	4	0.98	0.90	0.01 ± 0.00	120.0	0.012 ± 0.011	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
FLUORE	Phoenix SS	10/94	5	0.98	1.24	0.01 ± 0.00	290.0	0.028 ± 0.021	0.000 ± 0.006	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	11/94	5	0.99	1.25	0.02 ± 0.00	125.0	0.019 ± 0.015	0.000 ± 0.004	0.006 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	12/94	5	1.00	0.81	0.02 ± 0.00	150.0	0.027 ± 0.019	0.000 ± 0.006	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	01/95	5	0.99	1.15	0.01 ± 0.00	290.0	0.023 ± 0.017	0.000 ± 0.004	0.006 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	02/95	5	0.99	1.62	0.02 ± 0.00	120.0	0.016 ± 0.012	0.000 ± 0.004	0.008 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Phoenix SS	03/95	4	0.99	1.23	0.01 ± 0.00	220.0	0.021 ± 0.014	0.000 ± 0.002	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	04/94	5	0.98	1.95	1.84 ± 0.16	89.2	0.000 ± 0.000	1.642 ± 0.300	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	05/94	5	0.98	1.15	4.36 ± 0.31	57.4	0.000 ± 0.001	2.502 ± 0.432	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	06/94	5	0.97	1.24	7.15 ± 0.49	33.0	0.000 ± 0.000	2.356 ± 0.411	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	07/94	6	0.99	0.71	8.70 ± 0.68	25.7	0.000 ± 0.000	2.235 ± 0.380	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	08/94	5	0.97	1.42	3.16 ± 0.23	47.1	0.000 ± 0.001	1.488 ± 0.250	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
FORMAL	Phoenix SS	09/94	4	0.98	0.90	4.79 ± 0.31	67.7	0.000 ± 0.001	3.241 ± 0.606	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
FORMAL	Phoenix SS	10/94	5	0.98	1.24	4.25 ± 0.28	109.3	0.000 ± 0.001	4.645 ± 0.770	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	11/94	5	0.99	1.25	3.36 ± 0.23	79.4	0.000 ± 0.001	2.667 ± 0.489	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	12/94	5	1.00	0.81	5.62 ± 0.44	82.0	0.000 ± 0.001	4.609 ± 0.739	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	01/95	5	0.99	1.15	4.41 ± 0.35	75.9	0.000 ± 0.001	3.346 ± 0.536	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	02/95	5	0.99	1.62	2.05 ± 0.15	137.8	0.000 ± 0.000	2.824 ± 0.478	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Phoenix SS	03/95	4	0.99	1.23	1.62 ± 0.13	115.7	0.000 ± 0.001	1.874 ± 0.295	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	04/94	5	0.98	1.95	1.38 ± 0.34	18.4	0.000 ± 0.000	0.254 ± 0.046	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	05/94	5	0.98	1.15	2.37 ± 0.44	16.3	0.000 ± 0.001	0.387 ± 0.067	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	06/94	5	0.97	1.24	1.93 ± 0.41	18.9	0.000 ± 0.000	0.364 ± 0.063	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.26		0.000 ± 0.000	0.345 ± 0.059	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.24		0.000 ± 0.001	0.230 ± 0.039	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	09/94	4	0.98	0.90	0.53 ± 0.29	94.5	0.000 ± 0.001	0.501 ± 0.094	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	10/94	5	0.98	1.24	0.74 ± 0.29	97.0	0.000 ± 0.001	0.718 ± 0.119	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
MEK	Phoenix SS	11/94	5	0.99	1.25	0.94 ± 0.32	43.8	0.000 ± 0.001	0.412 ± 0.075	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	12/94	5	1.00	0.81	0.68 ± 0.30	104.7	0.000 ± 0.001	0.712 ± 0.114	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	01/95	5	0.99	1.15	0.47 ± 0.28	110.0	0.000 ± 0.001	0.517 ± 0.083	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	02/95	5	0.99	1.62	0.97 ± 0.30	44.9	0.000 ± 0.000	0.436 ± 0.074	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Phoenix SS	03/95	4	0.99	1.23	1.69 ± 0.36	17.2	0.000 ± 0.001	0.290 ± 0.046	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	04/94	5	0.98	1.95	0.17 ± 0.01	94.7	0.050 ± 0.031	0.068 ± 0.025	0.043 ± 0.030	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	05/94	5	0.98	1.15	0.14 ± 0.01	129.3	0.054 ± 0.033	0.127 ± 0.114	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	06/94	5	0.97	1.24	0.16 ± 0.01	105.0	0.044 ± 0.029	0.120 ± 0.108	0.004 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	07/94	6	0.99	0.71	0.14 ± 0.01	113.6	0.043 ± 0.026	0.114 ± 0.101	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	08/94	5	0.97	1.42	0.12 ± 0.01	110.0	0.056 ± 0.035	0.076 ± 0.066	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	09/94	4	0.98	0.90	0.13 ± 0.02	158.5	0.040 ± 0.031	0.165 ± 0.160	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	10/94	5	0.98	1.24	0.26 ± 0.02	128.1	0.091 ± 0.062	0.236 ± 0.203	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.001
MNAPH1	Phoenix SS	11/94	5	0.99	1.25	0.27 ± 0.02	87.4	0.064 ± 0.044	0.110 ± 0.041	0.062 ± 0.032	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	12/94	5	1.00	0.81	0.32 ± 0.02	97.5	0.087 ± 0.056	0.190 ± 0.062	0.035 ± 0.018	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	01/95	5	0.99	1.15	0.30 ± 0.02	94.7	0.074 ± 0.049	0.138 ± 0.045	0.072 ± 0.036	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	02/95	5	0.99	1.62	0.29 ± 0.02	89.0	0.053 ± 0.035	0.117 ± 0.040	0.088 ± 0.046	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Phoenix SS	03/95	4	0.99	1.23	0.14 ± 0.01	107.9	0.068 ± 0.042	0.077 ± 0.025	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	04/94	5	0.98	1.95	0.35 ± 0.03	93.7	0.085 ± 0.049	0.151 ± 0.058	0.092 ± 0.069	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	05/94	5	0.98	1.15	0.27 ± 0.02	185.2	0.091 ± 0.053	0.409 ± 0.357	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	06/94	5	0.97	1.24	0.34 ± 0.03	137.6	0.074 ± 0.045	0.385 ± 0.339	0.009 ± 0.007	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	07/94	6	0.99	0.71	0.38 ± 0.03	117.1	0.074 ± 0.042	0.366 ± 0.314	0.005 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	08/94	5	0.97	1.42	0.26 ± 0.02	130.4	0.095 ± 0.055	0.243 ± 0.207	0.001 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	09/94	4	0.98	0.90	0.30 ± 0.04	199.7	0.068 ± 0.049	0.530 ± 0.500	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.001
MNAPH2	Phoenix SS	10/94	5	0.98	1.24	0.59 ± 0.05	157.5	0.156 ± 0.098	0.760 ± 0.635	0.013 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	11/94	5	0.99	1.25	0.59 ± 0.04	82.7	0.109 ± 0.068	0.245 ± 0.095	0.134 ± 0.073	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	12/94	5	1.00	0.81	0.69 ± 0.05	93.9	0.149 ± 0.087	0.424 ± 0.143	0.075 ± 0.041	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	01/95	5	0.99	1.15	0.65 ± 0.05	90.8	0.126 ± 0.076	0.308 ± 0.104	0.156 ± 0.082	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	02/95	5	0.99	1.62	0.70 ± 0.05	76.9	0.090 ± 0.054	0.259 ± 0.093	0.189 ± 0.105	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Phoenix SS	03/95	4	0.99	1.23	0.33 ± 0.03	91.2	0.116 ± 0.066	0.172 ± 0.057	0.013 ± 0.007	0.000 ± 0.000	0.000 ± 0.000
MP_XYL	Phoenix SS	04/94	5	0.98	1.95	8.46 ± 0.25	101.8	0.558 ± 0.082	7.860 ± 0.357	0.008 ± 0.038	0.000 ± 0.000	0.185 ± 0.040
MP_XYL	Phoenix SS	05/94	5	0.98	1.15	10.23 ± 0.27	115.2	0.602 ± 0.088	11.106 ± 2.335	0.000 ± 0.001	0.000 ± 0.000	0.081 ± 0.017
MP_XYL	Phoenix SS	06/94	5	0.97	1.24	9.10 ± 0.26	121.3	0.489 ± 0.075	10.458 ± 2.221	0.001 ± 0.004	0.000 ± 0.000	0.094 ± 0.020
MP_XYL	Phoenix SS	07/94	6	0.99	0.71	8.97 ± 0.24	117.2	0.489 ± 0.070	9.924 ± 2.056	0.000 ± 0.002	0.000 ± 0.000	0.100 ± 0.020
MP_XYL	Phoenix SS	08/94	5	0.97	1.42	5.74 ± 0.16	127.0	0.624 ± 0.093	6.607 ± 1.353	0.000 ± 0.002	0.000 ± 0.000	0.056 ± 0.013
MP_XYL	Phoenix SS	09/94	4	0.98	0.90	13.09 ± 0.39	114.9	0.447 ± 0.082	14.387 ± 3.275	0.000 ± 0.001	0.000 ± 0.000	0.203 ± 0.048

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
MP_XYL	Phoenix SS	10/94	5	0.98	1.24	18.98 ± 0.49	114.9	1.027 ± 0.165	20.624 ± 4.159	0.001 ± 0.004	0.000 ± 0.000	0.152 ± 0.031
MP_XYL	Phoenix SS	11/94	5	0.99	1.25	13.12 ± 0.38	103.0	0.716 ± 0.115	12.763 ± 0.582	0.012 ± 0.041	0.000 ± 0.000	0.025 ± 0.006
MP_XYL	Phoenix SS	12/94	5	1.00	0.81	23.32 ± 0.59	99.3	0.983 ± 0.147	22.056 ± 0.880	0.007 ± 0.023	0.000 ± 0.000	0.103 ± 0.019
MP_XYL	Phoenix SS	01/95	5	0.99	1.15	17.18 ± 0.44	98.5	0.832 ± 0.129	16.016 ± 0.638	0.014 ± 0.046	0.000 ± 0.000	0.064 ± 0.014
MP_XYL	Phoenix SS	02/95	5	0.99	1.62	14.30 ± 0.39	99.5	0.595 ± 0.091	13.512 ± 0.570	0.017 ± 0.058	0.000 ± 0.000	0.099 ± 0.020
MP_XYL	Phoenix SS	03/95	4	0.99	1.23	10.14 ± 0.26	96.6	0.762 ± 0.111	8.970 ± 0.351	0.001 ± 0.004	0.000 ± 0.000	0.062 ± 0.013
NAPHTH	Phoenix SS	04/94	5	0.98	1.95	0.72 ± 0.06	69.4	0.068 ± 0.033	0.257 ± 0.084	0.175 ± 0.127	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	05/94	5	0.98	1.15	0.46 ± 0.04	114.3	0.073 ± 0.036	0.453 ± 0.398	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	06/94	5	0.97	1.24	0.61 ± 0.07	82.6	0.060 ± 0.030	0.427 ± 0.378	0.017 ± 0.013	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	07/94	6	0.99	0.71	0.65 ± 0.05	72.8	0.059 ± 0.028	0.405 ± 0.350	0.009 ± 0.007	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	08/94	5	0.97	1.42	0.62 ± 0.05	56.0	0.076 ± 0.038	0.269 ± 0.230	0.002 ± 0.007	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	09/94	4	0.98	0.90	0.40 ± 0.05	160.8	0.054 ± 0.033	0.587 ± 0.558	0.002 ± 0.003	0.000 ± 0.000	0.000 ± 0.001
NAPHTH	Phoenix SS	10/94	5	0.98	1.24	0.89 ± 0.07	111.2	0.125 ± 0.067	0.841 ± 0.709	0.024 ± 0.014	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	11/94	5	0.99	1.25	1.15 ± 0.09	66.1	0.087 ± 0.046	0.418 ± 0.137	0.255 ± 0.135	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	12/94	5	1.00	0.81	1.09 ± 0.08	90.2	0.119 ± 0.059	0.722 ± 0.207	0.142 ± 0.076	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	01/95	5	0.99	1.15	1.24 ± 0.09	74.3	0.101 ± 0.052	0.524 ± 0.150	0.296 ± 0.152	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	02/95	5	0.99	1.62	1.36 ± 0.10	64.2	0.072 ± 0.037	0.442 ± 0.134	0.359 ± 0.195	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Phoenix SS	03/95	4	0.99	1.23	0.54 ± 0.04	76.1	0.093 ± 0.045	0.294 ± 0.083	0.024 ± 0.014	0.000 ± 0.000	0.000 ± 0.000
NMOC	Phoenix SS	04/94	5	0.98	1.95	203.93 ± 7.35	92.9	24.867 ± 10.197	132.041 ± 6.641	3.753 ± 1.724	0.000 ± 0.000	22.619 ± 3.644
NMOC	Phoenix SS	05/94	5	0.98	1.15	244.58 ± 8.23	99.9	26.827 ± 13.653	201.141 ± 12.614	0.009 ± 1.049	0.000 ± 0.000	9.852 ± 5.222
NMOC	Phoenix SS	06/94	5	0.97	1.24	229.52 ± 7.71	100.1	21.806 ± 12.122	189.407 ± 11.779	0.370 ± 0.975	0.000 ± 0.000	11.475 ± 5.067
NMOC	Phoenix SS	07/94	6	0.99	0.71	217.00 ± 7.07	101.1	21.788 ± 10.802	179.727 ± 10.731	0.202 ± 0.887	0.000 ± 0.000	12.257 ± 4.765
NMOC	Phoenix SS	08/94	5	0.97	1.42	199.24 ± 6.57	79.3	27.839 ± 11.510	119.655 ± 8.696	0.032 ± 1.002	0.000 ± 0.000	6.864 ± 3.423
NMOC	Phoenix SS	09/94	4	0.98	0.90	336.13 ± 11.92	93.0	19.907 ± 14.832	260.558 ± 16.809	0.049 ± 1.222	0.000 ± 0.000	24.766 ± 8.190
NMOC	Phoenix SS	10/94	5	0.98	1.24	411.91 ± 13.65	109.0	45.792 ± 22.904	373.518 ± 21.596	0.516 ± 2.050	0.000 ± 0.000	18.613 ± 9.306
NMOC	Phoenix SS	11/94	5	0.99	1.25	280.44 ± 10.03	93.9	31.949 ± 14.291	214.415 ± 9.189	5.463 ± 1.998	0.000 ± 0.000	3.118 ± 3.430
NMOC	Phoenix SS	12/94	5	1.00	0.81	455.30 ± 15.01	96.9	43.826 ± 18.940	370.530 ± 13.130	3.045 ± 1.956	0.000 ± 0.000	12.567 ± 5.114
NMOC	Phoenix SS	01/95	5	0.99	1.15	340.03 ± 11.19	96.8	37.085 ± 15.720	269.049 ± 10.110	6.334 ± 2.280	0.000 ± 0.000	7.859 ± 3.924
NMOC	Phoenix SS	02/95	5	0.99	1.62	280.82 ± 9.74	99.8	26.551 ± 13.140	226.994 ± 8.803	7.669 ± 2.478	0.000 ± 0.000	12.034 ± 3.653
NMOC	Phoenix SS	03/95	4	0.99	1.23	205.89 ± 6.66	95.3	33.948 ± 11.294	150.698 ± 6.718	0.509 ± 1.073	0.000 ± 0.000	7.524 ± 2.643
N_HEX	Phoenix SS	04/94	5	0.98	1.95	4.24 ± 0.14	90.4	0.304 ± 0.188	3.150 ± 1.262	0.000 ± 0.000	0.000 ± 0.000	0.380 ± 0.078
N_HEX	Phoenix SS	05/94	5	0.98	1.15	4.35 ± 0.14	210.8	0.328 ± 0.202	8.676 ± 3.401	0.000 ± 0.000	0.000 ± 0.000	0.165 ± 0.033
N_HEX	Phoenix SS	06/94	5	0.97	1.24	4.30 ± 0.15	200.7	0.266 ± 0.173	8.170 ± 3.235	0.000 ± 0.000	0.000 ± 0.000	0.193 ± 0.038
N_HEX	Phoenix SS	07/94	6	0.99	0.71	4.04 ± 0.14	203.6	0.266 ± 0.160	7.752 ± 2.994	0.000 ± 0.000	0.000 ± 0.000	0.206 ± 0.038
N_HEX	Phoenix SS	08/94	5	0.97	1.42	3.96 ± 0.14	141.8	0.340 ± 0.214	5.161 ± 1.970	0.000 ± 0.000	0.000 ± 0.000	0.115 ± 0.026
N_HEX	Phoenix SS	09/94	4	0.98	0.90	7.34 ± 0.25	162.1	0.243 ± 0.187	11.238 ± 4.771	0.000 ± 0.000	0.000 ± 0.000	0.416 ± 0.092
N_HEX	Phoenix SS	10/94	5	0.98	1.24	9.07 ± 0.29	187.2	0.559 ± 0.378	16.110 ± 6.058	0.000 ± 0.000	0.000 ± 0.000	0.313 ± 0.060
N_HEX	Phoenix SS	11/94	5	0.99	1.25	6.21 ± 0.22	89.5	0.390 ± 0.265	5.116 ± 2.059	0.000 ± 0.000	0.000 ± 0.000	0.052 ± 0.011
N_HEX	Phoenix SS	12/94	5	1.00	0.81	9.88 ± 0.31	97.0	0.535 ± 0.337	8.840 ± 3.112	0.000 ± 0.000	0.000 ± 0.000	0.211 ± 0.036
N_HEX	Phoenix SS	01/95	5	0.99	1.15	8.55 ± 0.28	81.9	0.453 ± 0.295	6.419 ± 2.257	0.000 ± 0.000	0.000 ± 0.000	0.132 ± 0.026
N_HEX	Phoenix SS	02/95	5	0.99	1.62	5.99 ± 0.19	99.2	0.324 ± 0.209	5.415 ± 2.015	0.000 ± 0.000	0.000 ± 0.000	0.202 ± 0.038
N_HEX	Phoenix SS	03/95	4	0.99	1.23	4.83 ± 0.15	85.7	0.415 ± 0.255	3.596 ± 1.242	0.000 ± 0.000	0.000 ± 0.000	0.126 ± 0.025
N_NON	Phoenix SS	04/94	5	0.98	1.95	0.60 ± 0.05	77.3	0.135 ± 0.053	0.323 ± 0.065	0.002 ± 0.008	0.000 ± 0.000	0.004 ± 0.008
N_NON	Phoenix SS	05/94	5	0.98	1.15	0.52 ± 0.05	116.2	0.146 ± 0.057	0.456 ± 0.313	0.000 ± 0.000	0.000 ± 0.000	0.002 ± 0.004
N_NON	Phoenix SS	06/94	5	0.97	1.24	0.42 ± 0.05	131.0	0.119 ± 0.049	0.429 ± 0.297	0.000 ± 0.001	0.000 ± 0.000	0.002 ± 0.004
N_NON	Phoenix SS	07/94	6	0.99	0.71	0.39 ± 0.05	135.4	0.119 ± 0.045	0.407 ± 0.275	0.000 ± 0.001	0.000 ± 0.000	0.002 ± 0.004
N_NON	Phoenix SS	08/94	5	0.97	1.42	0.41 ± 0.05	103.4	0.152 ± 0.060	0.271 ± 0.181	0.000 ± 0.000	0.000 ± 0.000	0.001 ± 0.003
N_NON	Phoenix SS	09/94	4	0.98	0.90	0.64 ± 0.05	110.2	0.109 ± 0.053	0.591 ± 0.438	0.000 ± 0.000	0.000 ± 0.000	0.005 ± 0.010
N_NON	Phoenix SS	10/94	5	0.98	1.24	0.70 ± 0.05	157.1	0.250 ± 0.107	0.846 ± 0.557	0.000 ± 0.001	0.000 ± 0.000	0.004 ± 0.007

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
N_NON	Phoenix SS	11/94	5	0.99	1.25	0.63 ± 0.05	111.4	0.174 ± 0.075	0.524 ± 0.106	0.003 ± 0.009	0.000 ± 0.000	0.001 ± 0.001
N_NON	Phoenix SS	12/94	5	1.00	0.81	0.93 ± 0.06	123.5	0.239 ± 0.095	0.905 ± 0.160	0.002 ± 0.005	0.000 ± 0.000	0.003 ± 0.004
N_NON	Phoenix SS	01/95	5	0.99	1.15	0.79 ± 0.05	109.4	0.202 ± 0.083	0.657 ± 0.116	0.003 ± 0.010	0.000 ± 0.000	0.002 ± 0.003
N_NON	Phoenix SS	02/95	5	0.99	1.62	0.51 ± 0.05	138.6	0.145 ± 0.059	0.555 ± 0.103	0.004 ± 0.013	0.000 ± 0.000	0.003 ± 0.004
N_NON	Phoenix SS	03/95	4	0.99	1.23	0.42 ± 0.05	132.1	0.185 ± 0.072	0.368 ± 0.064	0.000 ± 0.001	0.000 ± 0.000	0.002 ± 0.003
O_XYL	Phoenix SS	04/94	5	0.98	1.95	3.33 ± 0.13	93.5	0.282 ± 0.046	2.759 ± 0.200	0.004 ± 0.022	0.000 ± 0.000	0.068 ± 0.017
O_XYL	Phoenix SS	05/94	5	0.98	1.15	3.94 ± 0.16	106.0	0.304 ± 0.049	3.843 ± 0.494	0.000 ± 0.001	0.000 ± 0.000	0.029 ± 0.007
O_XYL	Phoenix SS	06/94	5	0.97	1.24	3.34 ± 0.14	116.8	0.247 ± 0.043	3.619 ± 0.470	0.000 ± 0.002	0.000 ± 0.000	0.034 ± 0.009
O_XYL	Phoenix SS	07/94	6	0.99	0.71	3.31 ± 0.13	112.3	0.247 ± 0.039	3.434 ± 0.435	0.000 ± 0.001	0.000 ± 0.000	0.037 ± 0.009
O_XYL	Phoenix SS	08/94	5	0.97	1.42	2.28 ± 0.09	115.1	0.316 ± 0.052	2.287 ± 0.286	0.000 ± 0.001	0.000 ± 0.000	0.021 ± 0.006
O_XYL	Phoenix SS	09/94	4	0.98	0.90	4.85 ± 0.21	108.8	0.226 ± 0.046	4.978 ± 0.693	0.000 ± 0.001	0.000 ± 0.000	0.074 ± 0.021
O_XYL	Phoenix SS	10/94	5	0.98	1.24	6.79 ± 0.26	113.6	0.519 ± 0.093	7.137 ± 0.880	0.001 ± 0.002	0.000 ± 0.000	0.056 ± 0.013
O_XYL	Phoenix SS	11/94	5	0.99	1.25	4.66 ± 0.18	104.2	0.362 ± 0.065	4.480 ± 0.327	0.006 ± 0.023	0.000 ± 0.000	0.009 ± 0.003
O_XYL	Phoenix SS	12/94	5	1.00	0.81	8.24 ± 0.29	100.5	0.497 ± 0.082	7.742 ± 0.494	0.003 ± 0.013	0.000 ± 0.000	0.038 ± 0.008
O_XYL	Phoenix SS	01/95	5	0.99	1.15	6.30 ± 0.23	96.4	0.420 ± 0.072	5.621 ± 0.358	0.006 ± 0.027	0.000 ± 0.000	0.024 ± 0.006
O_XYL	Phoenix SS	02/95	5	0.99	1.62	5.17 ± 0.18	98.4	0.301 ± 0.051	4.743 ± 0.319	0.008 ± 0.034	0.000 ± 0.000	0.036 ± 0.008
O_XYL	Phoenix SS	03/95	4	0.99	1.23	3.65 ± 0.13	97.5	0.385 ± 0.062	3.148 ± 0.197	0.001 ± 0.002	0.000 ± 0.000	0.023 ± 0.006
PA224M	Phoenix SS	04/94	5	0.98	1.95	3.13 ± 0.12	102.9	0.195 ± 0.066	2.799 ± 0.279	0.059 ± 0.031	0.000 ± 0.000	0.167 ± 0.245
PA224M	Phoenix SS	05/94	5	0.98	1.15	3.96 ± 0.14	88.3	0.211 ± 0.071	3.213 ± 0.371	0.000 ± 0.001	0.000 ± 0.000	0.073 ± 0.104
PA224M	Phoenix SS	06/94	5	0.97	1.24	3.47 ± 0.12	94.8	0.171 ± 0.060	3.026 ± 0.353	0.006 ± 0.003	0.000 ± 0.000	0.085 ± 0.120
PA224M	Phoenix SS	07/94	6	0.99	0.71	3.15 ± 0.10	99.6	0.171 ± 0.056	2.871 ± 0.326	0.003 ± 0.002	0.000 ± 0.000	0.091 ± 0.121
PA224M	Phoenix SS	08/94	5	0.97	1.42	2.14 ± 0.08	102.0	0.219 ± 0.075	1.912 ± 0.215	0.001 ± 0.002	0.000 ± 0.000	0.051 ± 0.081
PA224M	Phoenix SS	09/94	4	0.98	0.90	4.60 ± 0.16	97.9	0.157 ± 0.065	4.162 ± 0.520	0.001 ± 0.001	0.000 ± 0.000	0.183 ± 0.291
PA224M	Phoenix SS	10/94	5	0.98	1.24	7.64 ± 0.25	84.7	0.360 ± 0.132	5.967 ± 0.660	0.008 ± 0.004	0.000 ± 0.000	0.138 ± 0.188
PA224M	Phoenix SS	11/94	5	0.99	1.25	4.88 ± 0.17	100.5	0.251 ± 0.092	4.545 ± 0.455	0.085 ± 0.033	0.000 ± 0.000	0.023 ± 0.036
PA224M	Phoenix SS	12/94	5	1.00	0.81	8.55 ± 0.26	97.5	0.344 ± 0.117	7.853 ± 0.688	0.047 ± 0.018	0.000 ± 0.000	0.093 ± 0.114
PA224M	Phoenix SS	01/95	5	0.99	1.15	6.38 ± 0.21	96.4	0.291 ± 0.103	5.703 ± 0.499	0.098 ± 0.037	0.000 ± 0.000	0.058 ± 0.082
PA224M	Phoenix SS	02/95	5	0.99	1.62	5.11 ± 0.18	102.3	0.209 ± 0.073	4.811 ± 0.446	0.120 ± 0.047	0.000 ± 0.000	0.089 ± 0.119
PA224M	Phoenix SS	03/95	4	0.99	1.23	3.28 ± 0.10	107.5	0.267 ± 0.089	3.194 ± 0.275	0.008 ± 0.003	0.000 ± 0.000	0.056 ± 0.078
PAHSUM	Phoenix SS	04/94	5	0.98	1.95	1.46 ± 0.07	90.8	0.452 ± 0.098	0.501 ± 0.108	0.373 ± 0.148	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	05/94	5	0.98	1.15	1.08 ± 0.05	138.2	0.487 ± 0.106	1.006 ± 0.547	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	06/94	5	0.97	1.24	1.35 ± 0.08	102.4	0.397 ± 0.091	0.948 ± 0.520	0.037 ± 0.015	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	07/94	6	0.99	0.71	1.38 ± 0.06	95.2	0.395 ± 0.084	0.902 ± 0.481	0.017 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	08/94	5	0.97	1.42	1.19 ± 0.06	93.0	0.507 ± 0.112	0.597 ± 0.317	0.003 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	09/94	4	0.98	0.90	1.00 ± 0.07	167.0	0.361 ± 0.098	1.305 ± 0.767	0.004 ± 0.003	0.000 ± 0.000	0.000 ± 0.004
PAHSUM	Phoenix SS	10/94	5	0.98	1.24	2.00 ± 0.09	137.5	0.830 ± 0.197	1.868 ± 0.974	0.052 ± 0.017	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	11/94	5	0.99	1.25	2.37 ± 0.10	81.8	0.581 ± 0.138	0.815 ± 0.176	0.543 ± 0.158	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	12/94	5	1.00	0.81	2.60 ± 0.10	96.5	0.795 ± 0.176	1.410 ± 0.265	0.303 ± 0.089	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	01/95	5	0.99	1.15	2.56 ± 0.11	90.9	0.674 ± 0.154	1.023 ± 0.192	0.629 ± 0.178	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	02/95	5	0.99	1.62	2.74 ± 0.11	76.9	0.481 ± 0.109	0.862 ± 0.172	0.764 ± 0.228	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Phoenix SS	03/95	4	0.99	1.23	1.20 ± 0.05	103.8	0.621 ± 0.134	0.572 ± 0.106	0.052 ± 0.016	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	04/94	5	0.98	1.95	0.03 ± 0.00	116.7	0.029 ± 0.021	0.000 ± 0.002	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	05/94	5	0.98	1.15	0.03 ± 0.00	106.7	0.031 ± 0.023	0.001 ± 0.011	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	06/94	5	0.97	1.24	0.04 ± 0.00	65.0	0.025 ± 0.019	0.000 ± 0.010	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	07/94	6	0.99	0.71	0.04 ± 0.00	65.0	0.025 ± 0.018	0.001 ± 0.010	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	08/94	5	0.97	1.42	0.04 ± 0.00	80.0	0.032 ± 0.024	0.000 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	09/94	4	0.98	0.90	0.02 ± 0.00	120.0	0.023 ± 0.021	0.001 ± 0.015	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
PHENAN	Phoenix SS	10/94	5	0.98	1.24	0.02 ± 0.00	275.0	0.053 ± 0.042	0.001 ± 0.020	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	11/94	5	0.99	1.25	0.03 ± 0.00	153.3	0.037 ± 0.030	0.000 ± 0.004	0.009 ± 0.003	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
PHENAN	Phoenix SS	12/94	5	1.00	0.81	0.03 ± 0.00	186.7	0.051 ± 0.038	0.000 ± 0.006	0.005 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	01/95	5	0.99	1.15	0.03 ± 0.00	180.0	0.043 ± 0.033	0.000 ± 0.004	0.011 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	02/95	5	0.99	1.62	0.03 ± 0.00	143.3	0.030 ± 0.023	0.000 ± 0.004	0.013 ± 0.005	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Phoenix SS	03/95	4	0.99	1.23	0.02 ± 0.00	200.0	0.039 ± 0.029	0.000 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	04/94	5	0.98	1.95	1.33 ± 0.15	6.8	0.000 ± 0.000	0.090 ± 0.016	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	05/94	5	0.98	1.15	1.98 ± 0.21	6.9	0.000 ± 0.001	0.136 ± 0.024	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	06/94	5	0.97	1.24	1.85 ± 0.20	6.9	0.000 ± 0.000	0.128 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	07/94	6	0.99	0.71	1.88 ± 0.20	6.5	0.000 ± 0.000	0.122 ± 0.021	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	08/94	5	0.97	1.42	1.47 ± 0.17	5.5	0.000 ± 0.001	0.081 ± 0.014	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	09/94	4	0.98	0.90	1.46 ± 0.16	12.1	0.000 ± 0.001	0.177 ± 0.033	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
PROAL	Phoenix SS	10/94	5	0.98	1.24	1.97 ± 0.21	12.8	0.000 ± 0.001	0.253 ± 0.042	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	11/94	5	0.99	1.25	1.23 ± 0.14	11.8	0.000 ± 0.001	0.145 ± 0.027	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	12/94	5	1.00	0.81	1.21 ± 0.14	20.7	0.000 ± 0.001	0.251 ± 0.040	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	01/95	5	0.99	1.15	1.21 ± 0.15	15.0	0.000 ± 0.001	0.182 ± 0.029	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	02/95	5	0.99	1.62	0.80 ± 0.10	19.3	0.000 ± 0.000	0.154 ± 0.026	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Phoenix SS	03/95	4	0.99	1.23	0.96 ± 0.11	10.6	0.000 ± 0.001	0.102 ± 0.016	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	04/94	5	0.98	1.95	0.00 ± 0.00		0.004 ± 0.003	0.000 ± 0.001	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.00		0.005 ± 0.003	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	06/94	5	0.97	1.24	0.00 ± 0.00		0.004 ± 0.002	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.00		0.004 ± 0.002	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.00		0.005 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.00		0.003 ± 0.003	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
PYRENE	Phoenix SS	10/94	5	0.98	1.24	0.00 ± 0.00		0.007 ± 0.005	0.003 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	11/94	5	0.99	1.25	0.00 ± 0.00		0.005 ± 0.004	0.000 ± 0.001	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	12/94	5	1.00	0.81	0.01 ± 0.00	90.0	0.007 ± 0.005	0.000 ± 0.002	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	01/95	5	0.99	1.15	0.01 ± 0.00	90.0	0.006 ± 0.004	0.000 ± 0.002	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	02/95	5	0.99	1.62	0.00 ± 0.00		0.005 ± 0.003	0.000 ± 0.001	0.004 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Phoenix SS	03/95	4	0.99	1.23	0.00 ± 0.00		0.006 ± 0.003	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	04/94	5	0.98	1.95	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.004 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	05/94	5	0.98	1.15	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	06/94	5	0.97	1.24	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	07/94	6	0.99	0.71	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	08/94	5	0.97	1.42	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	09/94	4	0.98	0.90	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
RETENE	Phoenix SS	10/94	5	0.98	1.24	0.00 ± 0.00		0.001 ± 0.002	0.000 ± 0.001	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	11/94	5	0.99	1.25	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.001	0.005 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	12/94	5	1.00	0.81	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.001	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	01/95	5	0.99	1.15	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.001	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	02/95	5	0.99	1.62	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.007 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
RETENE	Phoenix SS	03/95	4	0.99	1.23	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
STYR	Phoenix SS	04/94	5	0.98	1.95	1.23 ± 0.10	69.0	0.447 ± 0.095	0.349 ± 0.106	0.051 ± 0.012	0.000 ± 0.000	0.002 ± 0.023
STYR	Phoenix SS	05/94	5	0.98	1.15	1.13 ± 0.09	84.9	0.482 ± 0.102	0.476 ± 0.260	0.000 ± 0.000	0.000 ± 0.000	0.001 ± 0.010
STYR	Phoenix SS	06/94	5	0.97	1.24	1.09 ± 0.09	77.6	0.392 ± 0.088	0.448 ± 0.247	0.005 ± 0.001	0.000 ± 0.000	0.001 ± 0.011
STYR	Phoenix SS	07/94	6	0.99	0.71	1.06 ± 0.09	77.4	0.391 ± 0.081	0.425 ± 0.229	0.003 ± 0.001	0.000 ± 0.000	0.001 ± 0.011
STYR	Phoenix SS	08/94	5	0.97	1.42	1.24 ± 0.09	63.2	0.500 ± 0.109	0.283 ± 0.150	0.000 ± 0.001	0.000 ± 0.000	0.001 ± 0.008
STYR	Phoenix SS	09/94	4	0.98	0.90	1.28 ± 0.10	76.4	0.358 ± 0.095	0.617 ± 0.365	0.001 ± 0.000	0.000 ± 0.000	0.002 ± 0.027
STYR	Phoenix SS	10/94	5	0.98	1.24	1.42 ± 0.11	120.8	0.822 ± 0.192	0.884 ± 0.463	0.007 ± 0.002	0.000 ± 0.000	0.002 ± 0.018
STYR	Phoenix SS	11/94	5	0.99	1.25	1.23 ± 0.10	98.7	0.574 ± 0.134	0.566 ± 0.172	0.074 ± 0.013	0.000 ± 0.000	0.000 ± 0.003
STYR	Phoenix SS	12/94	5	1.00	0.81	1.67 ± 0.11	108.3	0.787 ± 0.171	0.978 ± 0.260	0.041 ± 0.007	0.000 ± 0.000	0.002 ± 0.011

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
STYR	Phoenix SS	01/95	5	0.99	1.15	1.49 ± 0.10	98.2	0.666 ± 0.150	0.710 ± 0.189	0.086 ± 0.014	0.000 ± 0.000	0.001 ± 0.008
STYR	Phoenix SS	02/95	5	0.99	1.62	1.13 ± 0.09	104.5	0.477 ± 0.106	0.599 ± 0.168	0.104 ± 0.018	0.000 ± 0.000	0.001 ± 0.011
STYR	Phoenix SS	03/95	4	0.99	1.23	1.07 ± 0.09	95.0	0.610 ± 0.129	0.398 ± 0.104	0.007 ± 0.001	0.000 ± 0.000	0.001 ± 0.007
TOLUE	Phoenix SS	04/94	5	0.98	1.95	13.89 ± 0.38	104.3	1.018 ± 0.313	12.980 ± 0.619	0.000 ± 0.000	0.000 ± 0.000	0.493 ± 0.196
TOLUE	Phoenix SS	05/94	5	0.98	1.15	16.31 ± 0.42	130.0	1.098 ± 0.336	19.887 ± 7.062	0.000 ± 0.000	0.000 ± 0.000	0.215 ± 0.083
TOLUE	Phoenix SS	06/94	5	0.97	1.24	14.52 ± 0.39	136.8	0.892 ± 0.288	18.728 ± 6.719	0.000 ± 0.000	0.000 ± 0.000	0.250 ± 0.096
TOLUE	Phoenix SS	07/94	6	0.99	0.71	14.27 ± 0.36	132.6	0.892 ± 0.267	17.770 ± 6.218	0.000 ± 0.000	0.000 ± 0.000	0.267 ± 0.097
TOLUE	Phoenix SS	08/94	5	0.97	1.42	9.88 ± 0.25	132.8	1.139 ± 0.357	11.831 ± 4.092	0.000 ± 0.000	0.000 ± 0.000	0.150 ± 0.065
TOLUE	Phoenix SS	09/94	4	0.98	0.90	20.78 ± 0.59	130.5	0.815 ± 0.312	25.762 ± 9.909	0.000 ± 0.000	0.000 ± 0.000	0.540 ± 0.232
TOLUE	Phoenix SS	10/94	5	0.98	1.24	29.67 ± 0.73	132.2	1.874 ± 0.629	36.931 ± 12.582	0.000 ± 0.000	0.000 ± 0.000	0.406 ± 0.150
TOLUE	Phoenix SS	11/94	5	0.99	1.25	21.92 ± 0.60	102.4	1.307 ± 0.440	21.078 ± 1.010	0.000 ± 0.000	0.000 ± 0.000	0.068 ± 0.029
TOLUE	Phoenix SS	12/94	5	1.00	0.81	37.06 ± 0.89	103.9	1.794 ± 0.561	36.425 ± 1.527	0.000 ± 0.000	0.000 ± 0.000	0.274 ± 0.091
TOLUE	Phoenix SS	01/95	5	0.99	1.15	26.53 ± 0.64	106.1	1.518 ± 0.491	26.449 ± 1.107	0.000 ± 0.000	0.000 ± 0.000	0.171 ± 0.065
TOLUE	Phoenix SS	02/95	5	0.99	1.62	22.80 ± 0.59	103.8	1.086 ± 0.347	22.315 ± 0.988	0.000 ± 0.000	0.000 ± 0.000	0.262 ± 0.095
TOLUE	Phoenix SS	03/95	4	0.99	1.23	15.76 ± 0.38	103.9	1.389 ± 0.424	14.814 ± 0.610	0.000 ± 0.000	0.000 ± 0.000	0.164 ± 0.062

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
ACETAL	Tucson	06/95	4	0.87	5.03	3.53 ± 0.27	9.3	0.000 ± 0.001	0.327 ± 0.051	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Tucson	07/95	4	0.88	4.28	2.66 ± 0.22	13.0	0.000 ± 0.001	0.345 ± 0.065	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Tucson	08/95	5	0.94	1.59	0.62 ± 0.10	51.5	0.000 ± 0.000	0.319 ± 0.059	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Tucson	06/95	4	0.87	5.03	0.61 ± 0.73	50.5	0.000 ± 0.001	0.308 ± 0.047	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Tucson	07/95	4	0.88	4.28	0.55 ± 0.76	59.1	0.000 ± 0.001	0.325 ± 0.061	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Tucson	08/95	5	0.94	1.59	0.06 ± 0.60	500.0	0.000 ± 0.000	0.300 ± 0.055	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Tucson	06/95	4	0.87	5.03	0.01 ± 0.00	40.0	0.003 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Tucson	07/95	4	0.88	4.28	0.01 ± 0.00	40.0	0.003 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.001 ± 0.001	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Tucson	06/95	4	0.87	5.03	0.00 ± 0.00		0.010 ± 0.010	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Tucson	07/95	4	0.88	4.28	0.00 ± 0.00		0.008 ± 0.006	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.004 ± 0.004	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Tucson	06/95	4	0.87	5.03	0.00 ± 0.03		0.000 ± 0.001	0.039 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Tucson	07/95	4	0.88	4.28	0.04 ± 0.03	102.5	0.000 ± 0.001	0.041 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Tucson	08/95	5	0.94	1.59	0.00 ± 0.02		0.000 ± 0.000	0.038 ± 0.007	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Tucson	06/95	4	0.87	5.03	0.00 ± 0.00		0.005 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Tucson	07/95	4	0.88	4.28	0.01 ± 0.00	40.0	0.004 ± 0.003	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BENZE	Tucson	06/95	4	0.87	5.03	2.53 ± 0.07	101.2	0.656 ± 0.348	1.575 ± 0.206	0.000 ± 0.000	0.330 ± 0.154	0.000 ± 0.000
BENZE	Tucson	07/95	4	0.88	4.28	2.00 ± 0.06	119.9	0.519 ± 0.225	1.661 ± 0.263	0.000 ± 0.000	0.185 ± 0.100	0.033 ± 0.008
BENZE	Tucson	08/95	5	0.94	1.59	2.49 ± 0.08	87.9	0.297 ± 0.143	1.533 ± 0.238	0.000 ± 0.000	0.279 ± 0.233	0.080 ± 0.009
BIPHEN	Tucson	06/95	4	0.87	5.03	0.01 ± 0.00	280.0	0.028 ± 0.022	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Tucson	07/95	4	0.88	4.28	0.01 ± 0.00	220.0	0.022 ± 0.015	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Tucson	08/95	5	0.94	1.59	0.01 ± 0.00	130.0	0.013 ± 0.009	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Tucson	06/95	4	0.87	5.03	0.14 ± 0.02	199.3	0.141 ± 0.099	0.138 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Tucson	07/95	4	0.88	4.28	0.15 ± 0.02	171.3	0.112 ± 0.064	0.145 ± 0.027	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Tucson	08/95	5	0.94	1.59	0.14 ± 0.02	141.4	0.064 ± 0.041	0.134 ± 0.025	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZ124M	Tucson	06/95	4	0.87	5.03	1.97 ± 0.14	104.5	0.578 ± 0.249	1.071 ± 0.240	0.000 ± 0.000	0.409 ± 0.388	0.000 ± 0.000
BZ124M	Tucson	07/95	4	0.88	4.28	1.86 ± 0.13	98.1	0.458 ± 0.160	1.129 ± 0.307	0.000 ± 0.000	0.229 ± 0.251	0.009 ± 0.009
BZ124M	Tucson	08/95	5	0.94	1.59	2.85 ± 0.23	58.6	0.262 ± 0.102	1.042 ± 0.278	0.000 ± 0.000	0.346 ± 0.585	0.021 ± 0.011
BZ135M	Tucson	06/95	4	0.87	5.03	0.46 ± 0.05	127.0	0.117 ± 0.024	0.337 ± 0.040	0.000 ± 0.000	0.130 ± 0.153	0.000 ± 0.000
BZ135M	Tucson	07/95	4	0.88	4.28	0.45 ± 0.05	116.4	0.092 ± 0.016	0.356 ± 0.050	0.000 ± 0.000	0.073 ± 0.099	0.003 ± 0.003
BZ135M	Tucson	08/95	5	0.94	1.59	0.49 ± 0.06	101.6	0.053 ± 0.010	0.328 ± 0.046	0.000 ± 0.000	0.110 ± 0.230	0.007 ± 0.003
CROTON	Tucson	06/95	4	0.87	5.03	0.05 ± 0.04	52.0	0.000 ± 0.001	0.026 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Tucson	07/95	4	0.88	4.28	0.69 ± 0.24	4.1	0.000 ± 0.001	0.028 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Tucson	08/95	5	0.94	1.59	0.21 ± 0.09	12.4	0.000 ± 0.000	0.026 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Tucson	06/95	4	0.87	5.03	0.01 ± 0.00	320.0	0.032 ± 0.027	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Tucson	07/95	4	0.88	4.28	0.01 ± 0.00	260.0	0.026 ± 0.018	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Tucson	08/95	5	0.94	1.59	0.01 ± 0.00	150.0	0.015 ± 0.011	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Tucson	06/95	4	0.87	5.03	0.06 ± 0.01	160.0	0.096 ± 0.084	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Tucson	07/95	4	0.88	4.28	0.04 ± 0.00	190.0	0.076 ± 0.054	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Tucson	08/95	5	0.94	1.59	0.02 ± 0.00	220.0	0.044 ± 0.034	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Tucson	06/95	4	0.87	5.03	0.00 ± 0.00		0.014 ± 0.013	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Tucson	07/95	4	0.88	4.28	0.00 ± 0.00		0.011 ± 0.009	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.006 ± 0.006	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Tucson	06/95	4	0.87	5.03	0.00 ± 0.00		0.000 ± 0.001	0.001 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Tucson	07/95	4	0.88	4.28	0.00 ± 0.00		0.000 ± 0.000	0.001 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.000 ± 0.000	0.001 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Tucson	06/95	4	0.87	5.03	0.02 ± 0.00	280.0	0.056 ± 0.050	0.000 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
DMN267	Tucson	07/95	4	0.88	4.28	0.02 ± 0.00	225.0	0.045 ± 0.032	0.000 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Tucson	08/95	5	0.94	1.59	0.01 ± 0.00	260.0	0.026 ± 0.021	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ETBZ	Tucson	06/95	4	0.87	5.03	1.40 ± 0.07	94.1	0.185 ± 0.051	0.818 ± 0.131	0.000 ± 0.000	0.314 ± 0.171	0.000 ± 0.000
ETBZ	Tucson	07/95	4	0.88	4.28	1.13 ± 0.06	105.6	0.147 ± 0.033	0.863 ± 0.167	0.000 ± 0.000	0.176 ± 0.111	0.007 ± 0.003
ETBZ	Tucson	08/95	5	0.94	1.59	1.05 ± 0.05	110.9	0.084 ± 0.021	0.796 ± 0.151	0.000 ± 0.000	0.266 ± 0.258	0.018 ± 0.004
FLUORA	Tucson	06/95	4	0.87	5.03	0.01 ± 0.00	60.0	0.004 ± 0.003	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Tucson	07/95	4	0.88	4.28	0.01 ± 0.00	50.0	0.003 ± 0.002	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.002 ± 0.001	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Tucson	06/95	4	0.87	5.03	0.01 ± 0.00	190.0	0.019 ± 0.016	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Tucson	07/95	4	0.88	4.28	0.01 ± 0.00	150.0	0.015 ± 0.010	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.009 ± 0.007	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Tucson	06/95	4	0.87	5.03	0.83 ± 0.11	86.9	0.000 ± 0.001	0.721 ± 0.111	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Tucson	07/95	4	0.88	4.28	0.83 ± 0.12	91.6	0.000 ± 0.001	0.760 ± 0.142	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Tucson	08/95	5	0.94	1.59	0.65 ± 0.10	107.8	0.000 ± 0.000	0.701 ± 0.129	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Tucson	06/95	4	0.87	5.03	0.89 ± 0.29	12.6	0.000 ± 0.001	0.112 ± 0.017	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Tucson	07/95	4	0.88	4.28	1.20 ± 0.35	9.8	0.000 ± 0.001	0.117 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Tucson	08/95	5	0.94	1.59	0.40 ± 0.23	27.0	0.000 ± 0.000	0.108 ± 0.020	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Tucson	06/95	4	0.87	5.03	0.08 ± 0.01	123.8	0.062 ± 0.047	0.037 ± 0.030	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Tucson	07/95	4	0.88	4.28	0.06 ± 0.01	146.7	0.049 ± 0.031	0.039 ± 0.038	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Tucson	08/95	5	0.94	1.59	0.05 ± 0.00	128.0	0.028 ± 0.019	0.036 ± 0.034	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Tucson	06/95	4	0.87	5.03	0.18 ± 0.01	123.9	0.105 ± 0.075	0.118 ± 0.092	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Tucson	07/95	4	0.88	4.28	0.12 ± 0.01	172.5	0.083 ± 0.048	0.124 ± 0.117	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Tucson	08/95	5	0.94	1.59	0.12 ± 0.01	134.2	0.047 ± 0.031	0.114 ± 0.106	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MP_XYL	Tucson	06/95	4	0.87	5.03	4.44 ± 0.13	108.8	0.690 ± 0.126	3.198 ± 0.600	0.000 ± 0.000	0.941 ± 0.529	0.000 ± 0.000
MP_XYL	Tucson	07/95	4	0.88	4.28	3.69 ± 0.11	121.2	0.547 ± 0.081	3.372 ± 0.769	0.000 ± 0.000	0.527 ± 0.342	0.025 ± 0.009
MP_XYL	Tucson	08/95	5	0.94	1.59	3.74 ± 0.15	114.5	0.313 ± 0.052	3.112 ± 0.696	0.000 ± 0.000	0.798 ± 0.797	0.060 ± 0.011
NAPHTH	Tucson	06/95	4	0.87	5.03	0.35 ± 0.03	61.4	0.084 ± 0.051	0.131 ± 0.102	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Tucson	07/95	4	0.88	4.28	0.22 ± 0.02	92.3	0.066 ± 0.033	0.137 ± 0.131	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Tucson	08/95	5	0.94	1.59	0.19 ± 0.02	86.8	0.038 ± 0.021	0.127 ± 0.119	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NMOC	Tucson	06/95	4	0.87	5.03	178.46 ± 7.20	59.6	30.761 ± 9.964	57.924 ± 10.183	0.000 ± 0.000	13.350 ± 6.269	0.000 ± 0.000
NMOC	Tucson	07/95	4	0.88	4.28	144.41 ± 4.87	73.2	24.365 ± 8.114	61.060 ± 7.119	0.000 ± 0.000	7.474 ± 3.059	3.035 ± 1.863
NMOC	Tucson	08/95	5	0.94	1.59	154.56 ± 6.30	64.7	13.954 ± 5.764	56.372 ± 4.969	0.000 ± 0.000	11.313 ± 2.799	7.326 ± 3.045
N_HEX	Tucson	06/95	4	0.87	5.03	2.66 ± 0.08	119.3	0.376 ± 0.289	2.499 ± 0.875	0.000 ± 0.000	0.282 ± 0.139	0.000 ± 0.000
N_HEX	Tucson	07/95	4	0.88	4.28	2.28 ± 0.08	139.5	0.298 ± 0.186	2.634 ± 1.119	0.000 ± 0.000	0.158 ± 0.090	0.051 ± 0.017
N_HEX	Tucson	08/95	5	0.94	1.59	1.99 ± 0.08	151.1	0.170 ± 0.119	2.431 ± 1.015	0.000 ± 0.000	0.239 ± 0.210	0.123 ± 0.021
N_NON	Tucson	06/95	4	0.87	5.03	0.39 ± 0.05	81.8	0.168 ± 0.082	0.131 ± 0.080	0.000 ± 0.000	0.020 ± 0.023	0.000 ± 0.000
N_NON	Tucson	07/95	4	0.88	4.28	0.29 ± 0.05	97.6	0.133 ± 0.053	0.138 ± 0.103	0.000 ± 0.000	0.011 ± 0.015	0.001 ± 0.002
N_NON	Tucson	08/95	5	0.94	1.59	0.25 ± 0.05	88.8	0.076 ± 0.033	0.128 ± 0.093	0.000 ± 0.000	0.017 ± 0.035	0.001 ± 0.002
O_XYL	Tucson	06/95	4	0.87	5.03	1.92 ± 0.08	94.7	0.349 ± 0.071	1.107 ± 0.127	0.000 ± 0.000	0.363 ± 0.222	0.000 ± 0.000
O_XYL	Tucson	07/95	4	0.88	4.28	1.44 ± 0.07	114.9	0.276 ± 0.046	1.167 ± 0.163	0.000 ± 0.000	0.203 ± 0.143	0.009 ± 0.004
O_XYL	Tucson	08/95	5	0.94	1.59	1.45 ± 0.09	107.9	0.158 ± 0.029	1.077 ± 0.147	0.000 ± 0.000	0.308 ± 0.334	0.022 ± 0.005
PA224M	Tucson	06/95	4	0.87	5.03	1.69 ± 0.15	87.0	0.242 ± 0.101	0.925 ± 0.095	0.000 ± 0.000	0.290 ± 0.245	0.000 ± 0.000
PA224M	Tucson	07/95	4	0.88	4.28	1.60 ± 0.08	86.4	0.192 ± 0.065	0.976 ± 0.122	0.000 ± 0.000	0.162 ± 0.158	0.023 ± 0.052
PA224M	Tucson	08/95	5	0.94	1.59	1.70 ± 0.16	79.0	0.110 ± 0.041	0.900 ± 0.111	0.000 ± 0.000	0.246 ± 0.369	0.054 ± 0.064
PAHSUM	Tucson	06/95	4	0.87	5.03	0.76 ± 0.03	111.8	0.559 ± 0.151	0.291 ± 0.141	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Tucson	07/95	4	0.88	4.28	0.54 ± 0.02	138.5	0.443 ± 0.098	0.305 ± 0.180	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Tucson	08/95	5	0.94	1.59	0.42 ± 0.02	127.1	0.253 ± 0.062	0.281 ± 0.163	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Tucson	06/95	4	0.87	5.03	0.02 ± 0.00	180.0	0.036 ± 0.032	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Tucson	07/95	4	0.88	4.28	0.02 ± 0.00	140.0	0.028 ± 0.021	0.000 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
PHENAN	Tucson	08/95	5	0.94	1.59	0.01 ± 0.00	160.0	0.016 ± 0.013	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Tucson	06/95	4	0.87	5.03	1.08 ± 0.12	3.6	0.000 ± 0.001	0.039 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Tucson	07/95	4	0.88	4.28	1.28 ± 0.14	3.2	0.000 ± 0.001	0.041 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Tucson	08/95	5	0.94	1.59	0.29 ± 0.06	13.1	0.000 ± 0.000	0.038 ± 0.007	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Tucson	06/95	4	0.87	5.03	0.00 ± 0.00		0.005 ± 0.004	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Tucson	07/95	4	0.88	4.28	0.00 ± 0.00		0.004 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Tucson	06/95	4	0.87	5.03	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Tucson	07/95	4	0.88	4.28	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Tucson	08/95	5	0.94	1.59	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
STYR	Tucson	06/95	4	0.87	5.03	4.14 ± 0.53	17.0	0.553 ± 0.146	0.137 ± 0.067	0.000 ± 0.000	0.013 ± 0.022	0.000 ± 0.000
STYR	Tucson	07/95	4	0.88	4.28	0.79 ± 0.09	74.8	0.438 ± 0.094	0.145 ± 0.086	0.000 ± 0.000	0.008 ± 0.014	0.000 ± 0.005
STYR	Tucson	08/95	5	0.94	1.59	1.11 ± 0.12	35.7	0.251 ± 0.060	0.133 ± 0.077	0.000 ± 0.000	0.011 ± 0.033	0.001 ± 0.006
TOLUE	Tucson	06/95	4	0.87	5.03	5.92 ± 0.16	139.7	1.259 ± 0.481	5.727 ± 1.817	0.000 ± 0.000	1.283 ± 0.568	0.000 ± 0.000
TOLUE	Tucson	07/95	4	0.88	4.28	6.27 ± 0.19	124.7	0.997 ± 0.310	6.037 ± 2.325	0.000 ± 0.000	0.718 ± 0.366	0.066 ± 0.042
TOLUE	Tucson	08/95	5	0.94	1.59	5.53 ± 0.15	133.7	0.571 ± 0.198	5.574 ± 2.107	0.000 ± 0.000	1.087 ± 0.855	0.160 ± 0.052

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
ACETAL	Casa Grande	06/95	5	0.82	8.42	3.50 ± 0.27	10.1	0.000 ± 0.000	0.352 ± 0.053	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Casa Grande	07/95	3	0.87	5.29	2.21 ± 0.20	19.3	0.000 ± 0.001	0.427 ± 0.064	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Casa Grande	08/95	5	0.88	4.68	1.60 ± 0.15	18.9	0.000 ± 0.000	0.302 ± 0.047	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Casa Grande	06/95	5	0.82	8.42	1.72 ± 0.76	19.2	0.000 ± 0.000	0.331 ± 0.050	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Casa Grande	07/95	3	0.87	5.29	0.45 ± 0.60	89.1	0.000 ± 0.001	0.401 ± 0.060	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Casa Grande	08/95	5	0.88	4.68	0.04 ± 0.64	710.0	0.000 ± 0.000	0.284 ± 0.044	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Casa Grande	06/95	5	0.82	8.42	0.01 ± 0.00	30.0	0.002 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Casa Grande	07/95	3	0.87	5.29	0.01 ± 0.00	50.0	0.004 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.001 ± 0.001	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Casa Grande	06/95	5	0.82	8.42	0.00 ± 0.00		0.006 ± 0.006	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Casa Grande	07/95	3	0.87	5.29	0.00 ± 0.00		0.011 ± 0.010	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.002 ± 0.002	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Casa Grande	06/95	5	0.82	8.42	0.00 ± 0.03		0.000 ± 0.000	0.042 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Casa Grande	07/95	3	0.87	5.29	0.02 ± 0.02	255.0	0.000 ± 0.001	0.051 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.03		0.000 ± 0.000	0.036 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Casa Grande	06/95	5	0.82	8.42	0.00 ± 0.00		0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Casa Grande	07/95	3	0.87	5.29	0.00 ± 0.00		0.006 ± 0.004	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BENZE	Casa Grande	06/95	5	0.82	8.42	1.69 ± 0.05	125.9	0.434 ± 0.203	1.693 ± 0.217	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BENZE	Casa Grande	07/95	3	0.87	5.29	2.08 ± 0.06	140.8	0.715 ± 0.371	2.054 ± 0.262	0.000 ± 0.000	0.118 ± 0.076	0.041 ± 0.005
BENZE	Casa Grande	08/95	5	0.88	4.68	1.42 ± 0.05	129.1	0.134 ± 0.076	1.454 ± 0.190	0.000 ± 0.000	0.203 ± 0.104	0.042 ± 0.008
BIPHEN	Casa Grande	06/95	5	0.82	8.42	0.01 ± 0.00	190.0	0.019 ± 0.013	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Casa Grande	07/95	3	0.87	5.29	0.01 ± 0.00	310.0	0.031 ± 0.024	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Casa Grande	08/95	5	0.88	4.68	0.01 ± 0.00	60.0	0.006 ± 0.005	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Casa Grande	06/95	5	0.82	8.42	0.16 ± 0.02	150.6	0.093 ± 0.057	0.148 ± 0.023	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Casa Grande	07/95	3	0.87	5.29	0.24 ± 0.02	138.8	0.154 ± 0.105	0.179 ± 0.027	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Casa Grande	08/95	5	0.88	4.68	0.12 ± 0.02	130.0	0.029 ± 0.021	0.127 ± 0.020	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZ124M	Casa Grande	06/95	5	0.82	8.42	4.87 ± 0.32	31.5	0.382 ± 0.145	1.151 ± 0.254	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZ124M	Casa Grande	07/95	3	0.87	5.29	5.64 ± 0.37	38.8	0.631 ± 0.265	1.397 ± 0.306	0.000 ± 0.000	0.147 ± 0.192	0.011 ± 0.007
BZ124M	Casa Grande	08/95	5	0.88	4.68	9.17 ± 0.72	14.9	0.118 ± 0.054	0.988 ± 0.222	0.000 ± 0.000	0.251 ± 0.262	0.011 ± 0.010
BZ135M	Casa Grande	06/95	5	0.82	8.42	0.36 ± 0.05	121.9	0.077 ± 0.014	0.362 ± 0.041	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZ135M	Casa Grande	07/95	3	0.87	5.29	0.73 ± 0.09	84.5	0.127 ± 0.026	0.440 ± 0.050	0.000 ± 0.000	0.046 ± 0.076	0.004 ± 0.002
BZ135M	Casa Grande	08/95	5	0.88	4.68	0.57 ± 0.07	73.5	0.024 ± 0.005	0.311 ± 0.036	0.000 ± 0.000	0.080 ± 0.103	0.004 ± 0.003
CROTON	Casa Grande	06/95	5	0.82	8.42	0.02 ± 0.03	140.0	0.000 ± 0.000	0.028 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Casa Grande	07/95	3	0.87	5.29	0.19 ± 0.07	17.9	0.000 ± 0.001	0.034 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Casa Grande	08/95	5	0.88	4.68	0.14 ± 0.06	17.1	0.000 ± 0.000	0.024 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Casa Grande	06/95	5	0.82	8.42	0.01 ± 0.00	210.0	0.021 ± 0.016	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Casa Grande	07/95	3	0.87	5.29	0.01 ± 0.00	350.0	0.035 ± 0.029	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Casa Grande	08/95	5	0.88	4.68	0.01 ± 0.00	60.0	0.006 ± 0.006	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Casa Grande	06/95	5	0.82	8.42	0.04 ± 0.00	160.0	0.064 ± 0.049	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Casa Grande	07/95	3	0.87	5.29	0.03 ± 0.00	350.0	0.105 ± 0.089	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Casa Grande	08/95	5	0.88	4.68	0.02 ± 0.00	100.0	0.020 ± 0.018	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Casa Grande	06/95	5	0.82	8.42	0.01 ± 0.00	90.0	0.009 ± 0.008	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Casa Grande	07/95	3	0.87	5.29	0.00 ± 0.00		0.015 ± 0.014	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.003 ± 0.003	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Casa Grande	06/95	5	0.82	8.42	0.00 ± 0.00		0.000 ± 0.000	0.001 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Casa Grande	07/95	3	0.87	5.29	0.00 ± 0.00		0.000 ± 0.001	0.001 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.000 ± 0.000	0.001 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Casa Grande	06/95	5	0.82	8.42	0.02 ± 0.00	185.0	0.037 ± 0.029	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
DMN267	Casa Grande	07/95	3	0.87	5.29	0.01 ± 0.00	620.0	0.062 ± 0.053	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Casa Grande	08/95	5	0.88	4.68	0.01 ± 0.00	120.0	0.012 ± 0.011	0.000 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ETBZ	Casa Grande	06/95	5	0.82	8.42	1.10 ± 0.05	91.0	0.122 ± 0.029	0.879 ± 0.138	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ETBZ	Casa Grande	07/95	3	0.87	5.29	1.10 ± 0.04	126.5	0.202 ± 0.054	1.067 ± 0.167	0.000 ± 0.000	0.113 ± 0.085	0.009 ± 0.003
ETBZ	Casa Grande	08/95	5	0.88	4.68	0.72 ± 0.04	138.2	0.038 ± 0.011	0.755 ± 0.121	0.000 ± 0.000	0.193 ± 0.116	0.009 ± 0.003
FLUORA	Casa Grande	06/95	5	0.82	8.42	0.01 ± 0.00	40.0	0.002 ± 0.002	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Casa Grande	07/95	3	0.87	5.29	0.01 ± 0.00	70.0	0.004 ± 0.003	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.001 ± 0.001	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Casa Grande	06/95	5	0.82	8.42	0.01 ± 0.00	120.0	0.012 ± 0.009	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Casa Grande	07/95	3	0.87	5.29	0.01 ± 0.00	210.0	0.021 ± 0.017	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.004 ± 0.004	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Casa Grande	06/95	5	0.82	8.42	0.76 ± 0.10	101.8	0.000 ± 0.000	0.774 ± 0.117	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Casa Grande	07/95	3	0.87	5.29	0.84 ± 0.11	111.8	0.000 ± 0.001	0.939 ± 0.142	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Casa Grande	08/95	5	0.88	4.68	0.66 ± 0.10	100.8	0.000 ± 0.000	0.665 ± 0.103	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Casa Grande	06/95	5	0.82	8.42	0.95 ± 0.30	12.5	0.000 ± 0.000	0.119 ± 0.018	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Casa Grande	07/95	3	0.87	5.29	1.00 ± 0.30	14.5	0.000 ± 0.001	0.145 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Casa Grande	08/95	5	0.88	4.68	0.47 ± 0.25	21.9	0.000 ± 0.000	0.103 ± 0.016	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Casa Grande	06/95	5	0.82	8.42	0.08 ± 0.01	100.0	0.041 ± 0.028	0.039 ± 0.031	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Casa Grande	07/95	3	0.87	5.29	0.05 ± 0.01	230.0	0.067 ± 0.051	0.048 ± 0.038	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Casa Grande	08/95	5	0.88	4.68	0.03 ± 0.00	153.3	0.012 ± 0.010	0.034 ± 0.027	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Casa Grande	06/95	5	0.82	8.42	0.18 ± 0.01	108.3	0.069 ± 0.044	0.126 ± 0.097	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Casa Grande	07/95	3	0.87	5.29	0.11 ± 0.01	242.7	0.114 ± 0.080	0.153 ± 0.117	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Casa Grande	08/95	5	0.88	4.68	0.07 ± 0.01	185.7	0.021 ± 0.016	0.109 ± 0.085	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MP_XYL	Casa Grande	06/95	5	0.82	8.42	3.29 ± 0.10	118.3	0.456 ± 0.073	3.437 ± 0.635	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MP_XYL	Casa Grande	07/95	3	0.87	5.29	4.12 ± 0.12	128.4	0.752 ± 0.134	4.170 ± 0.767	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MP_XYL	Casa Grande	08/95	5	0.88	4.68	2.59 ± 0.11	142.9	0.141 ± 0.027	2.951 ± 0.555	0.000 ± 0.000	0.338 ± 0.262	0.031 ± 0.007
NAPHTH	Casa Grande	06/95	5	0.82	8.42	0.30 ± 0.02	65.0	0.055 ± 0.030	0.140 ± 0.108	0.000 ± 0.000	0.578 ± 0.357	0.031 ± 0.009
NAPHTH	Casa Grande	07/95	3	0.87	5.29	0.39 ± 0.04	66.9	0.091 ± 0.054	0.170 ± 0.131	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Casa Grande	08/95	5	0.88	4.68	0.11 ± 0.01	124.5	0.017 ± 0.011	0.120 ± 0.095	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NMOC	Casa Grande	06/95	5	0.82	8.42	175.87 ± 6.24	51.0	20.339 ± 6.585	62.245 ± 4.400	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NMOC	Casa Grande	07/95	3	0.87	5.29	304.86 ± 10.87	94.6	33.547 ± 11.094	75.523 ± 8.607	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NMOC	Casa Grande	08/95	5	0.88	4.68	193.99 ± 7.86	41.5	6.269 ± 3.983	53.445 ± 6.618	0.000 ± 0.000	4.792 ± 4.009	3.781 ± 2.839
N_HEX	Casa Grande	06/95	5	0.82	8.42	3.60 ± 0.13	82.3	0.248 ± 0.168	2.685 ± 0.925	0.000 ± 0.000	8.206 ± 4.510	3.818 ± 1.760
N_HEX	Casa Grande	07/95	3	0.87	5.29	6.21 ± 0.21	61.9	0.410 ± 0.308	3.258 ± 1.117	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
N_HEX	Casa Grande	08/95	5	0.88	4.68	1.97 ± 0.09	134.6	0.076 ± 0.063	2.306 ± 0.809	0.000 ± 0.000	0.101 ± 0.069	0.064 ± 0.013
N_NON	Casa Grande	06/95	5	0.82	8.42	0.26 ± 0.05	96.9	0.111 ± 0.047	0.141 ± 0.085	0.000 ± 0.000	0.173 ± 0.094	0.064 ± 0.018
N_NON	Casa Grande	07/95	3	0.87	5.29	0.24 ± 0.05	150.8	0.183 ± 0.087	0.171 ± 0.103	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
N_NON	Casa Grande	08/95	5	0.88	4.68	0.49 ± 0.06	34.3	0.034 ± 0.018	0.121 ± 0.074	0.000 ± 0.000	0.007 ± 0.012	0.001 ± 0.001
O_XYL	Casa Grande	06/95	5	0.82	8.42	1.40 ± 0.07	101.4	0.231 ± 0.041	1.189 ± 0.134	0.000 ± 0.000	0.012 ± 0.016	0.001 ± 0.002
O_XYL	Casa Grande	07/95	3	0.87	5.29	1.64 ± 0.08	119.8	0.380 ± 0.075	1.443 ± 0.162	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
O_XYL	Casa Grande	08/95	5	0.88	4.68	1.06 ± 0.06	125.1	0.071 ± 0.015	1.021 ± 0.118	0.000 ± 0.000	0.130 ± 0.110	0.011 ± 0.003
PA224M	Casa Grande	06/95	5	0.82	8.42	1.67 ± 0.13	70.4	0.160 ± 0.058	0.994 ± 0.101	0.000 ± 0.000	0.223 ± 0.149	0.011 ± 0.004
PA224M	Casa Grande	07/95	3	0.87	5.29	1.62 ± 0.08	99.4	0.264 ± 0.107	1.206 ± 0.122	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PA224M	Casa Grande	08/95	5	0.88	4.68	1.21 ± 0.11	93.7	0.049 ± 0.022	0.854 ± 0.088	0.000 ± 0.000	0.104 ± 0.121	0.028 ± 0.041
PAHSUM	Casa Grande	06/95	5	0.82	8.42	0.71 ± 0.02	95.2	0.366 ± 0.089	0.310 ± 0.149	0.000 ± 0.000	0.178 ± 0.165	0.028 ± 0.056
PAHSUM	Casa Grande	07/95	3	0.87	5.29	0.66 ± 0.04	149.7	0.611 ± 0.161	0.377 ± 0.180	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Casa Grande	08/95	5	0.88	4.68	0.27 ± 0.01	141.1	0.114 ± 0.033	0.267 ± 0.130	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Casa Grande	06/95	5	0.82	8.42	0.03 ± 0.00	76.7	0.023 ± 0.019	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Casa Grande	07/95	3	0.87	5.29	0.02 ± 0.00	195.0	0.039 ± 0.035	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species • Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
PHENAN	Casa Grande	08/95	5	0.88	4.68	0.01 ± 0.00	70.0	0.007 ± 0.007	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Casa Grande	06/95	5	0.82	8.42	0.77 ± 0.10	5.5	0.000 ± 0.000	0.042 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Casa Grande	07/95	3	0.87	5.29	0.73 ± 0.11	7.0	0.000 ± 0.001	0.051 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Casa Grande	08/95	5	0.88	4.68	0.57 ± 0.09	6.3	0.000 ± 0.000	0.036 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Casa Grande	06/95	5	0.82	8.42	0.00 ± 0.00		0.003 ± 0.002	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Casa Grande	07/95	3	0.87	5.29	0.00 ± 0.00		0.006 ± 0.004	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Casa Grande	06/95	5	0.82	8.42	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Casa Grande	07/95	3	0.87	5.29	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Casa Grande	08/95	5	0.88	4.68	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
STYR	Casa Grande	06/95	5	0.82	8.42	3.31 ± 0.42	15.5	0.365 ± 0.085	0.147 ± 0.071	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
STYR	Casa Grande	07/95	3	0.87	5.29	1.15 ± 0.11	68.3	0.602 ± 0.156	0.179 ± 0.085	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
STYR	Casa Grande	08/95	5	0.88	4.68	1.88 ± 0.19	13.2	0.113 ± 0.032	0.127 ± 0.062	0.000 ± 0.000	0.005 ± 0.011	0.000 ± 0.004
TOLUE	Casa Grande	06/95	5	0.82	8.42	5.61 ± 0.13	124.5	0.832 ± 0.280	6.154 ± 1.920	0.000 ± 0.000	0.008 ± 0.015	0.000 ± 0.005
TOLUE	Casa Grande	07/95	3	0.87	5.29	8.42 ± 0.20	111.4	1.373 ± 0.512	7.467 ± 2.320	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
TOLUE	Casa Grande	08/95	5	0.88	4.68	6.60 ± 0.18	97.2	0.257 ± 0.104	5.284 ± 1.679	0.000 ± 0.000	0.460 ± 0.281	0.082 ± 0.032
											0.789 ± 0.383	0.083 ± 0.045

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
ACETAL	Payson	12/94	1	0.75	1.95	0.88 ± 0.12	0.8	0.000 ± 0.000	0.007 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	01/95	5	0.92	2.20	2.02 ± 0.24	29.1	0.000 ± 0.000	0.588 ± 0.098	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	02/95	3	0.98	1.21	2.09 ± 0.18	19.6	0.000 ± 0.000	0.409 ± 0.064	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	03/95	5	0.98	1.51	1.43 ± 0.28	28.0	0.000 ± 0.000	0.400 ± 0.066	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	04/95	4	0.91	1.80	1.80 ± 0.16	3.0	0.000 ± 0.000	0.054 ± 0.009	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	05/95	4	0.95	2.12	2.43 ± 0.20	17.0	0.000 ± 0.001	0.414 ± 0.068	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	06/95	4	0.92	3.47	3.47 ± 0.26	19.5	0.000 ± 0.001	0.677 ± 0.107	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	07/95	4	0.97	1.23	3.54 ± 0.27	11.2	0.000 ± 0.001	0.397 ± 0.062	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETAL	Payson	08/95	5	0.87	3.78	2.15 ± 0.18	9.7	0.000 ± 0.000	0.209 ± 0.042	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	12/94	1	0.75	1.95	0.32 ± 0.78	2.2	0.000 ± 0.000	0.007 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	01/95	5	0.92	2.20	6.03 ± 1.20	9.2	0.000 ± 0.000	0.552 ± 0.092	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	02/95	3	0.98	1.21	9.18 ± 1.42	4.2	0.000 ± 0.000	0.384 ± 0.061	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	03/95	5	0.98	1.51	0.97 ± 2.01	38.8	0.000 ± 0.000	0.376 ± 0.062	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	04/95	4	0.91	1.80	0.54 ± 0.76	9.3	0.000 ± 0.000	0.050 ± 0.009	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	05/95	4	0.95	2.12	0.29 ± 0.76	134.1	0.000 ± 0.001	0.389 ± 0.064	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	06/95	4	0.92	3.47	4.15 ± 0.81	15.3	0.000 ± 0.001	0.636 ± 0.101	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	07/95	4	0.97	1.23	1.17 ± 0.74	31.9	0.000 ± 0.001	0.373 ± 0.058	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACETO	Payson	08/95	5	0.87	3.78	0.14 ± 0.67	140.0	0.000 ± 0.000	0.196 ± 0.040	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	01/95	5	0.92	2.20	0.00 ± 0.00		0.001 ± 0.001	0.001 ± 0.001	0.005 ± 0.005	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	02/95	3	0.98	1.21	0.01 ± 0.00	60.0	0.001 ± 0.001	0.001 ± 0.000	0.004 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	03/95	5	0.98	1.51	0.00 ± 0.00		0.001 ± 0.001	0.001 ± 0.000	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	04/95	4	0.91	1.80	0.00 ± 0.00		0.002 ± 0.001	0.000 ± 0.000	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	05/95	4	0.95	2.12	0.01 ± 0.00	40.0	0.003 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	06/95	4	0.92	3.47	0.02 ± 0.00	30.0	0.004 ± 0.003	0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	07/95	4	0.97	1.23	0.03 ± 0.00	13.3	0.003 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPE	Payson	08/95	5	0.87	3.78	0.01 ± 0.00	20.0	0.001 ± 0.001	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.004 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	01/95	5	0.92	2.20	0.03 ± 0.01	116.7	0.002 ± 0.002	0.006 ± 0.002	0.027 ± 0.014	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	02/95	3	0.98	1.21	0.02 ± 0.01	145.0	0.003 ± 0.003	0.004 ± 0.001	0.022 ± 0.010	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	03/95	5	0.98	1.51	0.01 ± 0.00	190.0	0.003 ± 0.003	0.005 ± 0.001	0.011 ± 0.005	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	04/95	4	0.91	1.80	0.02 ± 0.01	55.0	0.004 ± 0.003	0.001 ± 0.000	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	05/95	4	0.95	2.12	0.00 ± 0.00		0.008 ± 0.007	0.000 ± 0.001	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	06/95	4	0.92	3.47	0.00 ± 0.00		0.011 ± 0.009	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	07/95	4	0.97	1.23	0.01 ± 0.00	90.0	0.009 ± 0.007	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACNAPY	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.004 ± 0.004	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	12/94	1	0.75	1.95	0.00 ± 0.03		0.000 ± 0.000	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	01/95	5	0.92	2.20	0.00 ± 0.02		0.000 ± 0.000	0.071 ± 0.012	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	02/95	3	0.98	1.21	0.00 ± 0.03		0.000 ± 0.000	0.049 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	03/95	5	0.98	1.51	0.12 ± 0.07	40.0	0.000 ± 0.000	0.048 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	04/95	4	0.91	1.80	0.00 ± 0.03		0.000 ± 0.000	0.007 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	05/95	4	0.95	2.12	0.02 ± 0.03	250.0	0.000 ± 0.001	0.050 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	06/95	4	0.92	3.47	0.00 ± 0.03		0.000 ± 0.001	0.081 ± 0.013	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	07/95	4	0.97	1.23	0.00 ± 0.03		0.000 ± 0.001	0.048 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ACROLN	Payson	08/95	5	0.87	3.78	0.00 ± 0.03		0.000 ± 0.000	0.025 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	01/95	5	0.92	2.20	0.01 ± 0.00	60.0	0.001 ± 0.001	0.000 ± 0.002	0.005 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	02/95	3	0.98	1.21	0.01 ± 0.00	60.0	0.002 ± 0.002	0.000 ± 0.001	0.004 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	03/95	5	0.98	1.51	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.001	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
ANTHRA	Payson	04/95	4	0.91	1.80	0.00 ± 0.00		0.002 ± 0.001	0.000 ± 0.000	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	05/95	4	0.95	2.12	0.00 ± 0.00		0.004 ± 0.003	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	06/95	4	0.92	3.47	0.00 ± 0.00		0.006 ± 0.004	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	07/95	4	0.97	1.23	0.00 ± 0.00		0.005 ± 0.003	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ANTHRA	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BENZE	Payson	12/94	1	0.75	1.95	0.45 ± 0.03	75.6	0.306 ± 0.122	0.034 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BENZE	Payson	01/95	5	0.92	2.20	4.93 ± 0.13	86.1	0.121 ± 0.072	3.340 ± 0.221	0.579 ± 0.260	0.000 ± 0.000	0.205 ± 0.030
BENZE	Payson	02/95	3	0.98	1.21	3.19 ± 0.09	98.0	0.235 ± 0.114	2.322 ± 0.145	0.463 ± 0.193	0.000 ± 0.000	0.106 ± 0.012
BENZE	Payson	03/95	5	0.98	1.51	2.84 ± 0.08	99.4	0.179 ± 0.089	2.272 ± 0.148	0.235 ± 0.091	0.000 ± 0.000	0.138 ± 0.019
BENZE	Payson	04/95	4	0.91	1.80	0.65 ± 0.03	106.0	0.262 ± 0.110	0.304 ± 0.020	0.122 ± 0.047	0.000 ± 0.000	0.001 ± 0.000
BENZE	Payson	05/95	4	0.95	2.12	2.16 ± 0.05	118.4	0.520 ± 0.251	1.991 ± 0.279	0.027 ± 0.010	0.000 ± 0.000	0.020 ± 0.003
BENZE	Payson	06/95	4	0.92	3.47	3.12 ± 0.08	129.6	0.777 ± 0.310	3.256 ± 0.437	0.002 ± 0.001	0.000 ± 0.000	0.010 ± 0.002
BENZE	Payson	07/95	4	0.97	1.23	2.14 ± 0.07	124.3	0.581 ± 0.245	1.908 ± 0.253	0.000 ± 0.000	0.000 ± 0.000	0.170 ± 0.022
BENZE	Payson	08/95	5	0.87	3.78	1.10 ± 0.04	118.6	0.273 ± 0.146	1.005 ± 0.173	0.000 ± 0.000	0.000 ± 0.000	0.027 ± 0.004
BIPHEN	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.013 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	01/95	5	0.92	2.20	0.02 ± 0.00	135.0	0.005 ± 0.004	0.000 ± 0.002	0.022 ± 0.010	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	02/95	3	0.98	1.21	0.02 ± 0.00	135.0	0.010 ± 0.007	0.000 ± 0.001	0.017 ± 0.007	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	03/95	5	0.98	1.51	0.02 ± 0.00	85.0	0.008 ± 0.006	0.000 ± 0.001	0.009 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	04/95	4	0.91	1.80	0.01 ± 0.00	160.0	0.011 ± 0.007	0.000 ± 0.000	0.005 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	05/95	4	0.95	2.12	0.01 ± 0.00	230.0	0.022 ± 0.016	0.000 ± 0.001	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	06/95	4	0.92	3.47	0.02 ± 0.00	165.0	0.033 ± 0.020	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	07/95	4	0.97	1.23	0.02 ± 0.00	125.0	0.025 ± 0.016	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BIPHEN	Payson	08/95	5	0.87	3.78	0.01 ± 0.00	120.0	0.012 ± 0.009	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	12/94	1	0.75	1.95	0.04 ± 0.02	172.5	0.066 ± 0.034	0.003 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	01/95	5	0.92	2.20	0.60 ± 0.04	82.3	0.026 ± 0.021	0.384 ± 0.027	0.084 ± 0.084	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	02/95	3	0.98	1.21	0.32 ± 0.02	120.0	0.050 ± 0.032	0.267 ± 0.018	0.067 ± 0.062	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	03/95	5	0.98	1.51	0.23 ± 0.02	144.8	0.038 ± 0.025	0.261 ± 0.018	0.034 ± 0.030	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	04/95	4	0.91	1.80	0.03 ± 0.02	363.3	0.056 ± 0.031	0.035 ± 0.003	0.018 ± 0.015	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	05/95	4	0.95	2.12	0.14 ± 0.02	207.1	0.112 ± 0.071	0.174 ± 0.029	0.004 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	06/95	4	0.92	3.47	0.19 ± 0.02	237.9	0.167 ± 0.088	0.285 ± 0.046	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	07/95	4	0.97	1.23	0.19 ± 0.02	153.7	0.125 ± 0.069	0.167 ± 0.027	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BUDI13	Payson	08/95	5	0.87	3.78	0.07 ± 0.02	210.0	0.059 ± 0.041	0.088 ± 0.018	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZI24M	Payson	12/94	1	0.75	1.95	0.93 ± 0.05	31.5	0.270 ± 0.087	0.023 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZI24M	Payson	01/95	5	0.92	2.20	2.83 ± 0.18	89.8	0.107 ± 0.051	1.910 ± 0.944	0.470 ± 0.468	0.000 ± 0.000	0.054 ± 0.038
BZI24M	Payson	02/95	3	0.98	1.21	2.05 ± 0.14	94.5	0.207 ± 0.082	1.328 ± 0.618	0.375 ± 0.346	0.000 ± 0.000	0.028 ± 0.016
BZI24M	Payson	03/95	5	0.98	1.51	1.83 ± 0.13	92.1	0.158 ± 0.064	1.300 ± 0.633	0.190 ± 0.164	0.000 ± 0.000	0.037 ± 0.024
BZI24M	Payson	04/95	4	0.91	1.80	0.54 ± 0.05	93.3	0.231 ± 0.079	0.174 ± 0.087	0.099 ± 0.083	0.000 ± 0.000	0.000 ± 0.001
BZI24M	Payson	05/95	4	0.95	2.12	1.93 ± 0.13	95.3	0.459 ± 0.180	1.354 ± 0.325	0.022 ± 0.017	0.000 ± 0.000	0.005 ± 0.004
BZI24M	Payson	06/95	4	0.92	3.47	2.83 ± 0.19	102.6	0.685 ± 0.221	2.214 ± 0.509	0.002 ± 0.002	0.000 ± 0.000	0.003 ± 0.002
BZI24M	Payson	07/95	4	0.97	1.23	1.76 ± 0.11	105.5	0.513 ± 0.175	1.297 ± 0.295	0.000 ± 0.000	0.000 ± 0.000	0.046 ± 0.028
BZI24M	Payson	08/95	5	0.87	3.78	4.85 ± 0.44	19.2	0.241 ± 0.105	0.683 ± 0.201	0.000 ± 0.000	0.000 ± 0.000	0.007 ± 0.005
BZI35M	Payson	12/94	1	0.75	1.95	0.00 ± 0.05		0.054 ± 0.008	0.007 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZI35M	Payson	01/95	5	0.92	2.20	0.80 ± 0.07	110.4	0.021 ± 0.005	0.843 ± 0.029	0.000 ± 0.000	0.000 ± 0.000	0.019 ± 0.010
BZI35M	Payson	02/95	3	0.98	1.21	0.54 ± 0.05	118.1	0.042 ± 0.008	0.586 ± 0.019	0.000 ± 0.000	0.000 ± 0.000	0.010 ± 0.004
BZI35M	Payson	03/95	5	0.98	1.51	0.48 ± 0.06	129.0	0.032 ± 0.006	0.574 ± 0.019	0.000 ± 0.000	0.000 ± 0.000	0.013 ± 0.006
BZI35M	Payson	04/95	4	0.91	1.80	0.23 ± 0.05	53.9	0.047 ± 0.008	0.077 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
BZI35M	Payson	05/95	4	0.95	2.12	0.54 ± 0.05	96.5	0.093 ± 0.018	0.426 ± 0.053	0.000 ± 0.000	0.000 ± 0.000	0.002 ± 0.001
BZI35M	Payson	06/95	4	0.92	3.47	0.73 ± 0.08	114.5	0.138 ± 0.021	0.697 ± 0.083	0.000 ± 0.000	0.000 ± 0.000	0.001 ± 0.001
BZI35M	Payson	07/95	4	0.97	1.23	0.45 ± 0.05	117.3	0.104 ± 0.017	0.408 ± 0.048	0.000 ± 0.000	0.000 ± 0.000	0.016 ± 0.008

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
BZ135M	Payson	08/95	5	0.87	3.78	0.36 ± 0.05	74.2	0.049 ± 0.010	0.215 ± 0.033	0.000 ± 0.000	0.000 ± 0.000	0.003 ± 0.001
CROTON	Payson	12/94	1	0.75	1.95	0.00 ± 0.03		0.000 ± 0.000	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	01/95	5	0.92	2.20	0.00 ± 0.02		0.000 ± 0.000	0.047 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	02/95	3	0.98	1.21	0.00 ± 0.03		0.000 ± 0.000	0.032 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	03/95	5	0.98	1.51	0.18 ± 0.11	17.8	0.000 ± 0.000	0.032 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	04/95	4	0.91	1.80	0.03 ± 0.03	13.3	0.000 ± 0.000	0.004 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	05/95	4	0.95	2.12	0.08 ± 0.05	41.3	0.000 ± 0.001	0.033 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	06/95	4	0.92	3.47	0.47 ± 0.21	11.5	0.000 ± 0.001	0.054 ± 0.009	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	07/95	4	0.97	1.23	0.31 ± 0.10	10.3	0.000 ± 0.001	0.032 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
CROTON	Payson	08/95	5	0.87	3.78	0.20 ± 0.08	8.5	0.000 ± 0.000	0.017 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.015 ± 0.010	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	01/95	5	0.92	2.20	0.02 ± 0.00	150.0	0.006 ± 0.005	0.000 ± 0.004	0.024 ± 0.012	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	02/95	3	0.98	1.21	0.02 ± 0.00	150.0	0.011 ± 0.009	0.000 ± 0.003	0.019 ± 0.009	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	03/95	5	0.98	1.51	0.02 ± 0.00	95.0	0.009 ± 0.007	0.000 ± 0.003	0.010 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	04/95	4	0.91	1.80	0.01 ± 0.00	180.0	0.013 ± 0.009	0.000 ± 0.001	0.005 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	05/95	4	0.95	2.12	0.01 ± 0.00	270.0	0.026 ± 0.020	0.000 ± 0.001	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	06/95	4	0.92	3.47	0.02 ± 0.00	190.0	0.038 ± 0.024	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	07/95	4	0.97	1.23	0.02 ± 0.00	140.0	0.028 ± 0.019	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
D14523	Payson	08/95	5	0.87	3.78	0.01 ± 0.00	130.0	0.013 ± 0.011	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	12/94	1	0.75	1.95	0.01 ± 0.00	450.0	0.045 ± 0.029	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	01/95	5	0.92	2.20	0.05 ± 0.01	188.0	0.018 ± 0.017	0.005 ± 0.013	0.071 ± 0.052	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	02/95	3	0.98	1.21	0.07 ± 0.01	134.3	0.034 ± 0.027	0.004 ± 0.008	0.056 ± 0.038	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	03/95	5	0.98	1.51	0.04 ± 0.01	147.5	0.026 ± 0.021	0.004 ± 0.009	0.029 ± 0.018	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	04/95	4	0.91	1.80	0.03 ± 0.01	176.7	0.038 ± 0.026	0.000 ± 0.001	0.015 ± 0.009	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	05/95	4	0.95	2.12	0.03 ± 0.01	266.7	0.077 ± 0.060	0.000 ± 0.001	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	06/95	4	0.92	3.47	0.08 ± 0.01	142.5	0.114 ± 0.074	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	07/95	4	0.97	1.23	0.05 ± 0.01	170.0	0.085 ± 0.059	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DM1367	Payson	08/95	5	0.87	3.78	0.02 ± 0.00	200.0	0.040 ± 0.035	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.006 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	01/95	5	0.92	2.20	0.01 ± 0.00	110.0	0.003 ± 0.003	0.000 ± 0.002	0.008 ± 0.006	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	02/95	3	0.98	1.21	0.01 ± 0.00	110.0	0.005 ± 0.005	0.000 ± 0.001	0.006 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	03/95	5	0.98	1.51	0.01 ± 0.00	70.0	0.004 ± 0.003	0.000 ± 0.001	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	04/95	4	0.91	1.80	0.01 ± 0.00	80.0	0.006 ± 0.004	0.000 ± 0.000	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	05/95	4	0.95	2.12	0.02 ± 0.00	55.0	0.011 ± 0.010	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	06/95	4	0.92	3.47	0.03 ± 0.01	56.7	0.017 ± 0.012	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	07/95	4	0.97	1.23	0.00 ± 0.00		0.013 ± 0.010	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN12	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.006 ± 0.006	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	01/95	5	0.92	2.20	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.002	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	02/95	3	0.98	1.21	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.001	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	03/95	5	0.98	1.51	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.001	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	04/95	4	0.91	1.80	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	05/95	4	0.95	2.12	0.00 ± 0.00		0.000 ± 0.001	0.001 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	06/95	4	0.92	3.47	0.00 ± 0.00		0.000 ± 0.000	0.002 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	07/95	4	0.97	1.23	0.00 ± 0.00		0.000 ± 0.001	0.001 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN18	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	12/94	1	0.75	1.95	0.01 ± 0.00	260.0	0.026 ± 0.017	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	01/95	5	0.92	2.20	0.02 ± 0.00	250.0	0.010 ± 0.010	0.007 ± 0.008	0.033 ± 0.028	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	02/95	3	0.98	1.21	0.04 ± 0.00	127.5	0.020 ± 0.016	0.005 ± 0.005	0.026 ± 0.020	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
N_HEX	Payson	02/95	3	0.98	1.21	1.62 ± 0.06	124.8	0.134 ± 0.095	1.724 ± 0.595	0.000 ± 0.000	0.000 ± 0.000	0.163 ± 0.028
N_HEX	Payson	03/95	5	0.98	1.51	1.81 ± 0.07	110.6	0.102 ± 0.074	1.687 ± 0.611	0.000 ± 0.000	0.000 ± 0.000	0.213 ± 0.043
N_HEX	Payson	04/95	4	0.91	1.80	0.30 ± 0.04	126.0	0.150 ± 0.091	0.226 ± 0.084	0.000 ± 0.000	0.000 ± 0.000	0.002 ± 0.001
N_HEX	Payson	05/95	4	0.95	2.12	1.51 ± 0.06	230.9	0.298 ± 0.208	3.158 ± 1.185	0.000 ± 0.000	0.000 ± 0.000	0.031 ± 0.007
N_HEX	Payson	06/95	4	0.92	3.47	2.94 ± 0.09	191.3	0.445 ± 0.257	5.164 ± 1.858	0.000 ± 0.000	0.000 ± 0.000	0.016 ± 0.004
N_HEX	Payson	07/95	4	0.97	1.23	1.98 ± 0.08	182.9	0.333 ± 0.203	3.026 ± 1.077	0.000 ± 0.000	0.000 ± 0.000	0.263 ± 0.050
N_HEX	Payson	08/95	5	0.87	3.78	0.74 ± 0.05	242.0	0.156 ± 0.121	1.594 ± 0.734	0.000 ± 0.000	0.000 ± 0.000	0.041 ± 0.008
N_NON	Payson	12/94	1	0.75	1.95	0.00 ± 0.05	ERR	0.078 ± 0.028	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
N_NON	Payson	01/95	5	0.92	2.20	0.26 ± 0.05	114.2	0.031 ± 0.017	0.254 ± 0.047	0.008 ± 0.033	0.000 ± 0.000	0.004 ± 0.008
N_NON	Payson	02/95	3	0.98	1.21	0.21 ± 0.05	116.7	0.060 ± 0.027	0.176 ± 0.030	0.007 ± 0.025	0.000 ± 0.000	0.002 ± 0.003
N_NON	Payson	03/95	5	0.98	1.51	0.16 ± 0.05	140.6	0.046 ± 0.021	0.173 ± 0.032	0.003 ± 0.012	0.000 ± 0.000	0.003 ± 0.005
N_NON	Payson	04/95	4	0.91	1.80	0.09 ± 0.05	102.2	0.067 ± 0.026	0.023 ± 0.004	0.002 ± 0.006	0.000 ± 0.000	0.000 ± 0.000
N_NON	Payson	05/95	4	0.95	2.12	0.33 ± 0.05	90.9	0.133 ± 0.059	0.166 ± 0.109	0.000 ± 0.001	0.000 ± 0.000	0.001 ± 0.001
N_NON	Payson	06/95	4	0.92	3.47	0.42 ± 0.05	111.9	0.199 ± 0.072	0.271 ± 0.171	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
N_NON	Payson	07/95	4	0.97	1.23	0.22 ± 0.05	141.4	0.149 ± 0.057	0.159 ± 0.099	0.000 ± 0.000	0.000 ± 0.000	0.003 ± 0.006
N_NON	Payson	08/95	5	0.87	3.78	0.29 ± 0.06	53.1	0.070 ± 0.034	0.084 ± 0.068	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
O_XYL	Payson	12/94	1	0.75	1.95	0.74 ± 0.04	25.3	0.163 ± 0.025	0.024 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
O_XYL	Payson	01/95	5	0.92	2.20	2.39 ± 0.10	96.6	0.064 ± 0.014	2.171 ± 0.144	0.017 ± 0.086	0.000 ± 0.000	0.057 ± 0.015
O_XYL	Payson	02/95	3	0.98	1.21	1.59 ± 0.08	105.5	0.125 ± 0.023	1.510 ± 0.094	0.013 ± 0.064	0.000 ± 0.000	0.029 ± 0.006
O_XYL	Payson	03/95	5	0.98	1.51	1.61 ± 0.07	100.4	0.095 ± 0.018	1.477 ± 0.097	0.007 ± 0.030	0.000 ± 0.000	0.038 ± 0.009
O_XYL	Payson	04/95	4	0.91	1.80	0.43 ± 0.04	79.3	0.139 ± 0.022	0.198 ± 0.013	0.004 ± 0.015	0.000 ± 0.000	0.000 ± 0.000
O_XYL	Payson	05/95	4	0.95	2.12	1.64 ± 0.08	102.6	0.276 ± 0.051	1.399 ± 0.172	0.001 ± 0.003	0.000 ± 0.000	0.006 ± 0.002
O_XYL	Payson	06/95	4	0.92	3.47	2.42 ± 0.11	111.7	0.413 ± 0.063	2.288 ± 0.270	0.000 ± 0.001	0.000 ± 0.000	0.003 ± 0.001
O_XYL	Payson	07/95	4	0.97	1.23	1.38 ± 0.07	122.9	0.309 ± 0.050	1.340 ± 0.156	0.000 ± 0.000	0.000 ± 0.000	0.047 ± 0.011
O_XYL	Payson	08/95	5	0.87	3.78	0.81 ± 0.05	105.9	0.145 ± 0.030	0.706 ± 0.107	0.000 ± 0.000	0.000 ± 0.000	0.007 ± 0.002
PA224M	Payson	12/94	1	0.75	1.95	0.05 ± 0.05	266.0	0.113 ± 0.035	0.020 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PA224M	Payson	01/95	5	0.92	2.20	2.77 ± 0.10	95.3	0.044 ± 0.021	2.203 ± 0.201	0.255 ± 0.120	0.000 ± 0.000	0.139 ± 0.213
PA224M	Payson	02/95	3	0.98	1.21	1.96 ± 0.08	96.7	0.087 ± 0.033	1.532 ± 0.132	0.204 ± 0.089	0.000 ± 0.000	0.072 ± 0.086
PA224M	Payson	03/95	5	0.98	1.51	1.88 ± 0.08	93.8	0.066 ± 0.026	1.499 ± 0.135	0.104 ± 0.042	0.000 ± 0.000	0.094 ± 0.134
PA224M	Payson	04/95	4	0.91	1.80	0.40 ± 0.05	88.0	0.097 ± 0.031	0.200 ± 0.019	0.054 ± 0.022	0.000 ± 0.000	0.001 ± 0.002
PA224M	Payson	05/95	4	0.95	2.12	1.75 ± 0.08	79.3	0.192 ± 0.072	1.170 ± 0.129	0.012 ± 0.004	0.000 ± 0.000	0.014 ± 0.022
PA224M	Payson	06/95	4	0.92	3.47	2.44 ± 0.19	90.5	0.287 ± 0.089	1.912 ± 0.202	0.001 ± 0.001	0.000 ± 0.000	0.007 ± 0.012
PA224M	Payson	07/95	4	0.97	1.23	1.66 ± 0.08	87.4	0.214 ± 0.071	1.121 ± 0.118	0.000 ± 0.000	0.000 ± 0.000	0.116 ± 0.157
PA224M	Payson	08/95	5	0.87	3.78	0.76 ± 0.08	93.3	0.101 ± 0.042	0.590 ± 0.080	0.000 ± 0.000	0.000 ± 0.000	0.018 ± 0.027
PAHSUM	Payson	12/94	1	0.75	1.95	0.12 ± 0.00	222.5	0.260 ± 0.053	0.007 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	01/95	5	0.92	2.20	1.53 ± 0.08	139.2	0.103 ± 0.031	0.395 ± 0.078	1.632 ± 0.581	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	02/95	3	0.98	1.21	1.73 ± 0.08	102.7	0.199 ± 0.050	0.275 ± 0.051	1.302 ± 0.429	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	03/95	5	0.98	1.51	0.86 ± 0.04	126.0	0.153 ± 0.038	0.270 ± 0.052	0.661 ± 0.204	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	04/95	4	0.91	1.80	0.68 ± 0.04	89.4	0.225 ± 0.047	0.037 ± 0.008	0.346 ± 0.104	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	05/95	4	0.95	2.12	0.45 ± 0.03	197.6	0.446 ± 0.109	0.367 ± 0.191	0.076 ± 0.021	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	06/95	4	0.92	3.47	1.03 ± 0.04	122.8	0.661 ± 0.134	0.599 ± 0.299	0.005 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	07/95	4	0.97	1.23	0.87 ± 0.04	97.5	0.496 ± 0.107	0.352 ± 0.173	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PAHSUM	Payson	08/95	5	0.87	3.78	0.27 ± 0.01	154.1	0.233 ± 0.063	0.183 ± 0.118	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	12/94	1	0.75	1.95	0.01 ± 0.00	170.0	0.017 ± 0.011	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	01/95	5	0.92	2.20	0.02 ± 0.00	175.0	0.007 ± 0.007	0.000 ± 0.002	0.028 ± 0.011	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	02/95	3	0.98	1.21	0.02 ± 0.00	175.0	0.013 ± 0.011	0.000 ± 0.001	0.022 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	03/95	5	0.98	1.51	0.01 ± 0.00	210.0	0.010 ± 0.008	0.000 ± 0.001	0.011 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	04/95	4	0.91	1.80	0.02 ± 0.00	100.0	0.014 ± 0.010	0.000 ± 0.000	0.006 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	05/95	4	0.95	2.12	0.01 ± 0.00	300.0	0.028 ± 0.023	0.000 ± 0.004	0.002 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
PHENAN	Payson	06/95	4	0.92	3.47	0.02 ± 0.00	210.0	0.042 ± 0.029	0.000 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	07/95	4	0.97	1.23	0.03 ± 0.00	103.3	0.031 ± 0.022	0.000 ± 0.004	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PHENAN	Payson	08/95	5	0.87	3.78	0.01 ± 0.00	150.0	0.015 ± 0.014	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	12/94	1	0.75	1.95	0.20 ± 0.06	0.5	0.000 ± 0.000	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	01/95	5	0.92	2.20	0.68 ± 0.12	10.4	0.000 ± 0.000	0.071 ± 0.012	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	02/95	3	0.98	1.21	0.62 ± 0.08	7.9	0.000 ± 0.000	0.049 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	03/95	5	0.98	1.51	0.53 ± 0.17	9.1	0.000 ± 0.000	0.048 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	04/95	4	0.91	1.80	0.81 ± 0.10	0.9	0.000 ± 0.000	0.007 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	05/95	4	0.95	2.12	0.98 ± 0.11	5.1	0.000 ± 0.001	0.050 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	06/95	4	0.92	3.47	1.00 ± 0.12	8.1	0.000 ± 0.001	0.081 ± 0.013	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	07/95	4	0.97	1.23	1.09 ± 0.13	4.4	0.000 ± 0.001	0.048 ± 0.008	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PROAL	Payson	08/95	5	0.87	3.78	0.71 ± 0.10	3.5	0.000 ± 0.000	0.025 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	01/95	5	0.92	2.20	0.01 ± 0.00	100.0	0.001 ± 0.001	0.000 ± 0.001	0.009 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	02/95	3	0.98	1.21	0.01 ± 0.00	90.0	0.002 ± 0.002	0.000 ± 0.001	0.007 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	03/95	5	0.98	1.51	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	04/95	4	0.91	1.80	0.01 ± 0.00	40.0	0.002 ± 0.001	0.000 ± 0.000	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	05/95	4	0.95	2.12	0.00 ± 0.00		0.004 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	06/95	4	0.92	3.47	0.00 ± 0.00		0.006 ± 0.004	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	07/95	4	0.97	1.23	0.00 ± 0.00		0.005 ± 0.003	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
PYRENE	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.002 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	01/95	5	0.92	2.20	0.01 ± 0.00	160.0	0.000 ± 0.000	0.000 ± 0.000	0.016 ± 0.009	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	02/95	3	0.98	1.21	0.01 ± 0.00	130.0	0.000 ± 0.001	0.000 ± 0.000	0.013 ± 0.007	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	03/95	5	0.98	1.51	0.01 ± 0.00	60.0	0.000 ± 0.000	0.000 ± 0.000	0.006 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	04/95	4	0.91	1.80	0.01 ± 0.00	30.0	0.000 ± 0.001	0.000 ± 0.000	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	05/95	4	0.95	2.12	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	06/95	4	0.92	3.47	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	07/95	4	0.97	1.23	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
RETENE	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
STYR	Payson	12/94	1	0.75	1.95	0.00 ± 0.04		0.258 ± 0.051	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
STYR	Payson	01/95	5	0.92	2.20	0.63 ± 0.07	95.4	0.102 ± 0.030	0.274 ± 0.076	0.223 ± 0.047	0.000 ± 0.000	0.002 ± 0.020
STYR	Payson	02/95	3	0.98	1.21	0.44 ± 0.04	129.1	0.198 ± 0.048	0.191 ± 0.050	0.178 ± 0.035	0.000 ± 0.000	0.001 ± 0.008
STYR	Payson	03/95	5	0.98	1.51	0.39 ± 0.04	110.3	0.151 ± 0.038	0.187 ± 0.051	0.091 ± 0.017	0.000 ± 0.000	0.001 ± 0.012
STYR	Payson	04/95	4	0.91	1.80	1.01 ± 0.08	29.0	0.221 ± 0.046	0.025 ± 0.007	0.047 ± 0.008	0.000 ± 0.000	0.000 ± 0.000
STYR	Payson	05/95	4	0.95	2.12	0.95 ± 0.08	65.5	0.438 ± 0.106	0.173 ± 0.090	0.011 ± 0.002	0.000 ± 0.000	0.000 ± 0.002
STYR	Payson	06/95	4	0.92	3.47	3.67 ± 0.40	25.6	0.655 ± 0.130	0.283 ± 0.142	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.001
STYR	Payson	07/95	4	0.97	1.23	0.60 ± 0.06	109.7	0.490 ± 0.103	0.166 ± 0.082	0.000 ± 0.000	0.000 ± 0.000	0.002 ± 0.015
STYR	Payson	08/95	5	0.87	3.78	1.92 ± 0.21	16.5	0.230 ± 0.061	0.087 ± 0.056	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.003
TOLUE	Payson	12/94	1	0.75	1.95	0.94 ± 0.04	75.6	0.587 ± 0.168	0.124 ± 0.038	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
TOLUE	Payson	01/95	5	0.92	2.20	10.72 ± 0.27	101.3	0.232 ± 0.099	10.216 ± 0.446	0.000 ± 0.000	0.000 ± 0.000	0.411 ± 0.170
TOLUE	Payson	02/95	3	0.98	1.21	7.47 ± 0.17	104.0	0.451 ± 0.158	7.103 ± 0.292	0.000 ± 0.000	0.000 ± 0.000	0.212 ± 0.069
TOLUE	Payson	03/95	5	0.98	1.51	7.32 ± 0.19	103.4	0.344 ± 0.123	6.951 ± 0.300	0.000 ± 0.000	0.000 ± 0.000	0.276 ± 0.107
TOLUE	Payson	04/95	4	0.91	1.80	1.61 ± 0.06	89.1	0.503 ± 0.151	0.930 ± 0.041	0.000 ± 0.000	0.000 ± 0.000	0.002 ± 0.001
TOLUE	Payson	05/95	4	0.95	2.12	6.72 ± 0.17	123.2	0.999 ± 0.346	7.239 ± 2.461	0.000 ± 0.000	0.000 ± 0.000	0.040 ± 0.018
TOLUE	Payson	06/95	4	0.92	3.47	10.00 ± 0.24	133.5	1.492 ± 0.427	11.837 ± 3.858	0.000 ± 0.000	0.000 ± 0.000	0.020 ± 0.010
TOLUE	Payson	07/95	4	0.97	1.23	6.35 ± 0.15	132.2	1.116 ± 0.338	6.936 ± 2.236	0.000 ± 0.000	0.000 ± 0.000	0.341 ± 0.125
TOLUE	Payson	08/95	5	0.87	3.78	3.59 ± 0.15	117.9	0.524 ± 0.202	3.654 ± 1.524	0.000 ± 0.000	0.000 ± 0.000	0.053 ± 0.022

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
DMN267	Payson	03/95	5	0.98	1.51	0.03 ± 0.00	113.3	0.015 ± 0.013	0.005 ± 0.005	0.014 ± 0.010	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	04/95	4	0.91	1.80	0.02 ± 0.00	155.0	0.023 ± 0.016	0.001 ± 0.001	0.007 ± 0.005	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	05/95	4	0.95	2.12	0.01 ± 0.00	480.0	0.045 ± 0.036	0.001 ± 0.006	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	06/95	4	0.92	3.47	0.03 ± 0.00	226.7	0.067 ± 0.045	0.001 ± 0.009	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	07/95	4	0.97	1.23	0.02 ± 0.00	250.0	0.050 ± 0.035	0.000 ± 0.005	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
DMN267	Payson	08/95	5	0.87	3.78	0.01 ± 0.00	240.0	0.024 ± 0.021	0.000 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ETBZ	Payson	12/94	1	0.75	1.95	0.00 ± 0.04		0.086 ± 0.018	0.018 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
ETBZ	Payson	01/95	5	0.92	2.20	1.86 ± 0.08	96.5	0.034 ± 0.011	1.703 ± 0.085	0.013 ± 0.055	0.000 ± 0.000	0.045 ± 0.012
ETBZ	Payson	02/95	3	0.98	1.21	1.24 ± 0.06	103.5	0.066 ± 0.016	1.184 ± 0.056	0.010 ± 0.041	0.000 ± 0.000	0.023 ± 0.005
ETBZ	Payson	03/95	5	0.98	1.51	1.23 ± 0.05	101.1	0.050 ± 0.013	1.159 ± 0.057	0.005 ± 0.019	0.000 ± 0.000	0.030 ± 0.008
ETBZ	Payson	04/95	4	0.91	1.80	0.26 ± 0.04	89.2	0.074 ± 0.016	0.155 ± 0.008	0.003 ± 0.010	0.000 ± 0.000	0.000 ± 0.000
ETBZ	Payson	05/95	4	0.95	2.12	1.11 ± 0.04	106.8	0.147 ± 0.036	1.034 ± 0.177	0.001 ± 0.002	0.000 ± 0.000	0.004 ± 0.001
ETBZ	Payson	06/95	4	0.92	3.47	1.84 ± 0.08	103.9	0.219 ± 0.045	1.691 ± 0.278	0.000 ± 0.001	0.000 ± 0.000	0.002 ± 0.001
ETBZ	Payson	07/95	4	0.97	1.23	1.04 ± 0.06	114.7	0.164 ± 0.036	0.991 ± 0.161	0.000 ± 0.000	0.000 ± 0.000	0.038 ± 0.009
ETBZ	Payson	08/95	5	0.87	3.78	0.54 ± 0.04	112.0	0.077 ± 0.021	0.522 ± 0.110	0.000 ± 0.000	0.000 ± 0.000	0.006 ± 0.002
FLUORA	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	01/95	5	0.92	2.20	0.01 ± 0.00	110.0	0.001 ± 0.001	0.001 ± 0.001	0.009 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	02/95	3	0.98	1.21	0.01 ± 0.00	90.0	0.001 ± 0.001	0.001 ± 0.000	0.007 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	03/95	5	0.98	1.51	0.00 ± 0.00		0.001 ± 0.001	0.000 ± 0.000	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	04/95	4	0.91	1.80	0.01 ± 0.00	40.0	0.002 ± 0.001	0.000 ± 0.000	0.002 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	05/95	4	0.95	2.12	0.00 ± 0.00		0.003 ± 0.002	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	06/95	4	0.92	3.47	0.01 ± 0.00	90.0	0.004 ± 0.003	0.005 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	07/95	4	0.97	1.23	0.00 ± 0.00		0.003 ± 0.002	0.003 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORA	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.001 ± 0.001	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	12/94	1	0.75	1.95	0.00 ± 0.00		0.009 ± 0.006	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	01/95	5	0.92	2.20	0.02 ± 0.00	100.0	0.003 ± 0.003	0.000 ± 0.002	0.017 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	02/95	3	0.98	1.21	0.01 ± 0.00	200.0	0.007 ± 0.005	0.000 ± 0.001	0.013 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	03/95	5	0.98	1.51	0.01 ± 0.00	120.0	0.005 ± 0.004	0.000 ± 0.001	0.007 ± 0.002	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	04/95	4	0.91	1.80	0.01 ± 0.00	120.0	0.008 ± 0.005	0.000 ± 0.000	0.004 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	05/95	4	0.95	2.12	0.01 ± 0.00	160.0	0.015 ± 0.011	0.000 ± 0.001	0.001 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	06/95	4	0.92	3.47	0.02 ± 0.00	110.0	0.022 ± 0.014	0.000 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	07/95	4	0.97	1.23	0.01 ± 0.00	170.0	0.017 ± 0.011	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FLUORE	Payson	08/95	5	0.87	3.78	0.00 ± 0.00		0.008 ± 0.007	0.000 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	12/94	1	0.75	1.95	0.10 ± 0.09	16.0	0.000 ± 0.000	0.016 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	01/95	5	0.92	2.20	1.20 ± 0.12	107.8	0.000 ± 0.000	1.293 ± 0.216	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	02/95	3	0.98	1.21	2.11 ± 0.18	42.6	0.000 ± 0.000	0.899 ± 0.141	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	03/95	5	0.98	1.51	0.93 ± 0.24	94.6	0.000 ± 0.000	0.880 ± 0.145	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	04/95	4	0.91	1.80	0.40 ± 0.09	29.5	0.000 ± 0.000	0.118 ± 0.020	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	05/95	4	0.95	2.12	0.53 ± 0.09	171.9	0.000 ± 0.001	0.911 ± 0.151	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	06/95	4	0.92	3.47	0.78 ± 0.10	190.9	0.000 ± 0.001	1.489 ± 0.236	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	07/95	4	0.97	1.23	0.80 ± 0.11	109.0	0.000 ± 0.001	0.872 ± 0.137	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
FORMAL	Payson	08/95	5	0.87	3.78	0.69 ± 0.10	66.7	0.000 ± 0.000	0.460 ± 0.093	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	12/94	1	0.75	1.95	0.22 ± 0.28	0.9	0.000 ± 0.000	0.002 ± 0.000	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	01/95	5	0.92	2.20	0.41 ± 0.24	48.8	0.000 ± 0.000	0.200 ± 0.033	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	02/95	3	0.98	1.21	1.45 ± 0.35	9.6	0.000 ± 0.000	0.139 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	03/95	5	0.98	1.51	1.94 ± 0.83	7.0	0.000 ± 0.000	0.136 ± 0.022	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	04/95	4	0.91	1.80	1.12 ± 0.32	1.6	0.000 ± 0.000	0.018 ± 0.003	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	05/95	4	0.95	2.12	1.15 ± 0.32	12.3	0.000 ± 0.001	0.141 ± 0.023	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	06/95	4	0.92	3.47	1.69 ± 0.36	13.6	0.000 ± 0.001	0.230 ± 0.036	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000

Source Contributions by Species - Arizona Hazardous Air Pollutant Risk Assessment Project

HAP Species	Site	Month	no	r_2	chi_2	Concentration ug/m3	mass %	Diesel Trucks ug/m3	Auto Exhaust ug/m3	Woodsmoke ug/m3	Liquid Gasoline ug/m3	Gasoline Vapor ug/m3
MEK	Payson	07/95	4	0.97	1.23	0.93 ± 0.31	14.5	0.000 ± 0.001	0.135 ± 0.021	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MEK	Payson	08/95	5	0.87	3.78	0.37 ± 0.25	19.2	0.000 ± 0.000	0.071 ± 0.014	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	12/94	1	0.75	1.95	0.02 ± 0.00	150.0	0.029 ± 0.017	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	01/95	5	0.92	2.20	0.21 ± 0.02	120.5	0.011 ± 0.010	0.054 ± 0.018	0.188 ± 0.118	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	02/95	3	0.98	1.21	0.18 ± 0.02	116.1	0.022 ± 0.016	0.037 ± 0.012	0.150 ± 0.087	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	03/95	5	0.98	1.51	0.10 ± 0.01	129.0	0.017 ± 0.012	0.036 ± 0.012	0.076 ± 0.041	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	04/95	4	0.91	1.80	0.08 ± 0.01	87.5	0.025 ± 0.015	0.005 ± 0.002	0.040 ± 0.021	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	05/95	4	0.95	2.12	0.06 ± 0.01	173.3	0.049 ± 0.034	0.046 ± 0.040	0.009 ± 0.004	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	06/95	4	0.92	3.47	0.11 ± 0.01	136.4	0.073 ± 0.042	0.076 ± 0.062	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	07/95	4	0.97	1.23	0.10 ± 0.01	99.0	0.054 ± 0.034	0.045 ± 0.036	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH1	Payson	08/95	5	0.87	3.78	0.03 ± 0.00	163.3	0.026 ± 0.020	0.023 ± 0.024	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	12/94	1	0.75	1.95	0.03 ± 0.00	173.3	0.049 ± 0.026	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	01/95	5	0.92	2.20	0.41 ± 0.04	132.0	0.019 ± 0.015	0.119 ± 0.042	0.403 ± 0.268	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	02/95	3	0.98	1.21	0.43 ± 0.04	102.8	0.038 ± 0.025	0.082 ± 0.028	0.322 ± 0.198	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	03/95	5	0.98	1.51	0.23 ± 0.02	119.1	0.029 ± 0.019	0.081 ± 0.028	0.164 ± 0.094	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	04/95	4	0.91	1.80	0.16 ± 0.01	86.3	0.042 ± 0.023	0.011 ± 0.004	0.085 ± 0.048	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	05/95	4	0.95	2.12	0.12 ± 0.01	209.2	0.083 ± 0.054	0.149 ± 0.124	0.019 ± 0.010	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	06/95	4	0.92	3.47	0.25 ± 0.02	147.2	0.124 ± 0.067	0.243 ± 0.195	0.001 ± 0.001	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	07/95	4	0.97	1.23	0.22 ± 0.02	107.3	0.093 ± 0.053	0.143 ± 0.113	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MNAPH2	Payson	08/95	5	0.87	3.78	0.08 ± 0.01	148.8	0.044 ± 0.031	0.075 ± 0.077	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MP_XYL	Payson	12/94	1	0.75	1.95	0.65 ± 0.04	60.2	0.322 ± 0.044	0.069 ± 0.013	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
MP_XYL	Payson	01/95	5	0.92	2.20	6.78 ± 0.18	95.9	0.127 ± 0.026	6.186 ± 0.257	0.036 ± 0.150	0.000 ± 0.000	0.154 ± 0.035
MP_XYL	Payson	02/95	3	0.98	1.21	4.65 ± 0.12	100.2	0.247 ± 0.041	4.301 ± 0.169	0.029 ± 0.111	0.000 ± 0.000	0.080 ± 0.014
MP_XYL	Payson	03/95	5	0.98	1.51	4.31 ± 0.11	104.8	0.189 ± 0.032	4.209 ± 0.173	0.015 ± 0.053	0.000 ± 0.000	0.104 ± 0.022
MP_XYL	Payson	04/95	4	0.91	1.80	1.00 ± 0.04	84.8	0.276 ± 0.040	0.563 ± 0.024	0.008 ± 0.027	0.000 ± 0.000	0.001 ± 0.001
MP_XYL	Payson	05/95	4	0.95	2.12	3.93 ± 0.11	117.2	0.547 ± 0.091	4.043 ± 0.813	0.002 ± 0.005	0.000 ± 0.000	0.015 ± 0.004
MP_XYL	Payson	06/95	4	0.92	3.47	6.10 ± 0.14	121.9	0.817 ± 0.112	6.611 ± 1.275	0.000 ± 0.001	0.000 ± 0.000	0.008 ± 0.002
MP_XYL	Payson	07/95	4	0.97	1.23	3.72 ± 0.10	124.0	0.612 ± 0.089	3.874 ± 0.739	0.000 ± 0.000	0.000 ± 0.000	0.128 ± 0.026
MP_XYL	Payson	08/95	5	0.87	3.78	2.34 ± 0.11	100.3	0.287 ± 0.053	2.041 ± 0.504	0.000 ± 0.000	0.000 ± 0.000	0.020 ± 0.004
NAPHTH	Payson	12/94	1	0.75	1.95	0.04 ± 0.00	105.0	0.039 ± 0.018	0.003 ± 0.002	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	01/95	5	0.92	2.20	0.68 ± 0.06	144.6	0.015 ± 0.011	0.202 ± 0.061	0.766 ± 0.498	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	02/95	3	0.98	1.21	0.86 ± 0.07	91.2	0.030 ± 0.017	0.141 ± 0.040	0.613 ± 0.368	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	03/95	5	0.98	1.51	0.37 ± 0.03	127.6	0.023 ± 0.013	0.138 ± 0.041	0.311 ± 0.175	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	04/95	4	0.91	1.80	0.28 ± 0.03	76.4	0.033 ± 0.016	0.019 ± 0.006	0.162 ± 0.089	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	05/95	4	0.95	2.12	0.16 ± 0.03	167.5	0.067 ± 0.037	0.165 ± 0.139	0.036 ± 0.018	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	06/95	4	0.92	3.47	0.42 ± 0.03	88.3	0.099 ± 0.045	0.269 ± 0.217	0.003 ± 0.003	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	07/95	4	0.97	1.23	0.36 ± 0.03	64.4	0.074 ± 0.036	0.158 ± 0.126	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NAPHTH	Payson	08/95	5	0.87	3.78	0.09 ± 0.01	131.1	0.035 ± 0.022	0.083 ± 0.086	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NMOC	Payson	12/94	1	0.75	1.95	30.71 ± 1.26	57.6	14.352 ± 3.058	1.255 ± 1.667	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
NMOC	Payson	01/95	5	0.92	2.20	209.38 ± 7.21	94.3	5.660 ± 6.575	103.923 ± 4.501	16.387 ± 3.869	0.000 ± 0.000	18.830 ± 2.868
NMOC	Payson	02/95	3	0.98	1.21	190.43 ± 5.96	97.1	11.010 ± 6.157	72.257 ± 3.480	13.102 ± 2.913	0.000 ± 0.000	9.733 ± 1.655
NMOC	Payson	03/95	5	0.98	1.51	126.99 ± 4.61	97.7	8.408 ± 5.215	70.714 ± 3.296	6.651 ± 1.665	0.000 ± 0.000	12.678 ± 1.823
NMOC	Payson	04/95	4	0.91	1.80	42.50 ± 1.47	66.6	12.282 ± 4.374	9.459 ± 2.037	3.457 ± 0.992	0.000 ± 0.000	0.091 ± 0.270
NMOC	Payson	05/95	4	0.95	2.12	118.54 ± 3.81	101.1	24.395 ± 9.406	73.212 ± 5.874	0.771 ± 1.153	0.000 ± 0.000	1.828 ± 1.202
NMOC	Payson	06/95	4	0.92	3.47	189.57 ± 6.44	93.9	36.443 ± 12.219	119.719 ± 8.706	0.055 ± 0.854	0.000 ± 0.000	0.927 ± 2.676
NMOC	Payson	07/95	4	0.97	1.23	161.86 ± 5.40	98.6	27.266 ± 9.205	70.149 ± 6.846	0.000 ± 0.000	0.000 ± 0.000	15.625 ± 2.970
NMOC	Payson	08/95	5	0.87	3.78	126.09 ± 4.90	51.5	12.797 ± 4.236	36.960 ± 3.474	0.000 ± 0.000	0.000 ± 0.000	2.428 ± 1.418
N_HEX	Payson	12/94	1	0.75	1.95	0.25 ± 0.04	91.6	0.175 ± 0.101	0.054 ± 0.018	0.000 ± 0.000	0.000 ± 0.000	0.000 ± 0.000
N_HEX	Payson	01/95	5	0.92	2.20	2.45 ± 0.08	116.9	0.069 ± 0.060	2.479 ± 0.910	0.000 ± 0.000	0.000 ± 0.000	0.317 ± 0.068

APPENDIX I

ESTIMATION OF FUTURE HAP EMISSIONS

TABLE OF CONTENTS

		Page
	EMISSIONS PROJECTION SUMMARY	S-1
1.0	METHODOLOGY	1-1
1.1	Projection Methodology for Maricopa County Sources	1-5
1.2	Projection Methodology for Casa Grande, Tucson, and Payson Sources	1-5
2.0	PROJECTION FACTOR DEVELOPMENT	2-1
2.1	On-Road Mobile Source Growth and Control Factors	2-6
2.2	Off-Road Mobile Source Growth and Control Factors	2-7
2.3	Halogenated Solvent Degreasing Growth and Control Factors	2-8
2.4	Perchloroethylene Dry Cleaning Growth and Control Factors	2-9
2.5	Solvent Usage Growth and Control Factors	2-10
2.6	Residential Wood Combustion Growth and Control Factors	2-11
2.7	Agricultural Pesticide Growth and Control Factors	2-12
2.8	Swimming Pool Chloroform Growth and Control Factors	2-13
3.0	REFERENCES	3-1

ATTACHMENT I-1: Listing of Growth and Control Factors

LIST OF TABLES

		Page
S-1	Baseline and Projected Emissions for the Casa Grande Modeling Domain	S-2
S-2	Baseline and Projected Emissions for the Payson Modeling Domain	S-3
S-3	Baseline and Projected Emissions for the Phoenix Modeling Domain	S-4
S-4	Baseline and Projected Emissions for the Tucson Modeling Domain	S-5
1-1	National Emission Standards Potentially Applicable to Sources in the Four Study Regions	1-3
2-1	Summary of Arizona HAP Growth Factor Development for Key Sources	2-2
2-2	Summary of Arizona HAP Control Factor Development for Key Success	2-4
2-3	On-Road Mobile Source Growth and Control Factors	2-7
2-4	Off-Road Mobile Source Growth and Control Factors	2-8
2-5	Halogenated Solvent Degreasing Growth and Control Factors	2-9
2-6	Perchloroethylene Dry Clean Growth and Control Factors	2-10
2-7	Industrial and Consumer Solvent Usage Growth and Control Factors	2-11
2-8	Residential Wood Combustion Growth and Control Factors	2-12
2-9	Agricultural Pesticide Application Growth and Control Factors	2-13
2-10	Swimming Pool Chloroform Growth and Control Factors	2-13

EMISSIONS PROJECTION SUMMARY

The section presents the emission results from the projection of the baseline emissions to the year 2005. Summaries of the annual emission changes for each of the four regions are presented in Tables S-1 (Casa Grande), S-2 (Payson), S-3 (Phoenix), and S-4 (Tucson). These tables present the baseline emissions, projected emissions, change in emissions, and percent change in emissions for each of the Compounds of Interest (COI). The methods used to project the baseline emissions are discussed in Section 1.0. In Section 2.0, the development of the projection factors is discussed, with particular focus on key source categories such as on-road and off-road mobile sources, solvent usage, agricultural pesticides, and residential wood combustion.

From the results of this analysis, several key observations can be made:

- Categories contributing significant emissions of the highest risk pollutants in the baseline inventory included on-road motor vehicles, off-road mobile sources, and solvent usage.

- Projected control measures that will result in Hazardous Air Pollutant (HAP) emission reductions consist of both federally mandated controls and local controls. At the federal level, National Emission Standards for Hazardous Air Pollutants (NESHAPs) are being developed. Locally, control measures developed as part of the State Implementation Plan for ozone control in Phoenix will also reduce HAP emissions. There are no local control measures affecting HAP emissions for Payson, Tucson, or Casa Grande.

- Significant growth is projected in emissions activity for all four regions due to population growth and economic expansion. However, as can be seen in Tables S-1 through S-4, the change in emissions varies by compound depending upon the interaction of growth in activity versus the impact of projected control measures.

- Compounds associated with combustion sources, such as 1,3-butadiene, acetaldehyde, formaldehyde, and benzene, show significant reductions in all four regions. Reductions in emissions from on-road mobile sources are a large contribution to this effect. These reductions are due to turnover of the vehicle fleet to lower emitting vehicles, and implementation of more stringent emission standards for new vehicles. The impact of these effects outweighs increases in emissions due to growth in vehicle travel in each region.

Table S-1

Baseline and Projected Emissions for the Casa Grande Modeling Domain (tons per year)

CAS No.	HAP	Baseline	Projected	Percent Change
106-46-7	1,4-Dichlorobenzene	8	11	33
106-99-0	1,3-Butadiene	12	9	-27
107-02-8	Acrolein	2	1	-19
108-88-3	Toluene	530	545	3
1582-09-8	Trifluralin	2	3	20
18540-29-9	Chromium (VI)	<1	<1	-87
25551-13-7	Trimethylbenzene	3	3	23
2921-88-2	Chlorpyrifos	14	16	20
298-00-0	Methyl Parathion	12	15	20
298-04-4	Disulfoton	<1	<1	20
50-00-0	Formaldehyde	63	57	-9
50-32-8	Benzo(a)pyrene	<1	<1	39
67-66-3	Chloroform	<1	<1	19
71-43-2	Benzene	99	77	-22
74-87-3	Chloromethane	<1	<1	19
7439-92-1	Lead	3	1	-67
7439-96-5	Manganese	5	6	33
7440-02-0	Nickel	<1	<1	35
7440-38-2	Arsenic	<1	<1	30
7440-39-3	Barium	3	4	34
7440-43-9	Cadmium	<1	<1	27
75-07-0	Acetaldehyde	16	13	-19
75-09-2	Dichloromethane	23	25	9
7664-41-7	Ammonia	133	176	33
79-01-6	Trichloroethylene	14	8	-46
82-68-8	Pentachloronitrobenzene	<1	<1	20

Table S-2

Baseline and Projected Emissions for the Payson Modeling Domain (tons per year)

CAS No.	HAP	Baseline	Projected	Percent Change
106-46-7	1,4-Dichlorobenzene	<1	<1	10
106-99-0	1,3-Butadiene	5	3	-36
107-02-8	Acrolein	<1	<1	-28
108-88-3	Toluene	58	44	-25
110-54-3	Hexane	21	16	-24
127-18-4	Perchloroethylene	1	<1	-41
18540-29-9	Chromium (VI)	<1	0	-100
218-01-9	Chrysene	<1	<1	-44
25551-13-7	Trimethylbenzene	<1	<1	18
50-00-0	Formaldehyde	24	16	-32
50-32-8	Benzo(a)pyrene	<1	<1	-43
67-66-3	Chloroform	<1	<1	10
71-43-2	Benzene	33	21	-37
74-87-3	Chloromethane	<1	<1	10
7439-92-1	Lead	<1	<1	-69
7439-96-5	Manganese	1	2	39
7440-02-0	Nickel	<1	<1	40
7440-38-2	Arsenic	<1	<1	33
7440-39-3	Barium	<1	1	38
7440-43-9	Cadmium	<1	<1	21
75-07-0	Acetaldehyde	4	3	-31
75-09-2	Dichloromethane	2	3	9
75-21-8	Ethylene Oxide	<1	<1	15
79-01-6	Trichloroethylene	1	<1	-47
91-20-3	Naphthalene	22	12	-44
POM	Polycyclic Organic Matter	47	27	-43

Table S-3

Baseline and Projected Emissions for the Phoenix Modeling Domain (tons per year)

CAS No.	HAP	Baseline	Projected	Percent Change
106-46-7	1,4-Dichlorobenzene	164	209	27
106-93-4	Dibromoethane	1	1	0
106-99-0	1,3-Butadiene	293	176	-40
107-02-8	Acrolein	40	33	-17
107-13-1	Acrylonitrile	3	4	20
127-18-4	Perchloroethylene	750	556	-26
18540-29-9	Chromium (VI)	<1	<1	-80
25551-13-7	Trimethylbenzene	215	250	16
50-00-0	Formaldehyde	1111	960	-14
50-32-8	Benzo(a)pyrene	<1	<1	-13
67-66-3	Chloroform	161	212	32
71-43-2	Benzene	1984	1215	-39
74-87-3	Chloromethane	6	8	32
7439-92-1	Lead	65	45	-31
7439-96-5	Manganese	151	207	37
7439-97-6	Mercury	3	4	46
7440-02-0	Nickel	11	15	37
7440-38-2	Arsenic	1	2	32
7440-39-3	Barium	90	123	36
7440-43-9	Cadmium	3	4	30
75-01-4	Vinyl Chloride	2	3	36
75-07-0	Acetaldehyde	298	222	-25
75-09-2	Dichloromethane	1131	1117	-1
75-21-8	Ethylene Oxide	13	16	25
79-01-6	Trichloroethylene	704	398	-43

Table S-4

Baseline and Projected Emissions for the Tucson Modeling Domain (tons per year)

CAS No.	HAP	Baseline	Projected	Percent Change
106-46-7	1,4-Dichlorobenzene	39	48	24
106-99-0	1,3-Butadiene	88	66	-26
107-02-8	Acrolein	11	11	-1
108-88-3	Toluene	2670	2707	1
108-90-7	Chlorobenzene	122	169	38
110-54-3	Hexane	692	651	-6
127-18-4	Perchloroethylene	180	123	-32
18540-29-9	Chromium (VI)	<1	0	-100
25551-13-7	Trimethylbenzene	32	40	23
50-00-0	Formaldehyde	327	317	-3
50-32-8	Benzo(a)pyrene	<1	<1	-37
67-66-3	Chloroform	28	34	21
71-43-2	Benzene	629	506	-19
74-87-3	Chloromethane	2	2	21
7439-92-1	Lead	20	11	-46
7439-96-5	Manganese	41	55	34
7440-02-0	Nickel	4	5	33
7440-38-2	Arsenic	<1	<1	28
7440-39-3	Barium	26	34	31
7440-43-9	Cadmium	<1	1	27
75-07-0	Acetaldehyde	93	86	-8
75-09-2	Dichloromethane	238	268	12
75-21-8	Ethylene Oxide	2	3	30
79-01-6	Trichloroethylene	125	70	-44

- Implementation of federal emission limits for certain classes of off-road mobile sources also helps to reduce emissions of combustion-related compounds. Federal limits on emissions from small gasoline engines less than 25 horsepower and larger compression-ignition engines over 50 horsepower contribute to reductions in emissions of compounds such as 1,3-butadiene, acetaldehyde, formaldehyde, and benzene. As with the on-road mobile sources, reductions from these controls are predicted to outweigh increased emissions arising from growth in the equipment population.

- Many of the metals emissions come from fugitive sources such as entrained road dust and windblown dust. Compounds in this category, including chromium, arsenic, cadmium, and nickel, are expected to increase due to increases in overall vehicle travel.

- Lead emissions, resulting primarily from on-road and off-road motor vehicles, as well as entrained road dust, are reduced significantly due to the pending elimination of leaded gasoline sales.

- For the four counties in this emissions inventory, halogenated solvent degreasing is the largest source of trichloroethylene emissions. It is also a significant source of perchloroethylene and methylene chloride (dichloromethane) emissions. Like most emission sources in Arizona, halogenated solvent degreasing will grow due to the effects of population growth and economic expansion. Control of halogenated solvent degreasing is being driven by a NESHAP promulgated by the U.S. Environmental Protection Agency (EPA).

- Implementation of NESHAP dry cleaning and degreasing rules for perchloroethylene will result in reduction of emissions for this compound of over 30%.

- Emissions from consumer and industrial solvents will increase due to expansion of population and industrial operations. 1,4-dichlorobenzene and dichloromethane are significant VOC compounds that result from both industrial and residential solvent usage. In addition, industrial solvent usage contributes trichloroethylene and chlorobenzene to the emissions inventory.

- Emissions from agricultural pesticides decreased in Maricopa County due to projected decreases in agricultural acreage. Pesticide emissions in the three other counties are projected to increase due to increases in agricultural activity.

- Emissions of polycyclic organic matter (POM) from residential wood combustion in Payson are predicted to decrease by over 35%. This decrease will be achieved through the use of lower emitting wood stoves in new residences and the replacement of existing stoves with lower emitting units. This effect is expected to outweigh increases in emissions due to population growth.

1.0 METHODOLOGY

Given the goal of the research program to provide options and recommendations for future programs, it is necessary to consider the impact of existing federally-mandated controls on future emissions. In particular, the 1990 Clean Air Act Amendments mandate reduction of hazardous air pollutant (HAP) emissions from various industries and wide-spread area sources over the next several years. The goal of this inventory is to estimate, or project, the impact of these controls on future emissions, using the existing inventory as a baseline.

For the projection year, we assumed 2005 for the following reasons:

- 2005 coincides with existing projected Maricopa County SIP inventory.
- 2005 is far enough into the future for most planned rules to have been implemented. For example, all National Emission Standard for Hazardous Air Pollutants (NESHAPs) must be promulgated by 2000 and should be implemented by 2005.
- The projection of emissions beyond 10 years into the future is highly speculative.

Projection of the baseline inventory to a future year involves the estimation of two separate and independent factors for each emission source category. Without the consideration for any additional controls, emissions for a source category may change due to changes in the amount of activity producing the emissions. For example, growth in the vehicle population and the vehicle miles travelled (VMT) will impact on-road motor vehicle emissions. Independent of the growth in emissions, control measures can be mandated to reduce emissions from individual sources. The inventory projection calculation combines growth and control parameters to project emissions from the base year to the future year, as follows:

$$Emissions_{projected} = Emissions_{base} * (Growth\ factor) * (Control\ factor)$$

To develop projection factors for each compound, projection factors were developed for volatile organic compounds (VOC) and particulate matter (PM). These factors were then applied to individual compounds based on whether the compound was VOC-based or particulate-based. Based on the risk assessment results, most of the highest risk pollutants are VOCs. In this method, all VOC-based compounds for a given source category would be assigned a single projection factor, and all PM-based compounds would be assigned a single projection factor. Except for certain sources, speciation of source category emissions into individual compounds was unchanged from the baseline methodology. One exception to this was lead emissions from gasoline engines and entrained road dust. In this case, a specific set of control factors were developed to reflect the elimination of leaded gasoline.

The growth factors used in this analysis reflect the anticipated growth in population and expected economic expansion. All emission estimates were projected forward to 2005 from the baseline emission estimates. The baseline date varies by emission source category, depending on the origin of the emission estimates (e.g., 1990 emission estimates from the Maricopa SIP or 1993 emissions calculated specifically for this study).

For the development of the control factors, we examined the impact of federal, state, and local control measures. These included federal NESHAP standards being developed for many sources, and state and local measures included in the State Implementation Plan (SIP) such as vehicle inspection and maintenance programs. A list of national emission standards potentially applicable to the four study regions is included in Table 1-1.

Because of the large number of HAP sources and the large number of control measures, we prioritized the control factor development in a two step process. In the first step, we identified those compounds in the baseline inventory producing the highest total risk. In the second step, we determined the source categories emitting significant amounts of each high risk compound. We then concentrated effort on developing control factors for those sources. This process highlighted the following source categories:

Table 1-1

National Emission Standards Potentially Applicable to Sources in the Four Study Regions[a]

Source Category	Promulgation Date
Chrome Plating	1995
Halogenated Solvent Cleaners	1994
Aerospace Industries	1995
Gasoline Distribution (Stage I)	1994
Industrial Process Cooling Towers	1994
Magnetic Tapes (surface coating)	1994
Printing and Publishing (surface coating)	1996
Off-site Waste (TSDF) Operations	1995
Wood Furniture (surface coating)	1995
POTW Emissions	1997
Secondary Aluminum Production	1997
Wood Treatment	1996
Boat Manufacturing	2000
Dry Cleaning	2000
Industrial Boilers	2000
Institutional/Commercial Boilers	2000
Metal Furniture (surface coating)	2000
Misc. Metal Parts and Products (surface coating)	2000
Municipal Landfills	2000
Paint Strippers	2000
Paper and Other Webs (surface coating)	2000
Plastic Parts and Products (surface coating)	2000
Rocket Engine Test Firing	2000
Site Remediation	2000
Stationary Internal Combustion Engines	2000
Stationary Turbines	2000

[a] NESHAPs for many more source categories will be developed than are shown here. This lists the NESHAPs most likely to be applicable to the four Arizona study regions.

- On-road motor vehicles;
- Off-road mobile sources, including lawn & garden equipment, aircraft, recreational equipment, industrial equipment, construction equipment, farming, and airport service; and
- Solvents, including degreasing, industrial and domestic solvent usage, architectural coatings, and dry cleaning.

Note that no major point sources are included in this list of key sources because of their relatively small contribution to the total baseline risk.

While it was necessary to prioritize the development of the control factors, we estimated growth factors for all point and area sources. For each category, we identified the underlying activity which would control the growth of the emissions. For example, for on-road motor vehicles, emissions are controlled by the amount of vehicle miles travelled in a region. In many cases, growth was determined based on projected economic changes. Whenever possible, data developed by state or local sources were used for the growth factor development.

As can be seen in Table 1-1, a number of federal emissions standards have not been promulgated. Many of these standards will impact specific sources within the four study regions, but will have only a small impact on the overall risk. Three standards specifically accounted for in the projection inventory are cooling towers, halogenated solvent cleaners, and dry cleaners. Taken together, all the remaining standards will tend to reduce the projected emissions below the levels accounted for in this inventory.

In addition, other effects not accounted for in this inventory will tend to lower future emissions in the future. These include impacts from criteria pollutant reductions and ozone depleting substances (ODS) not directly accounted for in the models or data used to develop the control factors. In some of these cases, HAP emissions will be reduced as a secondary impact. An example of this is the re-design of consumer solvent products to reduce ODS emissions. Another example is the introduction of alternative fuel vehicles into state and federal vehicle fleets in response to requirements of the 1992 National Energy Policy Act.

1.1 Projection Methodology for Maricopa County Sources

Projection factors for Maricopa County area sources were developed using data obtained from the Maricopa Association of Governments, (MAG) (Fieber and Duvall, 1994). This document includes growth and control parameters combined one single projection factor. Taking advantage of these existing data helped to retain consistency with the existing 2005 projection SIP inventory.

For some off-road mobile categories, controls for new engines have recently been promulgated that are not accounted for by the MAG data. These include national controls on new internal combustion engines greater than 50 horsepower, and small utility engines less than 25 horsepower. We used guidance provided by U.S. EPA (Lorang, 1994) for the projection of emissions from these sources.

Reduction of on-road motor vehicle emissions due to fleet turnover was estimated through the use of the U.S. EPA's MOBILE5a and PART5 emission factor models (U.S. EPA, 1993; U.S. EPA, 1995). For control of degreasing and dry cleaning emissions, we reconciled the existing projection factors for Maricopa County (Fieber and Duvall, 1994) with the NESHAP regulations. For point sources, growth and control factors developed for Maricopa County sources were applied (Fieber and Duvall, 1994).

1.2 Projection Methodology for Casa Grande, Tucson, and Payson Sources

Depending upon the source type, growth factors were developed from one of three sources: VMT projections (Arthur, 1995; Catchpole, 1995), population projections (Arizona Department of Economic Security, 1991; Arizona Department of Economic Security, 1993), or economic projections available from the Bureau of Economic Analysis (BEA) (BEA, 1994a; BEA, 1994b):

- VMT projections were used for on-road motor vehicles refueling emissions, and other petroleum product storage and transport.
- Population projections were used for processes such as residential fuel combustion, consumer solvent use, architectural coatings, and dry cleaning.

- Economic projection data were applied by SIC code for point sources and area sources including commercial/industrial fuel combustion, industrial processes, surface coating, degreasing, and commercial solvent use.

- Agricultural emissions, primarily from pesticides, were projected using BEA economic projection data.

For Casa Grande, Tucson, and Payson, control factors were developed and applied for the highest priority sources, based on the risk assessment results.

- Reduction of on-road mobile source emissions due to fleet turnover was estimated through the use of the U.S. EPA's MOBILE5a and PART5 emission factor models.

- Off-road mobile source emission reductions were estimated from the U.S. EPA guidance applied to Maricopa County (Lorang, 1994).

- Control factors for degreasing and dry cleaning emissions were based on the factors developed for Maricopa County reflecting the impact of federal NESHAP regulations (Federal Register, 1994a; Federal Register, 1994b).

2.0 PROJECTION FACTOR DEVELOPMENT

This section describes the development of projection factors for the four regions in the Arizona HAP emissions inventory. Growth factors were developed for all point and area source categories in the emissions inventory. Control factors for many point and area source categories were also developed for sources contributing the majority of the health risk. This section focuses specifically on the development of projection factors for the following key source categories:

- On-road mobile sources;
- Off-road mobile sources;
- Halogenated solvent degreasing;
- Perchloroethylene dry cleaning;
- Solvent usage (industrial and domestic);
- Residential wood combustion;
- Agricultural pesticides; and
- Chloroform from swimming pools.

Table 2-1 summarizes the data sources used to develop the growth factors while Table 2-2 summarizes the data sources that were used in the development of the control factors for these key source categories.

The following sections provide a detailed description of the development of the projection factors for each of the key source categories. Each description includes a listing of major compounds from each source category and an explanation of the driving influences that will affect future emissions (e.g., increasing population, legislative mandates, etc.). A summary table of growth and control factors used in the projected emissions analysis is included.

Table 2-1

Summary of Arizona HAP Growth Factor Development for Key Sources

Category	Sub-Category	Growth Factor
On-Road Mobile	Maricopa County	Growth factors based upon county-wide vehicle miles travelled (VMT) projections from Maricopa Association of Governments Transportation and Planning Office (MAGTPO).
	Pima, Pinal, and Gila Counties	Growth factors based upon state-wide VMT projections from Arizona Department of Transportation (ADOT).
Off-Road Mobile	Maricopa County	Growth factors based on information from *1992, 1996 and 2005 Baseline Projection Growth Factors for Ozone Emissions*, Maricopa County Environmental Management and Transportation Agency, November 1993.
	Pima, Pinal, and Gila Counties	Growth factors based on population statistics and Bureau of Economic Analysis (BEA) projections data. Recreational Vehicles - Population statistics Construction Equipment - BEA Construction projections Industrial Equipment - BEA Average Manufacturing projections Lawn & Garden Equipment - Population statistics Farm Equipment - BEA Farm projections Light Commercial Equipment - BEA Wholesale/Retail Trade projections Logging Equipment - No growth assumed Airport Service Equipment - BEA Transportation/Utilities projections Aircraft - BEA Transportation/Utilities projections Recreational Marine Vessels - Population statistics Locomotives - No growth assumed
Solvent Usage	Maricopa County	Growth factors for dry cleaning and industrial/domestic solvent usage based upon information from *1992, 1996 and 2005 Baseline Projection Growth Factors for Ozone Emissions*, Maricopa County Environmental Management and Transportation Agency, November 1993. Growth factor for degreasing solvent usage based upon population statistics.

Table 2-1

(Continued)

Category	Sub-Category	Growth Factor
(Degreasing, Dry Cleaning, Industrial Solvent Usage, and Domestic Solvent Usage)	Pima, Pinal, and Gila Counties	Growth factors based on population statistics and Bureau of Economic Analysis (BEA) projections data. Degreasing - Population statistics Dry Cleaning - Population statistics Industrial Solvent Usage - BEA Average Manufacturing projections Domestic Solvent Usage - Population statistics
Other	Residential Wood Combustion	Growth factor based on population statistics.
	Agricultural Pesticides	Growth factors for Maricopa County based upon information from *1992, 1996 and 2005 Baseline Projection Growth Factors for Ozone Emissions*, Maricopa County Environmental Management and Transportation Agency, November 1993. Growth factors for other counties based on Bureau of Economic Analysis (BEA) Farm projections.
	Chloroform from pools	Growth factor based on population statistics

Table 2-2

Summary of Arizona HAP Control Factor Development for Key Sources

Category	Sub-Category	Control Factor
On-Road Mobile Sources	Maricopa County	1993 Maricopa County input file parameters for U.S. EPA's MOBILE5a emission factor model adjusted for the year 2005. Control factor based on the ratio of 2005 emission factors to baseline emissions. Effects of committed measures relating to vehicle inspection and maintenance and fuel vapor pressure (H.B. 2001 and local government commitments), and fleet turnover are modeled. See text for details.
	Pima County	1993 Pima County input file parameters for MOBILE5a model adjusted for the year 2005. Control factor based on the ratio of 2005 emission factors to baseline emissions. Only the effects of fleet turnover are modeled.
	Pinal and Gila Counties	Original emissions were not estimated using MOBILE5a model. MOBILE5a input file parameters created for baseline emissions and 2005. Control factor based on the ration of 2005 emission factors to 1990 emission factors. Only the effects of fleet turnover are modeled.

Table 2-2

(Continued)

Category	Sub-Category	Control Factor
Off-Road Mobile Sources (Vehicles and Equipment)	All four counties	Effects of current and mandated federal rules as detailed in 11/28/94 memo from Phil Lorang to Regional Air Directors, "*Future Nonroad Emission Reduction Credits for Court-Ordered Nonroad Standards*" are modeled for the following categories: Recreational Vehicles Construction Equipment Industrial Equipment Lawn & Garden Equipment Farm Equipment Light Commercial Equipment Logging Equipment Airport Service Equipment No additional controls beyond the baseline emissions were assumed for aircraft, locomotives, and marine equipment and vessels.
Solvent Usage	Degreasing	Effects of National Emission Standard for Hazardous Air Pollutants (NESHAP) for halogenated solvent degreasing modeled.
	Dry Cleaning	Effects of National Emission Standard for Hazardous Air Pollutants (NESHAP) for perchloroethylene dry cleaning modeled.
	Industrial Solvent Usage	No control assumed.
	Domestic Solvent Usage	No control assumed.
Other	Residential Wood Combustion	Effects of fireplace and wood stove emissions modeled based upon information from *Technical Supporting Document for Payson, Arizona PM_{10} State Implementation Plan.*
	Agricultural Pesticides	No control assumed.
	Chloroform from pools	No control assumed.

2.1 On-Road Mobile Source Growth and Control Factors

In the four regions examined in this emissions inventory, on-road mobile sources contributed a major percentage of combustion-related VOC compounds. These compounds include 1,3-butadiene, acrolein, hexane, formaldehyde, benzene, and acetaldehyde. In addition, on-road mobile sources were large sources of evaporative VOC compounds from gasoline, such as benzene and toluene. Although PM is formed by on-road mobile source combustion, it is generally smaller in magnitude than PM from other area sources.

On-road mobile source emissions are subject to both considerable growth and control. Growth of on-road mobile source emissions is due to increasing population and VMT within the state of Arizona. As a result, the on-road mobile source growth factors used in this emissions inventory are significant.

Control of on-road mobile source emissions is the result of vehicle fleet turnover and adopted regulations and control measures. Over time, older vehicles are gradually removed from the total vehicle fleet and replaced by newer, lower-emitting vehicles. The net effect of this fleet turnover is a gradual lowering of the fleet average emissions. This natural fleet turnover was modeled using MOBILE5a for the baseline year (1990 or 1993 depending on the baseline year for the emission estimates) and the year 2005. This resulted in significant control factors in all four counties. In Maricopa County, this reduction is augmented by various control measures mandated by the Arizona Legislature. It was not possible to model all mandated regulations; however, the resultant effects from the major regulations were included in the Maricopa County control factor. The modeled regulations included:

- Implementation of the IM240 inspection and maintenance (I/M) program for 1981 and newer model year vehicles;
- Reduction of the pre-1981 vehicle waiver rate from 10% to 4%;
- Addition of functional pressure and purge checks to the existing anti-tampering program (ATP);
- Increase of ATP compliance rate from 97% to 98%;

- Addition of inspections for vehicle evaporative control system, PCV (positive crankcase ventilation) system, and gas cap to the existing ATP; and

- Reduction of gasoline RVP (Reid Vapor Pressure) from 7.5 to 7.0 pounds per square inch (psi).

Table 2-3 summarizes the on-road mobile source growth and control factors for each county. Control factors are presented as a range for each county with the high and low values for all seasons and vehicle types.

Table 2-3

On-Road Mobile Source Growth and Control Factors

County	Growth Factor	Control Factors[a]
Maricopa	1.48	0.34 - 0.90
Pima	1.34	0.43 - 0.96
Pinal	1.43	0.34 - 0.96
Gila	1.43	0.31 - 0.95

[a] Control factors included in this table are for VOC emissions only.

2.2 Off-Road Mobile Source Growth and Control Factors

Although off-road mobile sources contribute smaller amounts of VOC emissions to the overall inventory compared to on-road mobile sources, HAP emissions from non-road vehicles and equipment are significant to the inventory. In particular, off-road mobile sources are a significant contributor of 1,3-butadiene, benzene, formaldehyde, toluene, and acetaldehyde emissions.

Like on-road mobile source emissions, off-road mobile source emissions are subject to both growth and control factors. In general, off-road mobile source emissions will increase due to population increases and expansion of various economic sectors (e.g. manufacturing, trade, construction, farming, etc.). For Maricopa County, local growth factors were applied to the various off-road mobile source categories. In the other three counties,

emissions increases were modeled using economic projection statistics from the Bureau of Economic Analysis (BEA).

The basis for control of off-road mobile source emissions is a U.S. EPA memorandum describing resultant emission reductions due to court-ordered standards for non-road equipment. This memorandum states that a VOC control factor of 0.41 will be achieved in the year 2005 for gasoline-powered lawn & garden, light commercial, and logging equipment. All other equipment categories were assumed to have a VOC control factor of 1.00. Data were not available to estimate PM control factors for these sources.

Table 2-4 summarizes the off-road mobile source growth and control factors for each county. Growth factors are presented as a range for each county with the high and low values for all fuel types and equipment categories.

Table 2-4

Off-Road Mobile Source Growth and Control Factors

County	Growth Factors	VOC Control Factors
Maricopa	0.71 - 1.24	0.41 or 1.00
Pima	1.00 - 1.41	0.41 or 1.00
Pinal	1.00 - 1.44	0.41 or 1.00
Gila	1.00 - 1.44	0.41 or 1.00

2.3 Halogenated Solvent Degreasing Growth and Control Factors

For the four counties in this emissions inventory, halogenated solvent degreasing is the largest source of trichloroethylene emissions. It is also a significant source of perchloroethylene and methylene chloride (dichloromethane) emissions.

Like most source categories in Arizona, halogenated solvent degreasing will grow due to the effects of population growth and economic expansion. A growth factor

based on population growth from the 1993 baseline data was applied to halogenated solvent degreasing in all four counties.

Control of halogenated solvent degreasing is being driven by a National Emission Standard for Hazardous Air Pollutants (NESHAP) promulgated by the U.S. EPA. The halogenated solvent degreasing NESHAP (59 Federal Register 61801) estimates that full implementation of the rule would result in a 63% reduction in HAP emissions by 1997 compared to emissions that would result in the absence of the NESHAP. This translates into a control factor of 0.37. This control factor was assumed to remain valid through the year 2005.

Table 2-5 summarizes the halogenated solvent degreasing growth and control factors for each county.

Table 2-5

Halogenated Solvent Degreasing Growth and Control Factors

County	Growth Factors	Control Factors
Maricopa	1.32	0.37
Pima	1.21	0.37
Pinal	1.19	0.37
Gila	1.10	0.37

2.4 Perchloroethylene Dry Cleaning Growth and Control Factors

Perchloroethylene is the largest source of perchloroethylene emissions within the four counties in this emissions inventory. Perchloroethylene dry cleaning emissions will grow almost exclusively due to the effects of population growth. For Maricopa County, local growth factors were applied to this source category. For the other three counties, the growth factor was based upon projected population increases relative to the 1990 baseline for these data.

Like halogenated solvent degreasing, control of perchloroethylene dry cleaning emissions is being driven by a NESHAP rule. The perchloroethylene dry cleaning NESHAP (58 Federal Register 49354) estimates that full implementation of the rule would result in a 44% reduction in HAP emissions by 1996 compared to emissions that would result in the absence of the NESHAP. This translates into a control factor of 0.56. This control factor was assumed to remain valid through the year 2005.

Table 2-6 summarizes the perchloroethylene dry cleaning growth and control factors for each county.

Table 2-6

Perchloroethylene Dry Cleaning Growth and Control Factors

County	Growth Factors	Control Factors
Maricopa	1.41	0.56
Pima	1.30	0.56
Pinal	1.27	0.56
Gila	1.15	0.56

2.5 Solvent Usage Growth and Control Factors

Solvent usage is divided into two distinctively different sub-categories: industrial solvent usage (solvents used in various industrial processes such as wipe cleaning) and domestic solvent usage (solvents used in various consumer products such as room deodorants and disinfectants). 1,4-dichlorobenzene and dichloromethane are significant VOC compounds that result from both industrial and residential solvent usage. In addition, industrial solvent usage contributes trichloroethylene and chlorobenzene to the emissions inventory.

Growth of industrial solvent emissions is dependent upon the expansion of various industrial processes. In Maricopa County, local growth factors developed for the State Implementation Plan (SIP) inventory were applied to both industrial and consumer

solvent usage emissions. In the other three counties, the growth factor for industrial solvent usage was derived from BEA projection statistics for the manufacturing sector. Because the amount of consumer solvent usage is related to the overall population, the growth factor for consumer solvent usage was based upon population statistics.

Unlike the source categories examined in Sections 2.1 through 2.4, industrial and consumer solvent usage are assumed to have no applicable controls. As a result, the control factor is simply 1.00. Table 2-7 summarizes the industrial and consumer solvent usage growth and control factors for each county.

Table 2-7

Industrial and Consumer Solvent Usage Growth and Control Factors

County	Growth Factors	Control Factors
Maricopa - Industrial	1.15	1.00
Pima - Industrial	1.38	1.00
Pinal - Industrial	1.39	1.00
Gila - Industrial	1.39	1.00
Maricopa - Consumer	1.32	1.00
Pima - Consumer	1.21	1.00
Pinal - Consumer	1.19	1.00
Gila - Consumer	1.10	1.00

2.6 Residential Wood Combustion Growth and Control Factors

Residential wood combustion is a major contributor of several HAP compounds. These include chrysene, naphthalene, benzo(a)pyrene, and polycyclic organic matter (POM). This category also contributes significant amounts of 1,3-butadiene and formaldehyde.

Emissions from residential wood combustion will be affected by increased housing demands and regulatory control. The growth of residential wood combustion emissions is most directly related to the number of new housing units. Based on the available data, the growth factor for this category was based upon population statistics.

Reduction of residential wood combustion emissions will result from U.S. EPA emission standards for new wood burning devices. Over time, replacement of existing wood stoves will occur. The control factor in all four counties for residential wood combustion was based upon emissions projection information from the Payson, Arizona PM_{10} SIP. Table 2-8 summarizes the residential wood combustion growth and control factors for each county.

Table 2-8

Residential Wood Combustion Growth and Control Factors

County	Growth Factors	Control Factors
Maricopa	1.42	0.48
Pima	1.30	0.48
Pinal	1.27	0.48
Gila	1.15	0.48

2.7 Agricultural Pesticide Growth and Control Factors

Agricultural pesticide application is a unique contributor of several HAP compounds, which include trifluralin, chlorpyrifos, methyl parathion, and disulfoton. Emissions from pesticide application will be affected by changes in farming activity. In Maricopa County, a local growth factor developed for the SIP inventory was applied to agricultural pesticide application. For the other three counties, the growth factor was derived from BEA projection statistics for the farming sector. These growth factors are subject to be several industry trends, including decreasing farm acreage due to population increases and limited water availability. However, these trends are unquantifiable and, therefore, have been excluded from the development of these growth factors.

It is assumed that there are no applicable regulations that reduce emissions from for pesticide applications. As a result, the control factor is simply 1.00. The industry trend toward the use of lower emitting compounds has not been incorporated into this control factor due to the unquantifiable nature of this trend. Table 2-9 summarizes the growth and control factors for each county.

Table 2-9

Agricultural Pesticide Application Growth and Control Factors

County	Growth Factors	Control Factors
Maricopa	0.71	1.00
Pima	1.41	1.00
Pinal	1.20	1.00
Gila	1.20	1.00

2.8 Swimming Pool Chloroform Growth and Control Factors

Swimming pools contribute a large percentage of chloroform emissions. The growth of emissions is related to the number of new swimming pools, which in turn is related to population growth and new housing demand. For all four counties, the growth factor is based upon population statistics. There are no regulations leading to control of swimming pool chloroform emissions. As a result, the control factor is simply 1.00. Table 2-10 summarizes the growth and control factors for each county.

Table 2-10

Swimming Pool Chloroform Growth and Control Factors

County	Growth Factors	Control Factors
Maricopa	1.32	1.00
Pima	1.21	1.00
Pinal	1.19	1.00
Gila	1.10	1.00

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