Air Toxics Hot Spots Program

Appendices G-J

Guidance Manual for Preparation of Health Risk Assessments

Air, Community, and Environmental Research Branch

Office of Environmental Health Hazard Assessment

California Environmental Protection Agency

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Appendix G:

PAH Potency Factors and Selection of Potency Equivalency Factors (PEF) for PAHs based on Benzo(*a*)pyrene Potency

The Office of Environmental Health Hazard Assessment (OEHHA) has developed a Potency Equivalency Factor (PEF) procedure to assess the relative potencies of PAHs and PAH derivatives as a group. Benzo(a)pyrene (BaP) was chosen as the primary representative of the class of polycyclic aromatic hydrocarbons (PAHs) because of the large amount of toxicological data available on BaP (versus the relatively incomplete database for other PAHs), and because it serves as the referent PAH for the Potency Equivalency Factors. This procedure can address the impact of carcinogenic PAHs in ambient air since they are usually present together. This procedure was approved by the Scientific Review Panel (SRP) on Toxic Air Contaminants (TAC) as part of the Health Effects Assessment of Benzo(a)pyrene during the TAC identification process (OEHHA, 1993).

Due to the variety of data available on the carcinogenicity and mutagenicity of PAHs, an order of preference for the use of available data in assessing relative potency was developed. If a health effects evaluation and quantitative risk assessment leading to a cancer potency value had been conducted on a specific PAH, then those values were given the highest preference. Cancer potency values for PAHs developed by this process are shown in Table G-1.

TABLE G-1: POTENCIES OF PAHS AND DERIVATIVES[1]

Chemicals	Cancer potency factors (mg/kg-day)$^{-1}$	Unit risks (µg/m^3)$^{-1}$
Benzo[*a*]pyrene	11.5	1.1×10^{-3}
Dibenz[*a,h*]anthracene	4.1	1.2×10^{-4}
7,12-dimethylbenzanthracene	250	7.1×10^{-2}
3-methylcholanthrene	22	6.3×10^{-3}
Naphthalene[2]	0.12	3.4×10^{-5}
5-nitroacenaphthene	0.13	3.7×10^{-5}

[1] Source: OEHHA (1993); Collins *et al*. (1998); OEHHA (2009). It is assumed that unit risks for inhalation have the same relative activities as cancer potencies for oral intake.

If potency values have not been developed for specific compounds, a carcinogenic activity relative to BaP, rather than a true potency, can be developed. These relative activity values are referred to as Potency Equivalency Factors or PEFs. For air contaminants, the relative potency to BaP based on data from inhalation studies would be optimal. Otherwise, intrapulmonary or intratracheal administration studies would be most relevant, since such studies are in the target organ of interest. Next in order of

preference is information on activity by the oral route and skin painting. Intraperitoneal and subcutaneous administration rank at the bottom of the *in vivo* tests considered useful for PEF development because of their lack of relevance to environmental exposures. Next, in decreasing order of preference, are genotoxicity data, which exist for a large number of compounds. In many cases genotoxicity information is restricted to mutagenicity data. Finally, there are data on structure-activity relationships among PAH compounds. Structure-activity considerations may help identify a PAH as carcinogenic, but at this time have not been established as predictors of carcinogenic potency.

Using this order of preference, PEFs were derived for 21 PAHs and are presented in Table G-2 (OEHHA, 1993: Collins *et al.*, 1998).

The cancer potency comparisons show that some PAHs are more potent than BaP, while other PAHs analyzed were less or much less potent. These comparisons indicated that considering all PAHs to be equivalent in potency to BaP would likely overestimate the cancer potency of a PAH mixture, but such an assumption would be health protective and likely to be helpful in a screening estimate of PAH risks (OEHHA, 1993). If one assumes that PAHs are as carcinogenic as they are genotoxic, then their hazard relative to BaP would be dependent on their concentration in the environment. In light of the limited information available on other PAHs, BaP remains an important representative or surrogate for this group of air pollutants.

Detailed descriptions on the criteria used for developing individual PEFs can be found in OEHHA (2009). OEHHA continues to review all recent literature pertaining to the carcinogenicity and mutagenicity of PAHs. New cancer potency values for PAHs may be developed if an adequate health effects evaluation and quantitative risk assessment can be performed. Also, some current PEFs may be modified based on new data. Any changes to the potency values and PEFs for PAHs will be reflected in the HARP program when they occur. It is incumbent on the risk assessor to access the most recent version of the HARP program to ensure that the most up-to-date PAH potency values are used.

TABLE G-2. OEHHA PEF WEIGHTING SCHEME FOR PAHS AND THEIR RESULTING CANCER POTENCY VALUES.[1]

PAH or derivative	PEF	Unit Risk $(\mu g/m^3)^{-1}$	Inhalation Slope Factor $(mg/kg\text{-}day)^{-1}$	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$
benzo[a]pyrene (index compound)	**1.0**	**1.1 X 10^{-3}**	**3.9**	**1.2 X 10^{+1}**
benz[a]anthracene	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
benzo[b]fluoranthene	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
benzo[j]fluoranthene	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
benzo[k]fluoranthene	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
dibenz[a,j]acridine	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
dibenz[a,h]acridine	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
7H-dibenzo[c,g]carbazole	1.0	1.1 X 10^{-3}	3.9	1.2 X 10^{+1}
dibenzo[a,e]pyrene	1.0	1.1 X 10^{-3}	3.9	1.2 X 10^{+1}
dibenzo[a,h]pyrene	10	1.1 X 10^{-2}	3.9 X 10^{+1}	1.2 X 10^{+2}
dibenzo[a,i]pyrene	10	1.1 X 10^{-2}	3.9 X 10^{+1}	1.2 X 10^{+2}
dibenzo[a,l]pyrene	10	1.1 X 10^{-2}	3.9 X 10^{+1}	1.2 X 10^{+2}
indeno[1,2,3-cd]pyrene	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
5-methylchrysene	1.0	1.1 X 10^{-3}	3.9	1.2 X 10^{+1}
1-nitropyrene	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
4-nitropyrene	0.1	1.1 X 10^{-4}	3.9 X 10^{-1}	1.2
1,6-dinitropyrene	10	1.1 X 10^{-2}	3.9 X 10^{+1}	1.2 X 10^{+2}
1,8-dinitropyrene	1.0	1.1 X 10^{-3}	3.9	1.2 X 10^{+1}
6-nitrochrysene	10	1.1 X 10^{-2}	3.9 X 10^{+1}	1.2 X 10^{+2}
2-nitrofluorene	0.01	1.1 X 10^{-5}	3.9 X 10^{-2}	1.2 X 10^{-1}
chrysene	0.01	1.1 X 10^{-5}	3.9 X 10^{-2}	1.2 X 10^{-1}

[1]. Source: OEHHA (1993)

References

Collins, J.F., Brown, J.P., Alexeeff, G.V., and Salmon, A.G. 1998. Potency equivalency factors for some polycyclic aromatic hydrocarbons and polycyclic aromatic hydrocarbon derivatives. Regul. Toxicol. Pharmacol. 28:45-54.

OEHHA, 1993. Benzo[*a*]pyrene as a Toxic Air Contaminant. Part B. Health Effects of Benzo[*a*]pyrene. Air Toxicology and Epidemiology Section, Berkeley, CA.

OEHHA, 2009. <u>The Air Toxics Hot Spots Program Risk Assessment Guidelines; Part II. Technical Support Document for Describing Available Cancer Potency Factors</u>, Office of Environmental Health Hazard Assessment, May 2009.

Appendix H:

Recommendations for Estimating Concentrations of Longer Averaging Periods from the Maximum One-Hour Concentration for Screening Purposes

H.1   Introduction

The U.S. Environmental Protection Agency (U.S. EPA) AERSCREEN air dispersion model can be used to estimate the maximum one-hour concentration downwind from an emissions source. The AERSCREEN model results (or AERMOD with screening meteorological data) can also be used to estimate concentrations for longer averaging periods, such as the maximum annual average concentration. In addition, it is permissible to use the AERMOD air dispersion model in a screening mode with identical meteorological conditions as used in the AERSCREEN model to superimpose results from multiple sources.

This method to assess short-term and long-term impacts may be used as a first-level screening indicator to determine if a more refined analysis is necessary. In the event that representative meteorological data are not available, the screening assessment may be the only computer modeling method available to assess source impacts.

In California, this standard procedure will generally bias concentrations towards over-prediction in most cases when the source is a continuous release. However, in the case when a source is not continuous, these screening factors may not be biased towards over prediction. In this case, we recommend an alternative procedure for estimating screening value concentrations for longer averaging periods than one-hour for intermittent releases.

H.2   Current Procedures

The current screening factors used to estimate longer term averages (i.e., 3-hour, 8-hour, 24-hour, 30-day, and annual averages) from maximum one-hour concentrations in California are shown in Table H.1. The factors are U.S. EPA recommended values with the exception of the 30-day factor. The 30-day factor is an ARB recommended value (ARB, 1994). The maximum and minimum values are recommended limits to which one may diverge from the general case, (U.S. EPA, 1992). Diverging from the general case should only be done on a case by case basis with prior approval from the reviewing agency.

TABLE H.1 RECOMMENDED FACTORS TO CONVERT MAXIMUM 1-HOUR AVG. CONCENTRATIONS TO OTHER AVERAGING PERIODS (U.S. EPA, 2011, 1992; ARB, 1994).

Averaging Time	Range	Typical SCREEN3 Recommended	AERSCREEN Recommended
3 hours	0.8 - 1.0	0.9	1.0
8 hours	0.5 - 0.9	0.7	0.9
24 hours	0.2 - 0.6	0.4	0.6
30 days	0.2 - 0.3	0.3	
Annual	0.06 - 0.1	0.08	0.1

AERSCREEN automatically provides the converted concentration for longer than 1-hour averaging periods. For area sources, the AERSCREEN 3, 8, and 24-hour average concentrations are equal to the 1-hour concentration. No annual average concentration is calculated. SCREEN3 values are shown for comparison purposes.

H.3 Definitions

It is convenient to define the following terms relating to sources with respect to the duration of the release.

- Continuous Release – This is a release that is continuous over the duration of a year. An example of this type of release would be fugitive emissions from a 24-hour per day, 7-day per week operation or an operation that is nearly continuous.
- Intermittent Release – Many emissions fall under this category. These are emission types that are not continuous over the year. Any operation that has normal business hours (e.g., 8 am to 6 pm) would fall into this category.
- Systematic Release – These are intermittent releases that occur at a specific time of the day. As an example, these type of releases can occur when a process requires clean out at the end of the work day. Thereby releasing emissions only at the end of the workday systematically. Systematic releases are similar to intermittent releases with a shorter duration during the normal operating schedule.
- Random Release – These are intermittent releases that can occur any time during the operating schedule. An example of this type of release would be of the type that depends on batch processing. For example, a brake shop may emit pollutants only when the brakes are cleaned which happens randomly throughout the normal business hours.

H.4   Screening Factors

The U.S. EPA screening factors, as shown in Table H.1, compensate for the effects of varying conditions of wind speed, wind direction, ambient temperature, atmospheric stability, and mixing height over longer averaging periods, even though it is not explicitly indicated in the U.S. EPA Guidance (U.S. EPA, 1992). Figure H.1 shows the variability in wind direction over a 24-hour period. The data are averaged for two seven-day periods from data collected at Los Angeles International Airport (LAX). Figure H.1 was compiled for data collected in 1989 for January 1 to January 7 and June1 through June 7, 1989. The ordinate in Figure H.1 shows the months of the year. Only two months are plotted. The abscissa shows the hour of the day.

FIGURE H.1: HOURLY WIND DIRECTION -
**   LOS ANGELES, JANUARY (BOTTOM – 1) AND JUNE (TOP - 6)**
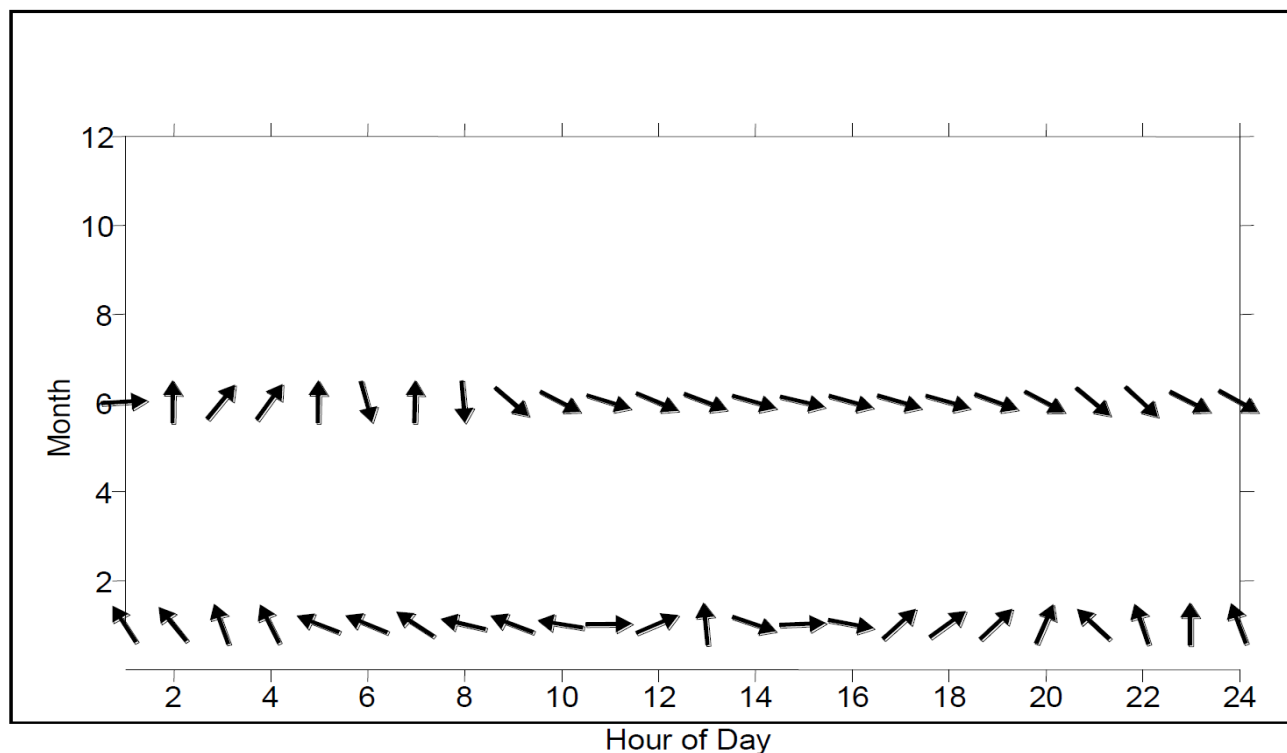
As seen in Figure H.1, the wind direction changes throughout all hours of the day. In
  ddition, the wind direction for LAX, in the overnight and early morning hours, can vary from January to June. During the afternoon hours of 1400 – 1600, the wind direction is similar in both months of January and June.

The standard U.S. EPA screening factor to estimate the maximum 24-hour concentration from the maximum 1-hour concentration is 0.4, as seen in Table H.1. Figure H.2 shows that for 15 of 24 hours the wind blows from the west-northwest during June. A 24-hour screening factor could be 0.6 (0.6 ≈ 15hrs/24hrs) based on wind direction alone. This is consistent with the upper bound of the adjustment factors shown in Table H.1. Including the variability for wind speed, ambient temperature, and

atmospheric stability could further reduce the estimated scaling factor of 0.6 closer towards the U.S. EPA recommended value of 0.4.

H.5 Intermittent Release

Support for the U.S. EPA screening factor is demonstrated for a continuous release (i.e., 24 hours per day) in the description above. It is important to be cautious when applying the U.S. EPA screening factors to an intermittent source for the purposes of estimating an annual average concentration (e.g., a business that may only emit during normal operating hours of 8 am to 6 pm).

Intermittent emissions, such as those from burning barrels, testing a standby diesel generator, or any normal business hour operation (e.g., 8am to 6pm Monday through Friday), could have the effect of eliminating some of the annual variability of meteorological conditions. For example, emissions only during the daytime could eliminate the variability of a drainage flow pattern in mountainous terrain. Guidance for estimating long-term averages for a screening approach and intermittent emissions is not available.

For a source located in the LAX meteorological domain, an emission pattern confined to the hours of 1400 to 1600 would eliminate any variability associated with the wind direction. In this case, estimating a 24-hour average with the U.S. EPA scaling factor of 0.4 would be incorrect.

In the event the emissions are intermittent but randomly distributed throughout the day, the scaling factor of 0.4 may be appropriate because the natural diurnal variability of meteorological conditions are concurrent with emissions. An additional pro-rating of the concentration, when estimating a 24-hour concentration, would be required to discount due to the intermittent nature of the emissions.

We recommend the following steps to estimate a screening based estimate of annual average concentrations from intermittent emissions.

1. Estimate the maximum one-hour concentration (χ_{1-hr}) based on the AERSCREEN model approach (or similar, e.g., AERMOD with screening meteorological data) for possible meteorological conditions consistent with the operating conditions and the actual hourly emission rate. It is acceptable to estimate downwind concentrations using all meteorological combinations available to AERSCREEN. However, it is possible to be selective for the choices of meteorological conditions and still be conservative. For example, daytime only emissions need not be evaluated for nighttime stable atmospheric conditions.

2. Estimate the concentration for the longest averaging period applicable based on the length of time of the systematic or randomly distributed emissions and the factors in Table H.1. For example, the longest averaging period concentration that may be estimated with the U.S. EPA scaling factors is an 8-hour concentration (χ_{8-hr}) for emissions that are systematically released for 12 hours.

Scaling factors between 8-hours and 12-hours are not available. In the case of the 8-hour concentration, the U.S. EPA screening factor of 0.7 ± 0.2 to estimate the maximum 8-hour concentration is appropriate.

The U.S. EPA Screening Guidance allows for deviation from the suggested conversion factor on a case-by-case basis. We recommend the lower end of the range for the conversion factor (i.e., 0.5 for the 8-hour average) when estimating an annual average concentration. This is because variability associated with seasonal differences in wind speed, wind direction, and atmospheric stability would not be addressed otherwise. As seen in Figure H.1, there are seasonal differences in the wind direction.

For example, if X is the length of time of systematic or randomly distributed emissions, the following scalars can apply.

- $X \leq 2$ hrs; Scalar = 1.0 to estimate a 1-hour average
- 3 hrs $\leq X \leq 7$ hrs; Scalar = 0.8 to estimate a 3-hour average
- 8 hrs $\leq X \leq 20$ hrs; Scalar = 0.5 to estimate an 8-hour average (the selection of 20 hours is arbitrary)
- 21 hrs $\leq X \leq 24$ hrs; this may be a continuous release, use standard screening procedures.

3. Estimate the annual average concentration (χ_{annual}) by assuming the longer averaging period estimated above is persistent for the entire year. In the above example the 8-hour concentration is assumed to be persistent for an entire year to estimate an annual average concentration (i.e., the annual average concentration is assumed to be equal to the 8-hour concentration).

In addition, the annual average concentration should be pro-rated over the final averaging period based on the pro-rated emissions (i.e., the calculation should include the fact that for some hours over the year, the emission rate is zero).

For example, if Y is the number of operating hours in the year (e.g., Y = X * 365), the following may apply.

$$(\chi_{annual}) = (\chi_{1\text{-}hr})\ (\text{Scalar})\ (Y/8760\text{hrs/yr})$$

4. The hourly emission rate should be calculated based on the assumed operating schedule in the steps above. An example for a facility operating Y hours per year follows.

$$(q_{hourly}) = (Q_{yearly})/(Y \text{ hrs/yr})$$

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

$$GLC = (\chi_{annual}) (q_{hourly})$$

$$= (\chi_{1\text{-}hr})(Scalar) (Yhrs/8760hrs) (Q_{yearly})/(Y \ hrs/yr)$$

$$= (\chi_{1\text{-}hr})(Scalar) (Q_{yearly})/(8760 \ hrs/yr)$$

Practically speaking, the above five steps condense down to determining three values. The first value is the maximum 1-hour concentration. The second value is the Scalar (either 1.0, 0.8, or 0.5). And the third value is the hourly emission rate estimated by emissions uniformly distributed over the entire year (8760 hours). The operating hours per year drops out of the calculations for an annual average concentration provided the emissions are based on an annual inventory (See step 5).

In the event that the acute averaging period is required and the emissions are based on an annual inventory, then the annual operating hours are required.

Below are four examples using the steps as outlined above. In each case, the annual average concentration is the desired value for use in risk assessment calculations.

Example 1 - Fugitive Gasoline Station Emissions

Emissions are **continuous** for 24 hours per day and 365 days per year.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), $\chi_{1\text{-}hr}$.

2. Estimate the annual average concentration, $\chi_{annual,}$ with the U.S. EPA screening factor of 0.08.

3. $(\chi_{annual}) = (\chi_{1\text{-}hr})(0.08)$

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 24 hours per day and 365 days per year (8760 hours per year).

5. $(q_{hourly}) = (Q_{yearly})/(8760 \ hrs/yr)$

6. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

$$GLC = (\chi_{annual}) (q_{hourly})$$

$$GLC = (\chi_{1\text{-}hr})(0.08) (Q_{yearly})/(8760 \ hrs/yr)$$

Example 2 - Dry Cleaner Emissions

Emissions are **intermittent** over the year but **systematic** for 10 hours per day, 5 days per week and 50 weeks per year.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), χ_{1-hr}.

2. Estimate the maximum 8-hour average concentration, $\chi_{8-hr,}$ with the U.S. EPA screening factor of 0.7 $\pm$0.2 as the longest averaging period of continuous release. The averaging period would need to be less than 10 hours. Use the lower range of the screening factor, 0.5, because the annual average is the final product and variability due to seasonal differences are not accounted for otherwise.

    $(\chi_{8-hr}) = (\chi_{1-hr})(0.5)$

3. Assume the worst-case 8-hour concentration is persistent throughout the year and pro-rate the concentration based on emissions over the year. For this dry cleaner, there are 2500 hours of operating condition emissions. Therefore the annual average is calculated as follows.

    (χ_{annual}) $= (\chi_{8-hr})$ (2500hrs/8760hrs)

    $= (\chi_{1-hr})(0.5)$ (2500hrs/8760hrs)

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 2500 hours per year.

    $(q_{hourly}) = (Q_{yearly})/(2500 \text{ hrs/yr})$

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

    $GLC = (\chi_{annual}) (q_{hourly})$

    $= (\chi_{1-hr})(0.5)$ (2500hrs/8760hrs) $(Q_{yearly})/(2500 \text{ hrs/yr})$

    $= (\chi_{1-hr})(0.5)$ $(Q_{yearly})/(8760 \text{ hrs/yr})$

Example 3 - Burning Barrel Emissions

Emissions are **intermittent** over the year and **random** during daylight hours for two hours per burn, two burns per week, and 52 weeks per year.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), χ_{1-hr}. Meteorological combinations may be restricted to daytime conditions for this screening analysis.

2. Estimate the maximum 8-hour average concentration, χ_{8-hr}, with the U.S. EPA screening factor of 0.7 ±0.2 as the longest averaging period where the emissions have the potential to be randomly distributed. Depending on the day of the year and latitude of the emissions, the daylight hours can vary. For this example, we assume the daylight hours can be as short as 10 hours per day to as long as 14 hours per day. Since the emissions are randomly distributed throughout the daylight hours, the longest averaging period we can scale with U.S. EPA scaling factors is a 10 hour average. In this case, the averaging period becomes the 8-hour average and the scaling factor becomes 0.7±0.2. Again since this is for an annual average, we use the lower end of the range, 0.5.

    $$(\chi_{8-hr}) = (\chi_{1-hr})(0.5)$$

3. Assume the worst-case 8-hour concentration is persistent throughout the year and pro-rate the concentration based on the emissions over the year. For the burning barrels there are 208 hours of operating condition emissions (208 hrs = (2hrs/burn)(2burns/wk)(52wk/yr)). Therefore the annual average concentration is calculated as follows.

    $$(\chi_{annual}) = (\chi_{8-hr}) \ (208hrs/8760hrs)$$

    $$= (\chi_{1-hr})(0.5) \ (208hrs/8760hrs)$$

4. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 208 hours per year.

    $$(q_{hourly}) = (Q_{yearly})/(208 \ hrs/yr)$$

5. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

    $$GLC = (\chi_{annual}) \ (q_{hourly})$$

    $$= (\chi_{1-hr})(0.5) \ (208hrs/8760hrs) \ (Q_{yearly})/(208 \ hrs/yr)$$

    $$= (\chi_{1-hr})(0.5) \ (Q_{yearly})/(8760 \ hrs/yr)$$

The above methods were used in a recent modeling evaluation for emissions from a burning barrel (example 3 above) (ARB, 2002). Table H.2, below, shows results from the modeling evaluation. Shown in Table H.2 are the maximum annual average concentrations based on the screening approach outlined above as well as a refined approach with site specific meteorological data from four locations, Alturas, Bishop, San Benito, and Escondido. As seen in Table H.2, the screening evaluation as described in the example overestimates the values calculated based on the refined analysis. This is the desired outcome of a screening approach.

TABLE H.2: MAXIMUM ANNUAL AVERAGE CONCENTRATION (χ/Q) ABOVE AMBIENT CONDITIONS - BURNING BARREL EMISSIONS

Met. City	**Alturas**	**Bishop**	**San Benito**	**Escondido**	Screening
D (m)	**(μg/m^3)/(g/s)**	**(μg/m^3)/(g/s)**	**(μg/m^3)/(g/s)**	**(μg/m^3)/(g/s)**	*(μg/m^3)/(g/s)*
20	44	61	85	110	*590*
50	12	16	22	30	*230*
100	4	5	7	9	*85*

Notes: (a) Annual χ/q is based on 208 hours of emissions at 1 g/s.

(b) χ/q is the concentration in μg/m^3 based on an hourly emission rate of 1 g/s.

Example 4 - Standby Diesel Engine Testing

Emissions are **intermittent** over the year and **systematic** for two hours per week and 50 weeks per year. The engine testing is conducted at 2 pm on Fridays.

1. Estimate the maximum 1-hour concentration with the AERSCREEN model (or similar screening modeling approach), χ_{1-hr}. Meteorological combinations may be restricted to daytime conditions in this screening analysis because the engine test is conducted at 2 pm.

2. In this case, the emission schedule is systematically fixed over a two hour period. Therefore, the longest averaging period which is applicable for the U.S. EPA screening factors is one-hour because a two-hour conversion factor is not available. Therefore, we assume the maximum 1-hour concentration is persistent for the entire year. We still prorate the concentration based on the emissions. There are 100 hours of engine testing per year. Therefore the annual average concentration becomes.

$$(\chi_{annual}) = (\chi_{1-hr}) \ (100hrs/8760hrs)$$

3. The hourly emission rate, q_{hourly}, for the annual average concentration is based on 100 hours per year.

$$(q_{hourly}) = (Q_{yearly})/(100 \ hrs/yr)$$

4. The annual average concentration (or ground level concentration GLC) can be estimated as follows.

$$GLC = (\chi_{annual})\,(q_{hourly})$$

$$= (\chi_{1-hr})\,(100hrs/8760hrs)\,(Q_{yearly})/(100\ hrs/yr)$$

$$= (\chi_{1-hr})\,(Q_{yearly})/(8760\ hrs/yr)$$

H.6 Implementation

The approach outlined above has been implemented in the Hot Spots Analysis and Reporting Program (HARP). The HARP software has been developed in consultation with OEHHA, Air Resources Board (ARB), and District representatives. The HARP software is the recommended model for calculating and presenting HRA results for the Hot Spots Program. Information on obtaining the HARP software can be found on the ARB's web site at www.arb.ca.gov. Note, since the HARP software is a tool that uses the methods specified in this document, the software will be available after these guidelines have undergone public and peer review, been endorsed by the state's Scientific Review Panel (SRP) on Toxic Air Contaminants, and adopted by OEHHA.

H.7 References

ARB (1994). ARB memorandum dated 4/11/94 from A. Ranzieri to J. Brooks on the subject, "One-hour to Thirty-day Average Screening Factor."

ARB (2002). Staff Report: Initial Statement of Reasons for the Proposed Airborne Toxic Control Measure to Reduce Emissions of Toxic Air Contaminants from Outdoor Residential Waste Burning, January 2002. California Air Resources Board.

U.S. EPA (1992). Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised, October 1992, EPA-454/R-92-019. U.S. Environmental Protection Agency, Research Triangle Park, NC.

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Appendix I:

Calculation Examples for Estimating Potential Health Impacts

This appendix provides four example calculations to illustrate the procedures for estimating the potential health impacts from a facility. The examples are intended to assist the risk assessor in understanding the steps associated with conducting the final step of risk assessment, risk characterization. The four examples provided in this appendix evaluate the inhalation cancer risk, the noncancer acute hazard quotient (HQ) and hazard index (HI), the noncancer 8-hour HQ and HI, and the multipathway (inhalation and oral) noncancer chronic HQ and HI. Specific requirements for health risk assessment (HRA) under the Hot Spots Program are presented in Chapter 8. The HARP software will perform the calculations that are presented here and required in the guidelines. See the ARB's website at www.arb.ca.gov for more information on HARP.

I.1   Sample Calculation for Inhalation Cancer Health Risk Assessment

The following example illustrates the steps for calculating cancer risk at the maximum exposed individual resident (MEIR) using the high-end point-estimate for the inhalation exposure pathway. For each included substance, the steps involved in this sample calculation include:

- Determine the annual average concentration and look-up the inhalation cancer potency factor for each substance
- For each age range, calculate the inhalation dose
- For each age range, calculate the cancer risk using the OEHHA cancer potency factor and the appropriate Age Sensitivity Factor
- Sum the cancer risks from each age range for the exposure duration of interest, and express the risk in chances per million

In this example, the inhalation dose and risk are calculated for the third trimester, ages 0<2, 2<9, 2<16, 16<30, and 16-70 using the high-end daily breathing rate for each age range.

This example focuses on the 30-year cancer risk calculation and does not cover the steps for completing a noninhalation or multipathway HRA. Algorithms to estimate point-estimate and stochastic multipathway exposure can be found in Chapter 5. For simplicity, it is recommended that the risk assessor use HARP to conduct a multipathway risk assessment or stochastic risk assessment.

Step one - Determine the annual average concentration at the MEIR and look-up the inhalation cancer potency factor for each emitted substance.

The risk assessor would obtain the annual average concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.1 presents the annual average concentrations at a hypothetical facility. In addition,

Table I.1 also presents inhalation cancer potency factors for each substance, which also can be found in Chapter 7 and Appendix L. Note that where no inhalation cancer potency has been developed for a substance, the tables in this example have been annotated with N/A since it will not be possible to conduct a quantitative risk assessment for these substances. As previously stated, this example does not take into account multipathway effects for the substances listed in Table I.1. It is recommended that the risk assessor use HARP for conducting such an analysis.

TABLE I.1   ANNUAL AVERAGE CONCENTRATIONS AT THE MEIR AND INHALATION CANCER POTENCY FACTORS

Substance	Annual Average Concentrations ($\mu g/m^3$)	Inhalation Cancer Potency Factor $(mg/kg\text{-}d)^{-1}$
Ammonia	160	N/A
Arsenic	0.0015	12
Benzene	5	0.10
Chlorine	0.08	N/A
Chlorobenzene	20	N/A
2,3,7,8-TCDD (dioxin)	0.000004	130,000
Nickel	0.02	0.91

Step two - Determine the inhalation dose for each substance.

Once you have determined the annual average concentration for the emitted substance, the equation below is used to calculate the inhalation dose for each age range and each substance. This equation is listed in Section 5.4.1 of this document, and is also described in the *Air Toxics Hot Spots Risk Assessment Guidelines: Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 2012)

$$Dose - air = (Cair)\left(\frac{BR}{BW}\right)(A)(EF)\left(1 \times 10^{-6}\right)$$

Where:

Dose-air = Dose through inhalation (mg/kg/d)
Cair   = Concentration in air ($\mu g/m^3$)
BR/BW   = Daily breathing rate normalized to body weight (L/kg BW-day). See Table I.2 for the daily breathing rate for each age range.
A   = Inhalation absorption factor
EF   = Exposure frequency (unitless, days/365 days)
1×10^{-6} = Milligrams to micrograms conversion (10^{-3} mg/μg), cubic meters to liters conversion (10^{-3} m³/l)

A summary of the exposure point-estimates and data distributions for use in risk assessment can be found in Chapter 5 of this document. For more detail on point-estimates and data distributions, see OEHHA (2012). The recommended default values presented in Table I.2 can be used when site-specific information is not available. Note that in this example the mean daily breathing rates listed in the table are for information purposes only. In some cases, the mean value can be used for a Tier-1 risk assessment or applied when multiple noninhalation routes of exposure dominate the risk (See Chapter 8.2.6).

TABLE I.2 RECOMMENDED DEFAULT VALUES FOR INHALATION DOSE EQUATION

Variable	Recommended Default Value		
EF	0.96 (350 days/365 days) Assumes 2-week vacation away from exposure		
Daily Breathing Rates (DBR) (L/kg BW-day) for 9, 30, and 70-year exposures in examples below	**Period**	**Mean**	**95th percentile**
	3rd trimester	225	**361**
	0<2 yrs	658	**1090**
	2<9 yrs	535	**861**
	2<16 yrs	452	**745**
	16<30 yrs	210	**335**
	16-70 yrs	185	**290**
	(For other DBRs see Chapter 5)		
A	1[a]		

[a] OEHHA's Hot Spots inhalation cancer potency factors for the Hot Spots program have already been adjusted where necessary to allow for the inhalation absorption factor, so a value of 1 is used in this equation.

The following equation shows the calculation for the inhalation dose of arsenic for the third trimester by using the annual average concentration for arsenic (Table I.1) and the recommended default values in Table I.2. Note that the high-end (95th percentile) daily breathing rates are used to estimate the third trimester inhalation dose in this example.

$$\text{Arsenic(dose}-\text{air)}_{\text{(third trimester)}} = \left(\frac{0.0015\,\mu g}{m^3}\right)\left(\frac{361\,liters}{kg-day}\right)(1)(0.96)\left(\frac{1\text{x}10^{-3}\,mg}{1\mu g}\right)\left(\frac{1\text{x}10^{-3}\,m^3}{liters}\right)$$

$$\text{Arsenic(dose}-\text{air)}_{\text{(third trimester)}} = 5.2\times10^{-7}\,mg/kg-day$$

To estimate the 30-year arsenic inhalation dose, this calculation is repeated using 95th percentile breathing rates for 0<2 years, 2<16 years, and 16<30 years shown in Table I.2:

$$\text{Arsenic(dose-air)}_{(0<2yrs)} = \left(\frac{0.0015\,\mu g}{m^3}\right)\left(\frac{1090\,liters}{kg-day}\right)(1)(0.96)\left(\frac{1\times10^{-3}\,mg}{1\mu g}\right)\left(\frac{1\times10^{-3}\,m^3}{liters}\right)$$

Arsenic (dose-air) $_{(0<2\,yrs)}$ = 1.6 × 10^{-6} mg/kg-day

$$\text{Arsenic(dose-air)}_{(2<16yrs)} = \left(\frac{0.0015\,\mu g}{m^3}\right)\left(\frac{745\,liters}{kg-day}\right)(1)(0.96)\left(\frac{1\times10^{-3}\,mg}{1\mu g}\right)\left(\frac{1\times10^{-3}\,m^3}{liters}\right)$$

Arsenic (dose-air) $_{(2<16\,yrs)}$ = 1.1 × 10^{-6} mg/kg-day

$$\text{Arsenic(dose-air)}_{(16<30yrs)} = \left(\frac{0.0015\,\mu g}{m^3}\right)\left(\frac{335\,liters}{kg-day}\right)(1)(0.96)\left(\frac{1\times10^{-3}\,mg}{1\mu g}\right)\left(\frac{1\times10^{-3}\,m^3}{liters}\right)$$

Arsenic (dose-air) $_{(16<30\,yrs)}$ = 4.8 × 10^{-7} mg/kg-day

To estimate 70-year exposure, the 95th percentile breathing rate for ages16-70 years in Table I.2 is used instead of the breathing rate for 16<30 years. The arsenic dose for the 16<70 year age bin is 4.2 × 10^{-7} mg/kg-day. Therefore, the age bins that are used for the 70-year scenario include the third trimester, ages 0<2 years, 2<16 years, and 16<70 years.

To estimate the 9-year arsenic inhalation dose, the 95th percentile breathing rates are used for the third trimester, ages 0<2 years and 2<9 years.

These calculations are repeated for each substance under evaluation using their respective annual average concentrations. For our hypothetical facility, we have calculated each inhalation dose for each substance by age bin. In reality, you only need to calculate the dose for the age bins that are required for the exposure duration of interest for your assessment (e.g., 30 years, etc.). However, Table I.3 shows the results from our analysis for all age bins so that potential risk for any exposure duration can be calculated.

TABLE I.3   CALCULATED INHALATION DOSES FOR SUBSTANCES

Substance	Calculated Dose (mg/kg-day)*					
	3rd Tri.	0<2 yrs	2<9 yrs	2<16 yrs	16<30 yrs	16-70 yrs
Ammonia	N/A	N/A	N/A	N/A	N/A	N/A
Arsenic	5.2×10^{-7}	1.6×10^{-6}	1.2×10^{-6}	1.1×10^{-6}	4.8×10^{-7}	4.2×10^{-7}
Benzene	1.7×10^{-3}	5.2×10^{-3}	4.1×10^{-3}	3.6×10^{-3}	1.6×10^{-3}	1.4×10^{-3}
Chlorine	N/A	N/A	N/A	N/A	N/A	N/A
Chlorobenzene	N/A	N/A	N/A	N/A	N/A	N/A
2,3,7,8-TCDD (dioxin)	1.4×10^{-9}	4.2×10^{-9}	3.3×10^{-9}	2.9×10^{-9}	1.3×10^{-9}	1.1×10^{-9}
Nickel	6.9×10^{-6}	2.1×10^{-5}	1.7×10^{-5}	1.4×10^{-5}	6.4×10^{-6}	5.6×10^{-6}

* The doses shown in this table are rounded and the rounded numbers are used for risk calculations in this example.

Step three – Determine inhalation cancer risk for the MEIR.

Once you have calculated the inhalation dose, then the cancer risk is calculated for each age bin and the risk estimates are summed for the exposure duration of interest. To complete this step, the dose for each age bin is multiplied by the inhalation cancer potency factor, the Age Sensitivity Factor (ASF), the exposure duration for the specified age bin over the averaging time (i.e., AT is always 70 years), and the fraction of time spent at home, to determine the cancer risk. Studies have shown that infants and children are more sensitive than adults to exposure to many carcinogens (OEHHA, 2009). Therefore, OEHHA applied ASFs to take into account the increased sensitivity to carcinogens during early-in-life exposure. OEHHA and ARB also evaluated information from activity patterns databases to estimate the percentage of the day that people are home (OEHHA, 2012). This information can be used to adjust exposure duration and risk from a specific facility's emissions, based on the assumption that exposure to the facility's emissions are not occurring while the person is away from home. The ASF and FAH variables are only used when estimating residential cancer risk (e.g., the MEIR)

The risk calculation is performed for each age bin and each substance. The total cancer risk for each substance is the sum of the cancer risks from each age bin for the exposure duration of interest.

$$\text{Cancer Risk} = \left(\text{Inhalation Dose}\,\frac{mg}{kg-day}\right)\left(\text{Cancer Potency}\,\frac{kg-day}{mg}\right)(ASF)(FAH)\left(\frac{EDyrs}{ATyrs}\right)$$

Where:

Cancer Risk	=	Unitless expression of risk (see below)
Inhalation Dose	=	In mg/kg-d
Cancer Potency	=	Chemical specific (in $(mg/kg-d)^{-1}$ or as kg-d/mg)
ASF	=	Age sensitivity factor (unitless)
FAH	=	Fraction of time spent at home (unitless)
ED	=	Exposure duration (years)
AT	=	Averaging time period over which exposure duration is averaged (always 70 years).

The age sensitivity factor, exposure duration, and fraction of time spent at home are shown for each age range in Table I.4. However, if it is determined there is a school located within the cancer risk isopleths of 1×10^{-6} (one chance per million) or greater for the duration of interest (e.g., 30-year analysis), then the fraction of time at the residence is assumed to be one (1) for ages 3rd trimester to less than 16. Thus, cancer risks and the associated isopleths must first be determined using one (1) as the fraction of time at the residence before the FAH values between ages 3rd trimester to less than 16 in Table I.4 can be utilized. See Chapter 8 for more information on calculating a zone of impact, isopleths, and population exposure. In this example, we assume there is no school located within the cancer risk isopleth of 1×10^{-6} or greater; therefore, the FAH factors in Table I.4 are used.

TABLE I.4   INPUTS FOR AGE SENSITIVITY FACTOR, EXPOSURE DURATION, AND THE FRACTION OF TIME SPENT AT HOME

	3rd Tri.	0<2 yrs	2<9 yrs	2<16 yrs	16<30 yrs	16-70 yrs
Age Sensitivity Factor	10	10	3	3	1	1
Exposure Duration (years)	0.25	2	7	14	14	54
Fraction of Time Spent at Home (FAH)	0.85*	0.85*	0.72*	0.72*	0.73	0.73

* FAH is 1 for ages 3rd trimester to less than 16 unless it is determined there is no school located within the cancer risk isopleth of 1×10^{-6} or greater.

The equation below shows the calculation of the cancer risk from arsenic for the third trimester:

$$\text{Arsenic Cancer Risk}_{(third\ trimester)} = \left(\frac{5.2\times10^{-7}\,mg}{kg-d}\right)\left(\frac{12kg-d}{mg}\right)(10)(0.85)\left(\frac{0.25yrs}{70yrs}\right)$$

$$\text{Arsenic Cancer Risk}_{\text{(third trimester)}} = 1.9 \times 10^{-7}$$

To estimate the 30-year cancer risk from arsenic, the cancer risk calculation is repeated for the age bins 0<2, 2<16, and 16<30 years using the appropriate age-related inputs for average daily inhalation dose, ASF, FAH and ED in Tables I.3 and I.4, respectively:

$$\text{Arsenic Cancer Risk}_{(0<2\,\text{yrs})} = \left(\frac{1.6 \times 10^{-6}\, mg}{kg-d}\right)\left(\frac{12kg-d}{mg}\right)(10)(0.85)\left(\frac{2yrs}{70yrs}\right)$$

$$\text{Arsenic Cancer Risk}_{(0<2\,\text{yrs})} \quad = 4.7 \times 10^{-6}$$

$$\text{Arsenic Cancer Risk}_{(2<16\,\text{yrs})} = \left(\frac{1.1 \times 10^{-6}\, mg}{kg-d}\right)\left(\frac{12kg-d}{mg}\right)(3)(0.72)\left(\frac{14yrs}{70yrs}\right)$$

$$\text{Arsenic Cancer Risk}_{(2<16\,\text{yrs})} = 5.7 \times 10^{-6}$$

$$\text{Arsenic Cancer Risk}_{(16<30\,\text{yrs})} = \left(\frac{4.8 \times 10^{-7}\, mg}{kg-d}\right)\left(\frac{12kg-d}{mg}\right)(1)(0.73)\left(\frac{14yrs}{70yrs}\right)$$

$$\text{Arsenic Cancer Risk}_{(16<30\,\text{yrs})} = 8.4 \times 10^{-7}$$

Calculated arsenic cancer risks for each age range are then summed together as shown in the example below to estimate the total 30-year cancer risk from arsenic:

Total Arsenic Cancer Risk $_{(30\text{-year})}$ = Arsenic Cancer Risk $_{(\text{third trimester})}$ +

Arsenic Cancer Risk $_{(0<2\,\text{yrs})}$ +

Arsenic Cancer Risk $_{(2<16\,\text{yrs})}$ +

Arsenic Cancer Risk $_{(16<30\,\text{yrs})}$

$$= (1.9 \times 10^{-7}) + (4.7 \times 10^{-6}) + (5.7 \times 10^{-6}) + (8.4 \times 10^{-7}) = 1.1 \times 10^{-5}$$

To estimate the 70-year cancer risk from arsenic, the cancer risk calculation is repeated for the last trimester to birth, ages 0<2, 2<16, and 16<70 using the appropriate age-related inputs from Tables I.3 and I.4. The calculated arsenic cancer risks for each

age range from the third trimester to age 70 are then summed together to estimate the total 70-year cancer risk from arsenic.

To estimate the 9-year cancer risk from arsenic, the cancer risk calculation is repeated for the last trimester to birth, ages 0<2 and 2<9 using the appropriate age-related inputs from Tables I.3 and I.4. The calculated arsenic cancer risks for each age range from the third trimester to age 9 are then summed together to estimate the 9-year cancer risk from arsenic.

Step four – Express cancer risk in chances per million.

The final step converts the cancer risk in scientific notation to a whole number that expresses the cancer risk in "chances per million"; to complete this step, multiply the estimated cancer risk by a factor of 1×10^6 (i.e., 1 million).

(Total Cancer Risk) (1×10^6) = Total Cancer Risk in chances per million

For a hypothetical facility, the equation below shows the calculation for the inhalation cancer risk of arsenic as a result of 30-year exposure to arsenic:

$$\left(1.1\times10^{-5}\right)\left(1\times10^{6}\right)=11\,chances\,per\,million$$

Use the substance-specific inhalation dose and inhalation cancer potency factor to determine the cancer risk for each substance by repeating these steps. Sum the individual substance cancer risks to give you the total facility (inhalation) cancer risk. Table I.5 shows the individual substance and total facility inhalation cancer risk. In this example, a hypothetical facility poses a (inhalation) cancer risk of 658 chances per million at the MEIR. Note, although not presented here, a facility emitting arsenic or dioxins should also evaluate cancer risk from noninhalation exposure pathways. *Note that although rounding was utilized for ease throughout this example, rounding should not take place until the final answer.*

TABLE I.5   HYPOTHETICAL FACILITY INHALATION 30-YEAR CANCER RISK

Substance	Cancer risk* (chances per million)
Ammonia	N/A*
Arsenic	11
Benzene	310
Chlorine	N/A**
Chlorobenzene	N/A**
2,3,7,8-TCDD (dioxin)	326
Nickel	11
Total Facility Inhalation Cancer Risk	**658**

* The calculated numbers in each step are rounded and the rounded numbers were used in succeeding calculation steps in this example.

 ** N/A: Inhalation cancer potency factor is not applicable.

While this example illustrates the steps used to calculate cancer risk using the inhalation dose algorithm, steps one through four can also be used to calculate noninhalation cancer risk and ultimately multipathway (inhalation and noninhalation pathway) cancer risk. To determine noninhalation cancer risk, an assessor should use the appropriate exposure pathway algorithm presented in Chapter 5. For example, equation 5.4.3.1.1 (Chapter 5) would be used to determine dose for the soil ingestion pathway. Once the assessor has determined the ingestion dose by age group, the cancer risk for that pathway is calculated using the substance-specific oral slope factor and the appropriate age-sensitivity factors. Oral slope factors can be found in Appendix L and Chapter 7. To calculate multipathway cancer risk, the cancer risks for all substances and exposure pathways are summed. See Chapter 8 for further discussion.

 I.2   Sample Calculation of Noncancer Acute Hazard Indices

Risk characterization for noncancer health impacts are expressed as a hazard quotient (for individual substances) or a hazard index (for multiple substances). In addition, all hazard quotients (HQ) and hazard indices (HI) must be determined by target organ system. The example below illustrates the approach for calculating a noncancer acute HQ and HI at the MEIR. The steps involved in this sample calculation include:
1) determining the 1-hour maximum concentration, the acute reference exposure level (REL), and the target organ systems for each substance; 2) calculating the acute HQ for each substance and applying the calculated HQ to the specified target organs for each substance; and 3) calculating the acute HI by summing each HQ from each substance by target organ system. As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding how to calculate an acute HQ and HI. Using HARP, both the acute HQ and HI will automatically be calculated at each receptor. No exposure duration adjustment should be made for acute noncancer assessments. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 8 and 9.

Step one - Determine the 1-hour maximum concentration at the MEIR, the acute reference exposure level, and the target organ systems for each emitted substance.

The risk assessor would obtain the 1-hour maximum concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.6 presents the maximum 1-hour concentrations, target organ systems, and acute RELs for seven substances. Note that where an acute REL has not been developed for a substance, the tables in this example have been annotated with "N/A".

TABLE I.6 CONCENTRATIONS, ACUTE RELS, AND TARGET ORGAN SYSTEM(S) FOR SUBSTANCES AT THE MEIR

Substance	1-hour Maximum Concentration ($\mu g/m^3$)	Acute REL ($\mu g/m^3$)	Target Organ System(s)
Ammonia	1900	3200	Respiratory System; Eye
Arsenic	0.03	0.20	Reproductive/Development ; Cardiovascular System; Nervous System
Benzene	0.54	27	Reproductive/Development; Immune System; Hematologic System
Chlorine	140	210	Respiratory System; Eye
Chlorobenzene	60	N/A	N/A
2,3,7,8-TCDD (dioxin)	0.00001	N/A	N/A
Nickel	0.08	0.20	Immune System

In this example, chlorobenzene and 2,3,7,8-TCDD (dioxin) do not have acute REL values. The acute RELs and their corresponding target organ system(s) can be found in Table 6.1 (Chapter 6) and also in Appendix L.

Step two - Determine the hazard quotient for each substance.

The hazard quotients for each substance are calculated by taking the acute maximum 1-hour concentration and dividing by the substance-specific acute REL. The following equation shows how to calculate the hazard quotient for ammonia.

$$\text{Acute Hazard Quotient} = \frac{\left(\begin{array}{c}Maximum\,1-hr\\Concentration\end{array}\right)}{\left(\begin{array}{c}Acute\\REL\end{array}\right)} \Rightarrow \text{Acute Hazard Quotient}_{(ammonia)} = \frac{\left(1900\,\mu g/m^3\right)}{\left(3200\,\mu g/m^3\right)} = 0.6$$

Step three – Determine the acute HI for all emitted substances.

The acute HQ calculated above for a substance applies to all the target organs listed under that substance. The acute HI is calculated by summing each hazard quotient for each substance by target organ system. For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ system (e.g., reproductive/development system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Table 6.1 for target organ system information. Note, do not add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to that for reproductive/development). Table I.7 shows individual hazard quotients for each substance and total hazard index.

TABLE I.7   INDIVIDUAL HAZARD QUOTIENTS AND TOTAL HAZARD INDEX FOR ACUTE EXPOSURE

Substance	Immune System	Reproductive/ Development	Hematologic System	Nervous System	Cardiovascular System	Respiratory System	Eye
Ammonia						0.6	0.6
Arsenic		0.2		0.2	0.2		
Benzene	0.02	0.02	0.02				
Chlorine						0.7	0.7
Chlorobenzene							
2,3,7,8-TCDD (dioxin)							
Nickel	0.4						
Total Acute Hazard Index*	**0.42**	**0.22**	**0.02**	**0.2**	**0.2**	**1.3**	**1.3**

* The total hazard index is the sum of the rounded individual hazard quotients for each target organ

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system and eyes. Exceeding a hazard index of one may indicate that there is the potential for adverse acute health impacts at this receptor location. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

I.3   Sample Calculation of Noncancer 8-Hour Hazard Indices

The 8-hour RELs are used to evaluate impacts to offsite workers. The 8-hour RELs also apply to exposure of children and teachers during school hours. Although not required in the HRA, 8-hour exposure modeling could also be performed at the discretion of the District to a residential scenario (i.e., the MEIR) where a facility operates only a portion of the day and exposure to residences are not adequately reflected by averaging concentrations over a 24 hour day.

The example below illustrates the approach for calculating a noncancer 8-hour HQ and HI at the maximum exposed individual worker (MEIW) from a noncontinuously emitting facility. An HQ expresses the noncancer 8-hour health impacts for an individual substance and an HI expresses the cumulative potential impacts for multiple substances. All HQs and hazard indices HIs must be determined by target organ system.

The steps involved in this sample calculation include: 1) estimating the daily 8-hour annual average concentration, determining the 8-hour reference exposure level (REL) and the target organ systems for each substance; 2) calculating the 8-hour HQ for each substance and applying the calculated HQ to the specified target organs for each substance; and 3) calculating the 8-hour HI by summing each HQ from each substance by target organ system. As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding the calculation of noncancer 8-hour HQ and HI. Using the HARP software, both the 8-hour HQ and HI will be automatically calculated at each receptor. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 8 and 9.

In this example, the facility emits for a typical schedule of eight hours per day and five days per week and the offsite worker's shift coincides with the facility's emission schedule.

Step one – Estimate the daily 8-hour annual average concentrations at the MEIW from the annual average using an adjustment factor; determine the 8-hour REL and target organ systems for each emitted substance

The risk assessor would obtain the annual average concentrations from the air dispersion modeling results. See Chapter 4 or Appendix M for information on modeling and approximating 8-hour exposure concentrations. This example uses an adjustment factor to approximate the concentration the worker is breathing. Since this is a noncontinuously emitting facility, the annual average concentration is adjusted to represent daily 8-hour average concentration. These steps have been completed for this example. However for completeness, the following equation shows how to calculate the adjustment factor. See Chapter 4 for more explanation.

$$WAF = \frac{H_{residential}}{H_{source}} \times \frac{D_{residential}}{D_{source}} = \frac{24}{8} \times \frac{7}{5} = 4.2$$

Where:

    WAF = the worker adjustment factor
    $H_{residential}$ = the number of hours per day the long-term residential concentration is based on (always 24 hours)
    H_{source} = the number of hours the source operates per day. In this example, we are assuming 8 hours per day.
    $D_{residential}$ = the number of days per week the long-term residential concentration is based on (always 7 days).
    D_{source} = the number of days the source operates per week. In this example, we are assuming 5 days per week.

The daily 8-hour annual average inhalation concentration is then estimated by multiplying the WAF with the annual average concentration:

$$\text{Concentration }_{(8\text{-hour average})} = \text{Concentration}_{(annual\ average)} \times (WAF)$$

Table I.8 shows the daily 8-hour annual average inhalation concentrations at a MEIW, target organ systems, and 8-hour RELs for seven substances. Note that where an 8-hour REL has not been developed for a substance, the tables in this example have been annotated with "N/A"; therefore, Table I.8 lists 8-hour RELs for arsenic and nickel. The 8-hour RELs and their corresponding target organ system(s) can be found in Table 6.2 (Chapter 6) and also in Appendix L.

TABLE I.8   ANNUAL AVERAGE CONCENTRATIONS AND ADJUSTED (AVERAGE DAILY) 8-HOUR CONCENTRATIONS FOR A FACILITY OPERATING 8 HRS/DAY, 5 DAYS/WEEK, AND THE 8-HOUR RELS AND TARGET ORGAN SYSTEM(S) FOR SUBSTANCES

Substance	Annual Average Conc. ($\mu g/m^3$)	Adjusted (Average Daily) 8-Hour Conc. ($\mu g/m^3$)	8-Hour REL ($\mu g/m^3$)	Target Organ System(s)
Ammonia	16	67	N/A	N/A
Arsenic	0.0015	0.0063	0.015	Cardiovascular System; Reproductive/Development; Nervous System: Respiratory System; Skin
Benzene	0.05	0.21	3	Hematologic system
Chlorine	0.08	0.3	N/A	N/A
Chlorobenzene	20	84	N/A	N/A
2,3,7,8-TCDD (dioxin)	0.000001	0.000004	N/A	N/A
Nickel	0.01	0.04	0.06	Respiratory System; Immune System

Step two - Determine the 8-hour hazard quotient for each substance.

Similar to the acute hazard quotient (HQ) calculation shown above, the 8-hour HQs are calculated by taking the noncontinuous source concentration, assumed to be based on an 8-hour facility operation from Table I.8, and dividing by the substance-specific 8-hour REL. The following equation shows how to calculate the hazard quotient for arsenic using the values presented in Table I.8:

$$
\begin{array}{l}
\text{8-Hour} \\
\text{Hazard} \\
\text{Quotient}
\end{array}
=
\dfrac{\left(\begin{array}{c}\textit{Average daily} \\ \textit{concentration}\end{array}\right)}{\left(\begin{array}{c} 8-Hour \\ REL \end{array}\right)}
\quad\Rightarrow\quad
\begin{array}{l}
\text{8-Hour} \\
\text{Hazard} \\
\text{Quotient}_{(arsenic)}
\end{array}
=
\dfrac{\left(0.0063\,\mu g/m^3\right)}{\left(0.015\,\mu g/m^3\right)}
= 0.4
$$

Step three – Determine the HI for all emitted substances.

The 8-hour HQ calculated above for a substance applies to all the target organs listed under that substance. The 8-hour HIs are calculated by summing each HQ for each substance by target organ system. Similar to the example calculation for the acute HI, add the HQs for all substances that impact a specific organ system, then repeat this step for the next target organ system (e.g., respiratory system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Table 6.2 for target organ system information for 8-hour RELs. Note: do not add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to the reproductive/developmental system). Table I.9 shows individual hazard quotients for each substance and total hazard index for each organ system using the information presented in Table I.8.

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system. Exceeding a hazard index of one may indicate that there is the potential for an adverse health impact at this receptor location with repeated daily 8-hour exposures. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

For the MEIW, ideally only an 8-hour noncancer hazard assessment is required. However, development of 8-hour RELs is an ongoing process and many substances that have chronic RELs do not yet have 8-hour RELs. If 8-hour RELs have not been developed yet for all of the emitted chemicals with a chronic REL, as in the example below, then a chronic noncancer hazard assessment is also performed.

TABLE I.9   INDIVIDUAL HAZARD QUOTIENTS AND TOTAL HAZARD INDEX FOR 8-HOUR EXPOSURE

Substance	Respiratory System	Reproductive/ Development	Nervous System	Cardiovascular System	Skin	Immune System	Hematologic
Ammonia							
Arsenic	0.4	0.4	0.4	0.4	0.4		
Benzene							0.07
Chlorine							
Chloro-benzene							
2,3,7,8-TCDD (dioxin)							
Nickel	0.7					0.7	
Total Hazard Index*	**1.1**	**0.4**	**0.4**	**0.4**	**0.4**	**0.7**	**0.07**

* The total hazard index is the sum of the rounded individual hazard quotients for each target organ

I.4   Sample Calculation of Noncancer Chronic Hazard Indices

The example below illustrates the approach for calculating a noncancer chronic HQ and HI at the MEIR. An HQ expresses the noncancer health impacts for an individual substance and an HI expresses the potential impacts for multiple substances. All hazard quotients (HQ) and hazard indices (HI) must be determined by target organ system. The steps involved in this sample calculation include: 1) determining the annual average concentration, the inhalation and oral chronic RELs, and the target organ systems for each substance; 2) calculating the inhalation chronic HQ for each substance and applying the calculated HQ to all target organs for each substance; 3) calculating the noninhalation chronic HQ for each substance and applying the calculated HQ to all target organs for each substance; and 4) calculating the chronic HI by summing each HQ (inhalation and noninhalation) from each substance by target organ system.

As discussed in Chapter 8, the following example is provided to assist the risk assessor in understanding the calculation of a chronic HQ and HI. Using the HARP software, both the chronic HQ and HI will be automatically calculated at each receptor. No exposure adjustments are applied to chronic noncancer assessments. Specific requirements for risk assessment under the Hot Spots Program can be found in Chapters 4, 8 and 9.

Step one - Determine the annual average concentrations at the MEIR and inhalation and oral chronic RELs for each emitted substance.

The risk assessor would obtain the substance-specific annual average concentrations from the air dispersion modeling results. This step has been completed for this example. Table I.10 presents the annual average concentrations, target organ systems, and chronic RELs for seven substances. All of the substances have a chronic REL value associated with them. In addition, arsenic, dioxins, and nickel are multipathway substances; therefore, oral and dermal exposure must be included as potential exposure pathways. The chronic RELs and their corresponding target organ system(s) can be found in Tables 6.3 and 6.4 (Chapter 6) and also in Appendix L.

Step two – Determine the inhalation chronic hazard quotient for each substance.

For inhalation exposure, the individual hazard quotients for each substance are calculated by taking the annual average concentration and dividing by its corresponding chronic inhalation REL. Using the information contained in Table I.10, the equation below is used to calculate the inhalation hazard quotient for arsenic.

$$\text{Chronic Hazard Quotient} = \frac{\left(\begin{array}{c}Annual\,Avg.\\Concentration\end{array}\right)}{\left(\begin{array}{c}Chronic\\REL\end{array}\right)} \Rightarrow \text{Chronic Hazard Quotient}_{(arsenic)} = \frac{\left(0.0015\;\mu g/m^3\right)}{\left(0.015\;\mu g/m^3\right)} = 0.1$$

The inhalation chronic HQ calculated above for a substance applies to all the target organs listed under that substance.

TABLE I.10   ANNUAL AVERAGE CONCENTRATIONS, CHRONIC RELS, AND TARGET ORGAN SYSTEMS FOR SUBSTANCES AT THE MEIR.

Substance	Annual Average Conc. ($\mu g/m^3$)	Chronic Inhalation REL ($\mu g/m^3$)	Target Organ System(s) (inhalation)	Chronic Oral REL (mg/kg-day)	Target Organ System(s) (oral/dermal)
Ammonia	160	200	Respiratory System	-	-
Arsenic	0.0015	0.015	Reproductive/ Development; Cardiovascular System; Nervous System; Respiratory System; Skin	0.0000035	Reproductive/ Development; Cardiovascular system; Nervous System; Respiratory System; Skin
Benzene	0.05	3	Hematologic System	-	-
Chlorine	0.08	0.2	Respiratory System	-	-
Chloro- benzene	20	1000	Alimentary System; Kidney; Reproductive/ Development	-	-
2,3,7,8- TCDD (dioxin)	0.000004	0.00004	Alimentary System (Liver); Reproductive/ Development; Endocrine System; Respiratory System; Hematologic System	0.00000001	Alimentary System (Liver); Reproductive/ Development; Endocrine System; Respiratory System; Hematologic System
Nickel	0.003	0.014	Respiratory System; Hematologic System	0.011	Reproductive/ Development

Step three – Determine the noninhalation hazard quotient for each substance.

For the substances that are subject to deposition, noninhalation (i.e., oral and dermal) exposure pathways need to be considered in the chronic hazard quotient evaluation. The point-estimates and algorithms for calculating the oral dose for all of the applicable exposure pathways and receptors (e.g., workers or residents) are explained in Chapter 5. Note, the HARP software uses the appropriate information and performs all the steps discussed in these examples.

As discussed in Section 8.3.3 for noncancer multipathway assessments, Tier I of the tiered approach to risk assessment states that the high-end point-estimates are used for the two dominant noninhalation exposure pathways and the non-dominant exposure pathways use the mean point-estimates to determine the dose and chronic health impacts at a residential receptor. To determine which exposure pathways are the two dominant ones, high-end point-estimates are used for all applicable noninhalation exposure pathways to see which two pathways provide the highest impacts for each substance. Once the two dominant noninhalation pathways are determined for each substance, the doses for the remaining noninhalation exposure pathway for that substance are recalculated using the average point-estimates. The 70-year dose (i.e., from calculating and adding the exposure contributions from the age 0 through 70 year bins) is used for residential receptors and the dose from the 16 through 70 year age bin is used for the worker evaluation. See Chapters 1, 4, 5, 6, and 8 for more information.

This example shows how to combine the impacts from multiple exposure pathways to obtain an oral (noninhalation) hazard quotient for a single substance. For each substance, the impacts for a specific exposure pathway are assessed by dividing the oral dose (derived from the annual average concentration) in milligrams per kilogram-day (mg/kg-day) by the oral chronic REL, expressed in units of (mg/kg-day) (Table 6.4). The next equation shows the HQ calculation for arsenic through the soil ingestion (SI) exposure pathway.

Note, prior to this point in this calculation, we are assuming several steps have taken place. These steps include: 1) the completion of air dispersion modeling to obtain the ground-level annual-average air concentration; 2) identification of the existing exposure pathways at the receptor location; 3) calculation of the concentration in the exposure media (e.g., for soil - Equation 5.3.2.A); 4) determination of the dominant noninhalation exposure pathway(s) for the substance; and 5) the calculation of the substance-specific dose for that exposure pathway (e.g., Equation 5.4.3.1 is used to calculate the dose from soil ingestion). See Chapter 5 for the algorithms for calculating the oral dose for all of the applicable exposure pathways and receptors.

For this example, the calculated dose for arsenic from soil ingestion is assumed to be 0.000000015 mg/kg-day.

$$\begin{matrix} \text{Chronic} \\ \text{Oral Hazard} \\ \text{Quotient} \end{matrix} = \frac{SI\ dose}{\left(\begin{matrix} Chronic \\ Oral\ REL \end{matrix}\right)} \Rightarrow \begin{matrix} \text{Chronic} \\ \text{Oral\ \ Hazard} \\ \text{Quotient}_{(SI\ arsenic)} \end{matrix} = \frac{(0.000000015 mg/kg - day)}{(0.0000035 mg/kg - day)}$$

$$= 0.04$$

For each substance, this step is repeated for each applicable noninhalation exposure pathway. As illustrated below, the (total) oral HQ for a substance is calculated by summing the HQs for all applicable exposure pathways. In this example, the chronic oral HQ is assumed to equal 0.1 from all exposure pathways.

$$\begin{matrix} \text{Chronic Oral} \\ \text{Hazard} \\ \text{Quotient*}_{(arsenic)} \end{matrix} = [HQ_{(SI)} + HQ_{(D)} + HQ_{(DW)} + HQ_{(MI)} + HQ_{(FI)} + HQ_{(HV)}]$$

$$\begin{matrix} \text{Chronic Oral} \\ \text{Hazard} \end{matrix} = 0.1$$

* Noninhalation pathways:

SI = soil ingestion   FI = fisher-caught fish
DW = drinking water   HV = homegrown vegetables
D = dermal absorption   BM = breast milk (not applicable for arsenic exposure)
MI = meat, milk & egg

The oral chronic HQ calculated above for a substance applies to all the target organs listed under that substance for the noninhalation pathway.

Step four – Determine the chronic HI

The chronic HI is calculated by summing each HQ (inhalation and noninhalation) for each substance by the target organ system(s). For example, add the HQs for all substances that impact the respiratory system, then repeat this step for the next target organ system (e.g., cardiovascular system). This step is repeated until all target organs (for the substances emitted) are individually totaled. See Tables 6.3 and 6.4 for target organ system information. Note, do not add together the HQs or HIs for different target organ systems (e.g., do not add the impacts for the respiratory system to the cardiovascular system). Table I.11 shows individual hazard quotients (inhalation and noninhalation) for each substance and the hazard index by target organ system. In this table, arsenic is highlighted in bold to identify how the information calculated above is presented and used.

In this example, an HQ of one was not equaled or exceeded for any individual substance. However, an HI (the sum of the hazard quotients for each target organ) of one was exceeded for the respiratory system. Exceeding a hazard index of one may indicate that there is the potential for adverse chronic health impacts at this receptor location. The District and OEHHA should be consulted when a hazard index exceeds one (see Section 8.3).

TABLE I.11 SUBSTANCE-SPECIFIC INHALATION AND NONINHALATION HAZARD QUOTIENTS AND THE HAZARD INDEX BY TARGET ORGAN SYSTEM

Substance	Respiratory System	Hematologic System	Alimentary System	Endocrine System	Reproductive/ Development	Kidney	Nervous System	Cardiovascular System	Skin
Ammonia	0.8								
Arsenic	*0.1(i)* *0.1(ni)*				*0.1(i)* *0.1(ni)*		*0.1(i)* *0.1(ni)*	*0.1(i)* *0.1(ni)*	*0.1(i)* *0.1(ni)*
Benzene		0.02							
Chlorine	0.4								
Chloro-benzene			0.02		0.02	0.02			
2,3,7,8-TCDD (dioxin)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)	0.1(i) 0.2(ni)				
Nickel	0.2(i)	0.2(i)			0.1(ni)				
Chronic Hazard Index*	**1.9**	**0.52**	**0.32**	**0.3**	**0.62**	**0.02**	**0.2**	**0.2**	**0.2**

i = inhalation pathway contribution
ni = noninhalation pathway contribution
* The total hazard index is the sum of the rounded individual hazard quotients for each target organ.

I.5   References

OEHHA, 2012a. *Air Toxics Hot Spots Program Risk Assessment Guidelines; Technical Support Document for Exposure Assessment and Stochastic Analysis*. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA Available online at http://www.oehha.ca.gov

OEHHA, 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA. May 2009. Available online at: http://www.oehha.ca.gov

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Appendix J:

Glossary of Acronyms and Definition of Selected Terms

Adverse Health Effect: As defined by U.S. EPA, an adverse health effect is a biochemical change, functional impairment, or pathologic lesion that affects the performance of the whole organism, or reduces an organism's ability to respond to an additional environmental challenge. A health effect from exposure to air contaminants may range from relatively mild temporary conditions, such as eye or throat irritation, shortness of breath, or headaches, to permanent and serious conditions, such as birth defects, cancer or damage to lungs, nerves, liver, heart, or other organs.

AERMOD: A steady-state, plume-based air dispersion model (developed by U.S. EPA) for estimating near-field impacts from a variety of industrial source types. This was designed to provide reasonable concentration estimates over a wide range of conditions with minimal discontinuities, to be easily implemented with reasonable input requirements and computer resource needs, to be based on up-to-date science that captures the essential physical processes while remaining simple, and to be easily revised as the science evolves. To the extent practicable, the structure of the input or the control file for AERMOD is the same as that for the previously used ISCST3 model.

Age Sensitivity Factor (ASF): ASFs are default weighting factors to account for potential increased sensitivity to carcinogens during early life stages including prenatal, postnatal and juvenile life stages. ASFs are applied to the cancer risk equation.

Air Dispersion Modeling: Algorithms, usually performed with a computer, that relate a mass emission rate, source configuration, and meteorological information to calculate ambient air concentrations.

Air District: The Air Pollution Control and Air Quality Management Districts are the agencies responsible for managing air quality on a regional or county basis. California is currently divided into 35 air districts.

Air monitoring: The periodic or continuous sampling and analysis of air pollutants in ambient air or from individual pollutant sources.

Air Toxics Hot Spots Act Emission Inventory Reports: Documents that contain information regarding emission sources, emitted substances, emission rates and release parameters, prepared under the Emission Inventory Criteria and Guidelines (also referred to as "Inventory Reports").

Air Toxics Hot Spots Information and Assessment Act of 1987 (AB 2588): (Health and Safety Code, Section 44300-44394) - A state law which established the "Hot Spots" Program to develop a statewide inventory of site-specific air toxic emissions, to assess the risk to public health from exposure to these emissions, to notify the public of any significant health risks and to reduce emissions below the significant risk levels.

Algorithm: A set of rules for solving a problem in a finite number of steps

California Air Resources Board (ARB): The State's lead air quality management agency consisting of an eleven-member board appointed by the Governor; in addition, the Air Resources Board has an Executive Office and a large staff of scientists and engineers to evaluate air pollution control measures. The ARB is responsible for attainment and maintenance of the state and federal air quality standards, and is fully responsible for motor vehicle pollution control. It oversees county and regional air pollution management programs.

Asthma: A chronic inflammatory disorder of the lungs characterized by wheezing, breathlessness, bronchoconstriction (resulting in chest tightness), and cough.

Atmospheric half-life: The time required for the concentration of a pollutant or reactant to fall to one-half of its initial value.

Benchmark Dose: That dose derived from linear regression, using one or more models of one or more dose-response curves, associated with a specific response rate (such as 1, 5, or 10%) in the test population. This is the starting dose (point of departure) to which uncertainty factors are applied to determine a reference exposure level (REL) using the benchmark dose approach.

Urban Block Groups (BGs): A geographical unit smaller than a census tract used for reporting census data. BGs contain roughly 1,100 persons.

Bioaccumulation: The concentration of a substance in a body or part of a body or other living tissue in a concentration higher than that of the surrounding environment

Bioconcentrate: The process of increasing contaminant concentration in biota up the food chain as contaminants are ingested and concentrated in tissues of organisms higher up in the chain.

Cancer burden: The estimated number of theoretical cancer cases in a defined population resulting from lifetime exposure to pollutants emitted from a facility.

Cancer potency factor (CPF): The theoretical upper bound probability of extra cancer cases occurring in an exposed population assuming a lifetime exposure to the chemical when the chemical dose is expressed in units of milligrams/kilogram body weight-day (mg/kg-d). The CPF is thus expressed in inverse units of mg/kg-d ($(mg/kg\text{-}d)^{-1}$).

California Air Pollution Control Officers Association (CAPCOA): A non-profit association of the air pollution control officers from all 35 air quality districts throughout California. CAPCOA was formed in 1975 to promote clean air and to provide a forum for sharing knowledge, experience, and information among the air quality regulatory agencies around the state.

Cal/EPA: The California Environmental Protection Agency is charged with developing, implementing and enforcing the state's environmental protection laws that ensure clean air, clean water, clean soil, safe pesticides and waste recycling and reduction. Its departments are at the forefront of environmental science, using cutting-edge research to shape the state's environmental laws. The Agency's boards and departments are: the Air Resources Board, the Department of Pesticide Regulation, the Department of Resources Recycling and Recovery (CalRecycle), the Department of Toxics Substances Control, the Office of Environmental Health Hazard Assessment, and the State Water Resources Control Board.

Chemical Abstract Services Registry Number (CAS): The Chemical Abstracts Service Registry Number (CAS) is a numeric designation assigned by the American Chemical Society's Chemical Abstracts Service and uniquely identifies a specific chemical compound. This entry allows one to conclusively identify a material regardless of the name or naming system used.

CCR: California Code of Regulations

CERCLA: Comprehensive Environmental Response, Compensation and Liability Act (Superfund), a federal regulation providing direction and financial support for the clean-up of major hazardous waste sites

Centroid Locations: The location at which calculated ambient concentration is assumed to represent the entire subarea, typically the geometric centroid of an area, but possibly the population-weighted centroid of the area.

Census Tract: A physical area used by the U.S. Census Bureau to compile population and other statistical data.

Criteria Air Pollutant: A pollutant for which the U.S. Environmental Protection Agency or the Air Resources Board has established an Ambient Air Quality Standard (AAQS). Examples include ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, lead, and PM_{10} and $PM_{2.5}$.

Default: A value used to account for a factor when specific information on that factor that applies to a specific situation is not available.

Developmental toxicity: Adverse effects on the developing organism that may result from exposure prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism. Major manifestations of developmental toxicity include: death of the developing organism; induction of structural birth defects; altered growth; and functional deficiency.

Dilution factor (χ/Q): A site-specific quantity defined as a ratio of the ground level concentration in $\mu g/m^3$ to the mass emission rate in g/s and represented by χ/Q.

Dose: A calculated amount of a substance estimated to be received by the subject, whether human or animal, as a result of exposure. Doses are generally expressed in terms of amount of chemical per unit body weight; typical units are mg/kg-day.

Dose-response assessment: The process of characterizing the relationship between the exposure to an agent and the incidence of an adverse health effect in exposed populations.

DTSC: California Department of Toxic Substances Control—DTSC regulates hazardous waste, cleans-up existing contamination, and looks for ways to reduce the hazardous waste produced in California. Its scientists, engineers, and specialized support staff make sure that companies and individuals handle, transport, store, treat, dispose of, and clean-up hazardous wastes appropriately. Through these measures, DTSC contributes to greater safety for all Californians, and less hazardous waste reaches the environment.

ED: Rural Enumeration District. A geographical unit smaller than a census tract used to report census data. EDs contain roughly 1,100 persons.

Emission Inventory Criteria and Guidelines: Regulation and Report adopted by the California Air Resources Board specifying criteria and procedures for the preparation of Air Toxics Hot Spots Act Emission Inventory Reports (Title 17, California Code or Regulations, Sections 93300-93300.5).

Endpoint: An observable or measurable biological or biochemical event including cancer used as an index of the effect of a chemical on a cell, tissue, organ, organism, etc.

Epidemiology: The study of the occurrence and distribution of a disease or physiological condition in human populations and of the factors that influence this distribution.

Exposure: Contact of an organism with a chemical, physical, or biological agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, digestive tract) and available for absorption.

Exposure Pathway: A route of exposure by which xenobiotics enter the human body, (e.g., inhalation, ingestion, dermal absorption).

Fugitive Dust: Dust particles that are introduced into the air through certain activities such as soil cultivation, or vehicles operating on open fields or dirt roadways. A subset of fugitive emissions.

Fugitive Emissions: Emissions not caught by a capture system which are often due to equipment leaks, evaporative processes, and windblown disturbances.

Gaussian Model: An air dispersion model based on the assumption that the time-averaged concentration of a species emitted from a point source has a Gaussian distribution about the mean centerline.

Genotoxic: Having an adverse effect on the genetic material (DNA) resulting in a mutation or in chromosome damage.

GLC: Estimated ground level concentration, usually for a specified averaging time (e.g., annual average, 1 hour, etc.).

GRAF: The Gastrointestinal Relative Absorption Factor, defined as the fraction of contaminant absorbed by the GI tract relative to the fraction of contaminant absorbed from the matrix (feed, water, other) used in the study(ies) that is the basis of either the cancer potency factor (CPF) or the Reference Exposure Level (REL)

Hot Spots Analysis and Reporting Program (HARP): A single integrated software package designed to promote statewide consistency, efficiency, and cost-effective implementation of health risk assessments and the Hot Spots Program. The HARP software package consists of three modules that include: 1) the Emissions Inventory Database Module, 2) the Air Dispersion Modeling Module, and 3) the Risk Analysis and Mapping Module.

Health Risk Assessment (HRA): The name of a computer program developed by the ARB, the OEHHA, and the University of California which was designed to aid in the computation of risk in the Hot Spots program

HSC: Health and Safety Code of the State of California

Haber's Law: The product of the concentration (C) and time of exposure (t) required to produce a specific physiologic effect is equal to a constant level or severity of response (K), or C * t = K.

Hazard Identification: The process of determining whether exposure to an agent can cause an increase in the incidence of an adverse health effect including cancer.

Health Risk Assessment: Health risk assessment is the characterization of the potential adverse health effects of human exposures to environmental hazards. In the Air Toxics Hot Spots program, a health risk assessment (HRA) is an evaluation or report that a risk assessor (e.g., district, consultant, or facility operator) develops to describe the potential a person or population may have of developing adverse health effects from exposure to a facility's emissions. Some health effects that are evaluated could include cancer, developmental effects, or respiratory illness. The pathways that can be included in an HRA depend on the toxic air pollutants that a person (receptor) may be exposed to, and can include breathing, the ingestion of soil, water, crops, fish, meat, milk, and eggs, and dermal exposure.

Health Risk Guidance Value (HRGV): A numerical value with which to compare an exposure level in order to determine the probability of occurrence of an adverse health effect. In the Hot Spots program the toxicity criteria or toxicity values are known as Reference Exposure Levels (RELs) for noncancer effects and as inhalation unit risk factors and cancer potency values for cancer effects.

Hazard Index (HI): The sum of individual acute or chronic hazard quotients (HQs) for each substance affecting a particular toxicological endpoint.

Hazard Quotient (HQ): The estimated ground level concentration divided by the reference exposure level for a single substance and a particular endpoint. For an acute HQ the one hour maximum concentration is divided by the acute Reference Exposure Level (REL) for the substance. For a repeated 8 hr HQ, the 8 hr average concentration is divided by the 8 hr REL. For a chronic HQ, the annual concentration is divided by the chronic REL.

Hot Spot: A location where emissions from specific sources may expose individuals and population groups to elevated risks of adverse health effects, including but not limited to cancer, and contribute to the cumulative health risks of emissions from other sources in the area.

Individual Excess Cancer Risk: The theoretical probability of an individual person developing cancer as a result of lifetime exposure to carcinogenic substances. The Individual Excess Cancer Risk is calculated by summing the potential cancer risks due to both inhalation and noninhalation routes of exposure, generally at the off-site point of maximum impact. This "individual" is the maximally exposed individual (MEI).

Inhalation (Breathing) Rate: The amount of air inhaled in a specified time period (e.g., per minute, per hour, per day, etc.); also called breathing rate and ventilation rate. This is an example of an exposure variate.

Inhalation unit risk factor: The theoretical upper bound probability of extra cancer cases occurring in the exposed population assuming a lifetime exposure to the chemical when the air concentration is expressed in units of microgram/cubic meter ($\mu g/m^3$). The unit risk factor is thus expressed as $(\mu g/m^3)^{-1}$.

Initiator carcinogen: A substance which causes the first stage of carcinogenesis, the conversion of a normal cell to a neoplastic cell. Initiation is considered to be a rapid, irreversible change often involving a change in the DNA caused by the initiator.

Interspecies: Between different species.

Intraspecies: Within the same species.

Industrial Source Complex Dispersion model (ISC3): Air modeling software that was previously used by U.S. EPA and the Hot Spots program. It incorporates three sub-programs into a single program. These are the short-term model (ISCST), the long term model (ISCLT), and the complex terrain model (COMPLEX).

Isopleth: A line on a map connecting points of equal value (e.g., risk, concentration, etc.).

Lowest-observed adverse effect level (LOAEL): The lowest dose or exposure level of a chemical in a study at which there is a statistically or biologically significant increase in the frequency or severity of an adverse effect in the exposed population as compared with an appropriate, unexposed control group.

Margin of safety: The ratio of the no-observed-adverse-effect level (NOAEL) to the estimated human exposure.

Mean: The arithmetic average**.**

MEI: Maximum exposed individual (theoretical)

MEIR: Maximum exposed individual resident (actual)

MEIW: Maximum exposed individual worker (actual)

Meteorology: The science that deals with the phenomena of the atmosphere especially weather and weather conditions. In the area of air dispersion modeling, *meteorology* is used to refer to climatological data needed to run an air dispersion model including: wind speed, wind direction, stability class and ambient temperature.

Milligram: One one-thousandth (10^{-3}) of a gram.

Molecular formula: The formula which identifies the atoms and the number of each kind in the molecules of a compound. Elements in the molecular formula are listed according to the Hill convention (C, H, then other elements in alphabetical order).

Molecular weight: The sum of the atomic weights of the atoms in a molecule. For example, methane (CH_4) is 16.043, the atomic weights are carbon = 12.011, hydrogen = 1.008.

Monte Carlo simulation: Application of random sampling to obtain an approximate value of an expression. Monte Carlo simulation involves computational algorithms that rely on repeated random sampling to obtain numerical results by running simulations many times over in order to calculate probability of a value.

Multipathway substance: A substance or chemical that once airborne from an emission source can, under environmental conditions, be taken into a human receptor by multiple exposure routes, such as inhalation, skin contact with contaminated surfaces, ingestion of soil contaminated by the emission, etc.

No Observed Adverse Effect Level (NOAEL): The highest experimental dose at which there is no statistically or biologically significant increase in frequency or severity of adverse non-cancer health effects in the exposed population compared with an appropriate, unexposed population. Effects may be produced at this level, but they are not considered to be adverse.

Noncarcinogenic Effects: Noncancer health effects which may include birth defects, organ damage, morbidity, and death.

Office of Environmental Health Hazard Assessment (OEHHA): The office within the California Environmental Protection Agency that is responsible for evaluating chemicals for adverse health impacts and establishing safe exposure levels. OEHHA also assists in performing health risk assessments and developing risk assessment procedures for air quality management purposes.

PM_{10}, $PM_{2.5}$: PM_{10} is particulate matter less than 10 μm in diameter; $PM_{2.5}$ is particulate matter less than 2.5 μm in diameter.

PMI: Off-site point of maximum impact. A location, with or without people currently present, at which the total cancer risk, or the total noncancer risk, has the highest numerical value.

Point Estimate: A single value estimate for a given variate.

Potency: The relative effectiveness, or risk, of a standard amount of a substance to cause a toxic response. This term is used particularly to refer to carcinogens.

Potency Slope (also referred to as "Slope Factor" or " Cancer Potency"): Used to calculate the probability or risk of cancer associated with an estimated exposure, based on the assumption in cancer risk assessments that risk is directly proportional to dose and that there is no threshold for carcinogenesis.   It is the slope of the dose-response curve extrapolated to low environmental exposures. It is expressed in per unit dose (usually mg per kg-day): thus cancer potency typically has the units $(mg/kg-day)^{-1}$.

Proposition 65: Safe Drinking Water and Toxic Enforcement Act of 1986, also known as Proposition 65. This Act is codified in California Health and Safety Code Section 25249.5, et seq. .The Proposition was intended by its authors to protect California citizens and the State's drinking water sources from chemicals known to cause cancer, birth defects or other reproductive harm, and to inform citizens about exposures to such chemicals.

Resource Conservation and Recovery Act (RCRA) of 1976: A federal law regulating disposal of hazardous waste.

Receptor: A location with or without people present at which the ground level concentration of an emitted chemical can be estimated.

Refined Models: Air dispersion models designed to provide more representative concentration estimates than screening models taking into account actual meteorological conditions.

Reference Concentration (RfC): An estimate, derived by the U.S. EPA (with an uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population, (including sensitive subgroups) that is likely to be without appreciable risk of deleterious non-cancer effects during a lifetime of exposure. The RfC is derived from a no or lowest observed adverse effect level from human or animal exposures, to which uncertainty factors are applied, and is expressed in units of mg or μg per m^3.

Reference Dose (RfD): An estimate delivered by the U.S. EPA (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subpopulations) that is likely to be without deleterious non-cancer effects during a lifetime. The RfD is reported in units of mg of substance/kg body weight/day for oral exposures.

Reference exposure level (REL): The REL is an exposure level at or below which no noncancer adverse health effect is anticipated to occur in a human population, including sensitive subpopulations, exposed for a specific duration. One hour acute RELs are designed to be protective for infrequent one hour maximum exposures. Eight-hour RELs are designed to be protective for repeated 8 hour exposures. Chronic RELs are designed to be protective for continuous long-term exposures. RELs are used to evaluate toxicity endpoints other than cancer. RELs are expressed in units of μg/m^3 for inhalation exposures and of mg/kg-d for noninhalation exposures.

Reproductive toxicity: Harmful effects on sexual function in males or females, fertility or gestation, caused by exposure of either parent to a substance. Reproductive toxicity also includes developmental effects on the offspring. See also developmental toxicity which refers to adverse effects on the offspring.

Risk: The estimated probability of adverse effects to human health, in this instance from the exposure to environmental hazards.

Risk Assessment: The characterization of the probability of adverse health effects to people from exposure to environmental hazards, in this case of exposure to chemical emissions.

Risk Management: An evaluation of the need for and feasibility of reducing risk. It includes consideration of magnitude of risk, available control technologies, and economic feasibility.

Risk Management and Prevention Program (RMPP): A program administered by the Office of Emergency Services (OES) and local agencies to reduce the frequency and severity of accidental releases of toxic materials.

SB 25 (Children's Environmental Health Protection Act): A state law (Senate Bill 25, Escutia, 1999) that amended the existing Toxic Air Contaminant and Criteria Air Pollutant laws and established requirements for the ARB and the OEHHA to examine the impacts of air pollution more explicitly on children's health. The act required the state to evaluate all ambient air quality standards to determine whether these standards adequately protect human health, particularly that of infants and children; to identify toxic air contaminants that disproportionately impact children, and to ensure that health assessments of toxic chemicals explicitly incorporate considerations of infants and children.

Scientific Review Panel on Toxic Air Contaminants or SRP: A nine-member panel appointed to advise the Air Resources Board, the Office of Environmental Health Hazard Assessment, and the Department of Pesticide Regulation in their evaluation of the adverse health effects and toxicity of substances being evaluated as Toxic Air Contaminants.

Screening Models: Dispersion models used to provide a maximum concentration that is likely to overestimate public exposure.

Sensitive Receptor: A location such as a hospital or daycare center where the human occupants are considered to be more sensitive to pollutants than "average".

Spatial Averaging: The method used in the Hot Spots Program for determining an average air concentration from a grid of receptors over a specified area.

Stationary source: A non-mobile source of air pollutants which can be either a point or area source.

Stochastic: A process that involves random variation.

Synergism: A pharmacologic or toxicologic interaction in which the combined effect of two or more chemicals is greater than the sum of the effects of each chemical alone.

Subcensus Tract: Smaller population unit within a census tract.

Surrogate: As used in this document refers to a single substance category used to represent a family of related chemical compounds, e.g., benzo(a)pyrene in place of POM (polycyclic organic matter).

Threshold, Nonthreshold: A threshold dose is the minimally effective dose of any chemical that is observed in a population to produce a response (e.g., enzyme change, liver toxicity, death). For most toxic effects, except carcinogenesis, there appear to be threshold doses. (Exceptions include observed cardiovascular mortality in humans from exposure to particulate matter, and the neurotoxic effects of lead). Nonthreshold substances are those substances, including nearly all carcinogens, that are known or assumed to have some risk of response at any dose above zero.

Toxic air contaminant (TAC): As defined by California Health and Safety Code, Section 39655 (a): an air pollutant which may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health. Substances, which have been identified by the United States Environmental Protection Agency as hazardous air pollutants (e.g. benzene, asbestos), shall be identified by the Board as toxic air contaminants.

Toxicology: The multidisciplinary study of toxicants, their harmful effects on biological systems, and the conditions under which these harmful effects occur. The mechanisms of action, detection, and treatment of the conditions produced by toxicants are studied.

Uncertainty: True uncertainty is that which is not known about a factor that influences its value.

URF: See inhalation unit risk factor.

UTM Coordinates: Universal Transverse Mercator Coordinates. Coordinates used to define a specific location on earth by means of two values (i.e., easting and northing coordinates).

United States Environmental Protection Agency (U.S. EPA): The mission of EPA is to protect human health and the environment. The agency sets national standards that are enforced by them or that states and tribes enforce through their own regulations. The agency also provide grants to state environmental programs, non-profits, educational institutions, and others for a wide variety of projects, from scientific studies that are used to make decisions to community cleanups. EPA's purpose is to ensure that: 1) All Americans are protected from significant risks to human health and the environment where they live, learn and work; 2) National efforts to reduce environmental risk are based on the best available scientific information; 3) Federal laws protecting human health and the environment are enforced fairly and effectively; 4) Environmental protection is an integral consideration in U.S. policies concerning natural resources, human health, economic growth, energy, transportation, agriculture, industry, and international trade, and these factors are similarly considered in establishing environmental policy; 5) All parts of society -- communities, individuals, businesses, and state, local and tribal governments -- have access to accurate information sufficient to effectively participate in managing human health and environmental risks; 6) Environmental protection contributes to making our communities and ecosystems diverse, sustainable and economically productive; and 7) The United States plays a leadership role in working with other nations to protect the global environment.

Vapor: The gaseous phase of materials at atmospheric temperature and pressure.

Vapor Pressure: The pressure exerted by a chemical vapor in equilibrium with its liquid or solid phase at any given temperature, used to calculate the rate of evaporation of a substance.

Variability: Ability to have different numerical values of a parameter, such as height or weight.

Variate: A variable quantity associated with a probability distribution, in the case of the Hot Spots program, for example, exposure factors (e.g. inhalation rate).

Volatile: Chemicals that rapidly pass off from the liquid state in the form of vapors.

Xenobiotic: A chemical or substance that is foreign to an organism or biological system. A chemical that is foreign to the species in which the chemical is being being studied.

Zone of impact: The area in the vicinity of the facility in which an individual is exposed to a specified cancer risk, usually one in a million or greater.