BAY AREA AIR QUALITY MANAGEMENT DISTRICT

Staff Report

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Appendix B

Methodology for Derivation of Toxic Air Contaminant (TAC) Trigger Levels

BAY AREA AIR QUALITY MANAGEMENT DISTRICT
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B1. INTRODUCTION

The toxic air contaminant (TAC) trigger levels in Table 2-5-1 of the Permit Handbook are used to determine the need for a health risk assessment (HRA) for projects involving new and modified sources. The TAC trigger levels are also used: (1) to establish permit requirements for certain sources that may otherwise qualify for permit exemptions, (2) as part of the applicability of the accelerated permit program, and (3) in determining permit fees. The TAC trigger levels are considered to be reasonable de minimis emission rates for use at a project-level. Projects with emissions below the TAC trigger levels are unlikely to cause, or contribute significantly to, adverse health risks.

The TAC trigger levels were calculated using: (1) target health risk levels that are considered de minimis for project-level risks, (2) the Office of Environmental Health Hazard Assessment/ Air Resources Board (OEHHA/ARB) health effect values, (3) generally conservative modeling procedures which establish the extent to which a TAC is transported and dispersed in the atmosphere after its release from the source, and (4) health-protective assumptions regarding the extent of an individual's exposure to an emitted TAC.

B2. Target Health Risk Levels

For chronic health risk, a lifetime cancer risk of 1.0 in a million (10^{-6}) and a non-cancer hazard index of 0.20, were used as the target health risk levels to derive the chronic trigger levels. These are the risk thresholds at which best available control technology for toxics (TBACT) is required under Regulation 2, Rule 5 and are unchanged from what were previously used to derive the trigger levels in 2010.

Where applicable, the chronic trigger level represents the lesser of the trigger levels determined based on the cancer and non-cancer target health risk levels. In general, for compounds that have both potential cancer and non-cancer adverse health effects, the chronic trigger level presented in Table 2-5-1 is based on the potential carcinogenic health effect, which is more health-protective.

For acute health risk, a hazard index of 1.0 was used as the target health risk level; this is an impact equal to the acute reference exposure level (REL). The acute REL represents an air concentration that is not likely to cause adverse effects in a human population, including sensitive subgroups, exposed on an intermittent basis for a one-hour period. An acute hazard index of 1.0 is also the project risk limit required under Regulation 2, Rule 5.

B3. Health Effect Values

Table 2-5-1 of the Permit Handbook incorporates the most recent health effect values adopted by OEHHA/ARB (as of March 2016) for use in the Air Toxics Hot Spots (ATHS) Program. These include cancer potency factors (CPFs) for carcinogens, and RELs for non-carcinogenic health effects. Some TACs do not appear on Table 2-5-1 because there may not be sufficient data available for OEHHA to establish a CPF or REL. Prior to use in Regulation 2, Rule 5, the District will review any new or revised health effects value adopted by OEHHA/ARB after March 2016. Typically within one year of OEHHA/ARB's adoption of new toxicity criteria, the District will evaluate the new criteria for feasibility of implementation, enforcement, compliance with project risk limits and inclusion in Table 2-5-1.

Although OEHHA has provided acute RELs for carbon monoxide (CO), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2) using the State Ambient Air Quality Standards, trigger levels were not developed for these criteria pollutants because they are regulated in other District programs.

The trigger levels for polycyclic aromatic hydrocarbons (PAHs), polychlorinated dibenzo-p-dioxins (PCDDs, or dioxins), polychlorinated dibenzofurans (PCDFs, or furans), and dioxin-like polychlorinated biphenyls (PCBs) were based on compound groupings. The trigger levels were expressed as B(a)P-equivalent and TCDD-equivalents in order to address cumulative exposures to applicable PAH and PCDD/PCDF/dioxin-like PCB congeners, respectively.

Although acute severity exposure levels (e.g., mild, severe, and life-threatening effects) have been identified for each acute REL, all acute trigger levels were developed based on the same exposure assumptions and target risk levels, regardless of the severity of the adverse health effect corresponding to the acute REL.

B4. Modeling Procedures

The trigger levels in Table 2-5-1 are based on the same screening-level dispersion modeling procedure that was used to develop the trigger levels in Regulation 2, Rule 5, amended January 6, 2010. This involves the use of a cavity effects screening procedure that relates emission rate to one-hour average ambient air concentrations (i.e., dispersion factors, or Chi/Q) where dispersion is affected by aerodynamic downwash from a nearby building. The cavity region occurs immediately adjacent to the lee side of the building and is often the "worst-case" dispersion scenario where receptor areas are in close proximity to the source being evaluated. The cavity effects equation (Equation 1 below) is used to derive the trigger levels; this equation is provided in EPA's Screening Procedures for Estimating the Air Quality Impact of Stationary Sources (EPA, 1992) and is incorporated into the EPA SCREEN3 model (EPA, 1995).

Equation 1: $c_{1\text{-}hr} = q_{1\text{-}hr} / (1.5 * A * u)$

where:
- $c_{1\text{-}hr}$ = one-hour average concentration in air, g/m^3
- $q_{1\text{-}hr}$ = one-hour average emission rate, g/s
- A = cross sectional area of the building normal to the wind, m^2
- u = wind speed, m/s

The cavity effects equation requires the selection of the crosswind building area and the average wind speed. A value of 92.9 square meters was used for the crosswind building area (e.g., a building 25 feet high x 40 feet wide). The average wind speed was taken to be 2 meters per second, based on EPA screening modeling guidelines. Substituting the values for A and u into Equation 1, converting the concentration units to $\mu g/m^3$ instead of g/m^3, and converting the emission rate unit from g/s to lbs/hr yields the following:

$$C_{1\text{-}hr} = (Q_{1\text{-}hr} / (1.5 * 92.9 * 2)) * 1000000 * 453.6 / 3600$$
$$C_{1\text{-}hr} = 452 * Q_{1\text{-}hr}$$

Rearranging for emission rate as a function of the concentration yields:

Equation 2: $Q_{1\text{-}hr} = 2.21\text{ E-}3 * C_{1\text{-}hr}$

where:
- $C_{1\text{-}hr}$ = one-hour average concentration in air, $\mu g/m^3$
- $Q_{1\text{-}hr}$ = one-hour average emission rate, lbs/hour

For each TAC, the acute trigger level was calculated using Equation 2 and the TAC specific acute REL as the concentration. As discussed in section B2, an exposure concentration equivalent to the acute REL would result in the target acute hazard index of one.

For use in determining chronic trigger levels, a 0.1 multiplying factor representing the ratio between annual average and one-hour maximum concentrations was used. This is the high-end value of the range of multiplying factors provided in EPA screening modeling guidelines (EPA, 1982). Substituting the annual average concentration using the factor and converting the hourly emission rate to an annual emission rate Equation 2 yields the following:

Equation 2: $Q_{1\text{-}hr} = 2.21\ E\text{-}3 * C_{1\text{-}hr}$

$Q_{ann} = 2.21\ E\text{-}3 * (C_{ann} / 0.1) * 8760$

Equation 3: $Q_{ann} = 193.8 * C_{ann}$

where: C_{ann} = annual average concentration in air, $\mu g/m^3$

Q_{ann} = emission rate, lbs/year

The chronic trigger levels were calculated using Equation 3. For non-carcinogenic adverse health effects, a TAC specific concentration that would result in the target chronic hazard index of 0.2 is used in the calculation of the chronic trigger level. For carcinogenic health effects, a TAC specific concentration that would result in the target residential cancer risk of one in a million is used in the calculation of the chronic trigger level.

B5. Exposure Assumptions

The exposure assessment assumptions, that are provided in the December 2015 BAAQMD Air Toxics NSR Program HRA Guidelines, were used to estimate trigger levels. These assumptions are summarized in the table below

Table B-1 Residential Exposure Assumptions

Age Range	Daily Breathing Rates		Age Sensitivity Factors	Exposure Duration (ED), years	Fraction of Time Spent at Home (FAH)
	Percentile	L/kg BW day			
3rd Trimester	95	361	10	0.25	1
0 <2 years	95	1090	10	2	1
2 <16 years	80	572	3	14	1
16 <30 years	80	261	1	14	0.73

OEHHA has identified a list of substances that require multi-pathway risk analysis, which are listed in Table B-2. The trigger levels for these compounds have been determined based on the minimum residential multi-pathway exposure routes, which are inhalation, incidental soil ingestion, and dermal contact. For lead, lead compounds, dioxins, furans, PAHs, and PCBs, the breast-milk consumption pathway was also included per OEHHA recommendations. The multi-pathway exposure assessment was performed using CARB's Hotspots Analysis and Reporting Program (HARP) (Version 2.0) using default assumptions. A deposition rate of 0.05 meters per second for "uncontrolled sources" and the "warm" climate selection were chosen in the HARP runs for the multi-pathway risk analyses to yield conservative results. For the HARP cancer risk run, the "RMP using the Derived Method" scenario was selected. For the HARP chronic hazard index run, the "OEHHA Derived Method" scenario was selected.

Table B-2 Substances with Trigger Levels Based on Multi-pathway Exposures

4,4'-Methylene dianiline	Chromium VI & compounds
Fluorides & Hydrofluoric acid	Arsenic & compounds
Diethylhexylphthalate (DEHP)	Beryllium & compounds
Hexachlorocyclohexanes	Lead & compounds
PAHs	Mercury & compounds
PCBs	Nickel & compounds
Cadmium & compounds	Dioxins & Furans
Selenium & compounds	

B6. Trigger Level Calculations

For most of the toxic metals, the OEHHA CPFs and non-cancer RELs apply to the weight of the toxic metal atom contained in the overall compound. The metal compounds contain other elements along with the toxic metal atom (e.g., Nickel hydroxide, has a formula of $Ni(OH)_2$). To ensure that the trigger level is based only on the fraction of the overall weight of the emissions that are associated with health effects of the metal, a molecular weight adjustment factor (MWAF) was applied to derive the trigger level for the metal compounds.

Acute Trigger Levels

The target concentrations used to calculate the acute trigger levels are the acute RELs; this is equivalent to a target acute hazard index of one. Substituting "Acute TL" for $Q_{1\text{-hr}}$, "Acute REL" for $C_{1\text{-hr}}$, and applying the MWAF in Equation 2:

Equation 2: $Q_{1\text{-hr}} = 2.21\text{ E-3} * C_{1\text{-hr}}$

Acute TL = 2.21 E-3 * Acute REL / MWAF

The acute trigger levels presented in Table 2-5-1 were calculated as follows:

*Acute TL = 2.21 E-3 * Acute REL / MWAF*

where: Acute TL = Acute Trigger Level, pounds/hour

Acute REL = Acute Reference Exposure Level (chemical-specific), μg/m^3

MWAF = Molecular Weight Adjustment Factor. For toxic metals the MWAF is the ratio of the molecular weight of the metal atom and the molecular weight of the metal compound. For non-metal compounds the MWAF is one

Chronic Trigger Levels

The chronic trigger levels in Table 2-5-1 represent the lesser of the trigger levels calculated for a carcinogenic and non-carcinogenic adverse health effect.

Chronic Non-carcinogenic Trigger Levels

Chronic trigger levels based on non-carcinogenic adverse health effects were calculated based on a target concentration that is 20% of the chronic REL; this is equivalent to a target chronic hazard index of 0.2. For TACs with non-carcinogenic health effects and an inhalation-only exposure pathway, the chronic trigger levels were calculated using Equation 3, replacing Q_{ann} with "Chronic TL_{nc_inh}", C_{ann} with 20% of the chronic REL and applying the MWAF:

Equation 3: $Q_{ann} = 193.8 * C_{ann}$

Chronic TL_{nc_inh} = 193.8 * (0.2 * Chronic REL) / MWAF

*Chronic TL_{nc_inh} = 38.76 * Chronic REL / MWAF*

where:

Chronic TL_{nc_inh} = Chronic Trigger Level – non-cancer inhalation risk, pounds/year

Chronic REL = Chronic Reference Exposure Level (chemical-specific), μg/m^3

MWAF = Molecular Weight Adjustment Factor. For toxic metals the MWAF is the ratio of the molecular weight of the metal atom and the molecular weight of the metal compound. For non-metal compounds the MWAF is one

For each TAC with multiple exposure pathways for non-carcinogenic adverse health effects, HARP was used to calculate a chronic hazard index for a unit concentration; this value from HARP, "$HARP_{Chronic\_HI}$", can be used to calculate the chronic hazard index (HI) for TACs that have multi-pathway impacts as follows:

Chronic HI = $C_{ann} * (HARP_{Chronic\_HI})$

where: C_{ann} = annual average concentration in air, μg/m^3

$HARP_{Chronic\_HI}$ = Chronic HI from HARP for a unit concentration (chemical specific), Chronic HI / (μg/m^3)

Rearranging for C_{ann} and a target chronic HI of 0.2 yields:

$C_{ann} = 0.2 / (HARP_{Chronic\_HI})$

Substituting C_{ann} into Equation 3 and replacing Q_{ann} with "Chronic TL_{nc_mp}" yields:

Equation 3: $Q_{ann} = 193.8 * C_{ann}$
Chronic $TL_{nc\_mp} = 193.8 * (0.2 / HARP_{Chronic\_HI})$

The chronic trigger levels TACs with non-cancer multi-pathway adverse health effects were calculated as follows:

$$Chronic\ TL_{nc\_mp} = 38.76 / (HARP_{Chronic\_HI})$$

where:
Chronic TL_{nc_mp} = Chronic Trigger Level – non-cancer multi-pathway risk, pounds/year
$HARP_{Chronic\_HI}$ = Chronic HI HARP-value for a unit concentration (chemical specific), Chronic HI / ($\mu g/m^3$)

The HARP software automatically applies the appropriate MWAF for each chemical, so no MWAF adjustment is required.

Chronic Carcinogenic Trigger Levels

Chronic trigger levels based on carcinogenic health effects for the residential receptor were calculated for the inhalation exposure pathway using the following equations from the 2015 OEHHA Risk Assessment Guidelines for the Air Toxics Hot Spots Program:

$RISK_{inh} = (DOSE_{air} * CPF * ASF * ED/AT * FAH)_{age\_group}$
$DOSE_{air} = C_{ann} * \{BR/BW\} * A * EF * 10^{-6}$

Where:
$RISK_{inh}$ = Residential inhalation cancer risk
$DOSE_{air}$ = Daily inhalation dose (mg/kg-day)
CPF = Inhalation cancer potency factor ($mg/kg\text{-}day^{-1}$)
ASF = Age sensitivity factor for a specified age group (unitless)
ED = Exposure duration (in years) for a specified age group
AT = Averaging time for lifetime cancer risk, 70 years
FAH = Fraction of time spent at home
C_{ann} = Annual Average Concentration in air ($\mu g/m^3$)
BR/BW = Daily Breathing rate normalized to body weight (L/kg body weight - day)
A = Inhalation absorption factor (unitless) [default value is 1]
EF = Exposure frequency (unitless) [default for resident = 0.96 = 350 days/365 days]

Substituting $DOSE_{air}$ and the values for exposure assumptions from Table B-1 into the equation for $RISK_{inh}$ results in the following:

$$RISK_{inh} = (C_{ann} * \{BR/BW\} * A * EF * 10^{-6} * CPF * ASF * ED/AT * FAH)_{age\_group}$$

$$RISK_{inh} = (C_{ann} * 361 * 1 * (350/365) * 10^{-6} * CPF * 10 * 0.25/70 * 1)_{3rd\ trimester} + (C_{ann} * 1090 * 1 * (350/365) * 10^{-6} * CPF * 10 * 2/70 * 1)_{0<2years} + (C_{ann} * 572 * 1 * (350/365) * 10^{-6} * CPF * 3 * 14/70 * 1)_{2<16years} + (C_{ann} * 261 * 1 * (350/365) * 10^{-6} * CPF * 1 * 14/70 * 0.73)_{16<30years}$$

$RISK_{inh}$ = 6.766E-04 * (C_{ann} * CPF)

Rearranging this equation for C_{ann} and substituting the target cancer risk of one in a million for $RISK_{inh}$ yields:

C_{ann} = 1 E-06 / (6.766E-04 * CPF)
C_{ann} = 1.478E-03 / CPF

Substituting C_{ann} into Equation 3, replacing Q_{ann} with "Chronic TL_{cr_inh}" and applying the MWAF:

Equation 3: Q_{ann} = 193.8 * C_{ann}
Chronic TL_{cr_inh} = 193.8 * (1.478E-03 / CPF) / MWAF

The chronic trigger levels for carcinogenic TACs that have an inhalation-only pathway were calculated as follows:

Chronic TL_{cr_inh} = 0.2864 / CPF / MWAF

where:

Chronic TL_{cr_inh}	=	Chronic Trigger Level – inhalation cancer risk, pounds/year
CPF	=	Cancer Potency Factor (chemical – specific), $(mg/kg\text{-}day)^{-1}$
MWAF	=	Molecular Weight Adjustment Factor. For toxic metals the MWAF is the ratio of the molecular weight of the metal atom and the molecular weight of the metal compound. For non-metal compounds the MWAF is one

For each TAC with multiple exposure pathways, HARP was used to calculate a residential cancer risk for a unit concentration; this value from HARP, "$HARP_{cancer}$", can be used to calculate the cancer risk for TACs that have multi-pathway impacts as follows:

Cancer risk = C_{ann} * ($HARP_{cancer}$)
where: C_{ann} = annual average concentration in air, $\mu g/m^3$
$HARP_{cancer}$ = Cancer risk HARP value, Cancer risk / ($\mu g/m^3$)

Rearranging for C_{ann} and substituting the target one in a million cancer risk yields:

C_{ann} = 1E-06 / ($HARP_{cancer}$)

Substituting C_{ann} into Equation 3 and replacing Q_{ann} with "Chronic TL_{cr_mp}":

Equation 3: Q_{ann} = 193.8 * C_{ann}
Chronic TL_{cr_mp} = 193.8 * (1E-06 / $HARP_{cancer}$)

The chronic trigger levels for carcinogenic TACs with multi-pathway impacts were calculated as follows:

$$\text{Chronic } TL_{cr\_mp} = 1.938E\text{-}04 / (HARP_{cancer})$$

where:

Chronic TL_{cr_mp}	=	Chronic Trigger Level – multi-pathway cancer risk, pounds/year
$HARP_{cancer}$	=	Cancer risk HARP-value for a unit concentration (chemical specific), Cancer risk / ($\mu g/m^3$)

The HARP software automatically applies the appropriate MWAF for each chemical, so no MWAF adjustment is required.

Differences in the chronic trigger levels listed in Table 2-5-1 of Regulation 2, Rule 5, amended January 6, 2010 and the proposed Table 2-5-1 in the Permit Handbook may be due to one or more of the following factors: (1) revised chemical-specific health effects values (e.g., CPFs and/or RELs) in the 2015 HRA Guidelines and the March 2016 "Table 1 Consolidated Table of OEHHA/ARB Approved Risk Assessment Health Value" update, (2) changes in default multi-pathway exposure parameters or calculations included in HARP2 relative to the original HARP (which was previously used), (3) changes in the exposure assumptions (breathing rates, exposure durations, fraction of time spent at home) and/or (4) changes in the cancer risk calculation methodology. Therefore, although a chemical-specific health effect value may not have been revised, the use of the new exposure assumptions and calculation methodology may result in a significant change in the trigger level.