

**Appendix C:
Health Risk Assessment Methodology
Supplemental Information**

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ACRONYMS and ABBREVIATIONS

ARB	Air Resources Board
BWHRA	Bay-Wide Health Risk Assessment
Cal/EPA	California Environmental Protection Agency
CEQ	Council on Environmental Quality
CEQA	California Environmental Quality Act
CSF	Cancer Slope Factor
DPM	Diesel Exhaust Particulate Matter
EC	Elemental Carbon
HEI	Health Effects Institute
kg	Kilogram
km	Kilometer
L	Liter
m ³	Cubic Meter
mg	Milligram
NRC	National Research Council
OEHHA	California's Office of Environmental Health Hazard Assessment
SCAQMD	South Coast Air Quality Management District
SRP	Scientific Review Panel
µg	Microgram
URF	Unit Risk Factor
USEPA	US Environmental Protection Agency
USGS	United States Geological Survey
WHO	World Health Organization

1 Health Risk Assessment Methodology, Supplemental Information

This Appendix provides details of the methodology used in the Bay Wide Health Risk Assessment (BWHRA) Tool to calculate exposure, individual cancer risk, and population-weighted cancer risk from Ports-associated sources of diesel exhaust particulate matter (DPM). Information is also provided regarding the basis of the DPM cancer slope factor (CSF). The Appendix concludes with a discussion of some of the key uncertainties of the health risk assessment.

1.1 Calculation of Exposure

In the BWHRA Tool, exposure of residential receptors to DPM in ambient air was calculated from the following equation:

$$Exposure = \frac{C_a \times BR \times EF \times ED \times CF}{AT} \quad (\text{Eq. C-1})$$

- C_a = Concentration of DPM in Air (mg/ m³)
- BR = Breathing Rate (302 L/kg-day)
- EF = Exposure Frequency (350 days/year)
- ED = Exposure Duration (70 years)
- CF = Conversion Factor (1000 L/m³).
- AT = Averaging Time (25,550 days)

Exposures were calculated using discrete DPM emission rates estimated for 2005 and 2020 and held constant over the subsequent respective 70-year averaging periods (see Appendix B).

1.1.1 Calculation of Individual Cancer-Risk Attributable to DPM

Individual cancer risk was estimated by calculating the upper-bound incremental probability that an individual will develop cancer over a lifetime as a direct result of exposure to DPM. The equation used to calculate potential excess cancer risk is:

$$Risk = Exposure \times CSF \quad (\text{Eq. C-2})$$

- $Exposure = Exposure\ to\ DPM\ in\ air\ (mg/kg-d)$
- $CSF = DPM\ cancer\ slope\ factor\ 1.1\ (mg/kg-d)^{-1}$.

1.1.2 Calculation of Population-Weighted Cancer-Risk Attributable to DPM

Cancer risks were also analyzed by calculating population-weighted average risk associated with Ports DPM sources for the baseline year of 2005 and for predicted emissions in 2020. Population-weighted risk was calculated as:

$$\text{Risk}_{\text{population-weighted}} = \frac{\sum \text{Risk}_i \times \text{Population}_i}{\sum \text{Population}_i} \quad (\text{Eq. C-3.})$$

Where:

Risk_i = estimated cancer risk at receptor *i*;

Population_i = population of area around receptor *i*.

In the context of population-weighted average risk, receptors represent point locations on two Cartesian grids distributed throughout the modeling domain. The spacing of the receptors within the grids, and the basis for that spacing, are described in section 3.5 of the main BWHRA Tool report. United States Census Bureau data for the year 2000 were used to calculate the population for both 2005 and 2020. Cancer risk for the population within the vicinity of each receptor was estimated by first calculating DPM-attributable cancer risk (Eq. C-2) and then multiplying that risk by the population in the area around the specific modeled receptor (*Risk_i* × *Population_i*). Population-weighted average residential cancer risk for the modeling domain was calculated by summing all receptor-related risks and dividing by the population within the modeling domain as shown in Eq.C-3.

1.2 OEHHA’s Cancer Slope Factor for Diesel Exhaust Particulate Matter

In 1998, the Scientific Review Panel (SRP) of the California Environmental Protection Agency (Cal/EPA) determined that diesel exhaust is carcinogenic to humans (Office of Environmental Health Hazard Assessment [OEHHA] 1998b), and the Air Resources Board (ARB) subsequently listed diesel exhaust as a toxic air contaminant (1998c). A key supporting document for the SRP determination was a human health risk assessment of diesel exhaust conducted by the OEHHA (1998a). OEHHA’s assessment focused on evaluating epidemiologic evidence of the relationship between exposure to diesel exhaust and the likelihood of developing lung cancer. Although multiple epidemiologic studies were considered by OEHHA (1998a), a study of railroad workers (Garshick et al. 1988) served as the primary basis for OEHHA’s unit risk factor (URF). Cal/EPA’s analysis (OEHHA 1999, 2002) resulted in a range of URFs for DPM, 1.3×10^{-4} to $2.4 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$, with a “reasonable estimate” recommended by the SRP of $3.4 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$. That URF translates to a CSF of $1.1 (\text{mg}/\text{kg})^{-1}$.

At approximately the time the OEHHA diesel exhaust risk assessment was finalized, the Diesel Epidemiology Expert Panel was formed by the Health Effects Institute (HEI) – a group that was jointly funded by the United States Environmental Protection Agency (USEPA) and by industry (HEI 1999). One of the specific goals of this Panel was to evaluate the Garshick et al. (1988)

data and to determine its suitability for quantitative risk assessment. Relying in part on the findings of the HEI Panel as well as on an independent analysis of the Garshick et al. (1988) data by Crump et al. (1991), the USEPA concluded that the existing epidemiological data on diesel exhaust were not adequate to support a quantitative assessment of the relationship between exposure and effect. As a consequence of this determination, the USEPA opted not to develop or otherwise identify a CSF or URF for diesel exhaust (USEPA 2002; 2004). This conclusion does not affect the USEPA's classification of diesel exhaust as a probable human carcinogen, but rather, only addresses the adequacy of available data to quantify the relationship between exposure and cancer in humans.

The limitations of the Garshick et al. (1988) data as identified by the HEI Panel (1999), Crump (1991), and the USEPA (2002, 2004) included: inadequate information on exposure to diesel exhaust (i.e., assigning who was exposed and who was not exposed); lack of knowledge of when workers first began working with diesel equipment; and lack of information on smoking and other lifestyle correlates of lung cancer risk. Of particular note, and a fact acknowledged by Garshick in a follow-up publication, is that lung cancer risks among the exposed cohort decreased with increasing length of exposure – the opposite trend from what is expected for a carcinogen. The results of a subsequent study (Garshick et al. 2004), in which the study cohort were followed for a longer period of time, found the same trend (Garshick et al. 2004). This suggests that the original observation of a negative correlation between exposure and lung cancer risk was not an artifact attributable to a truncated follow-up period. Nonetheless, OEHHA has retained its original recommendation for the URF for diesel exhaust of $3.4 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$. Those values are recommended for use in risk assessments conducted to support Proposition 65, the California Environmental Quality Act (CEQA), and various air toxics programs in California. Consistent with this usage, cancer risks in the BWHRA Tool associated with exposure to DPM are calculated based on the CSF derived from OEHHA's URF for DPM.

2 Uncertainties Associated with Health Risk Assessment

There is inherent uncertainty in all health risk assessments, with the source(s) of that uncertainty dependent on the specific assumptions and models used to estimate risk (Council on Environmental Quality [CEQ] 1989).

In accordance with recommendations for an uncertainty analysis described in CEQ (1989) and the National Research Council (NRC 1994), the key uncertainties and critical assumptions associated with the health risk estimation of the BWHRA Tool are described below. The uncertainties associated with air dispersion modeling used in the BWHRA Tool are discussed in Appendix B.

2.1 Uncertainty in the Carcinogenicity of DPM

Although there is general agreement among key US and European regulatory agencies (e.g., the World Health Organization [WHO] 1996) that DPM is a likely human carcinogen, there is considerable uncertainty in the nature of the relationship between DPM exposure and the likelihood of developing cancer. That uncertainty stems in part from a “general lack of understanding” of the mechanism(s) by which DPM elicits toxicity in humans (USEPA 2002). Additionally, it is not understood whether health effects linked to diesel emissions from older diesel engines are relevant to current emission profiles and their effects (USEPA 2002). There are also specific and significant questions regarding the appropriateness of the epidemiologic data used by OEHHA (1998a) to develop the CSF for DPM. Each of these factors, alone or in combination, have the potential to significantly affect the dose-response relationship – and thus the DPM CSF – and as a consequence, the level of risk attributed to DPM exposure. To illustrate the magnitude of potential uncertainty in risk estimates of DPM, it is informative to consider risk levels for DPM calculated using the component-based methodology contained in the USEPA’s *Guidelines for the Health Risk Assessment of Chemical Mixtures* (USEPA 1986) and the subsequent *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures* (USEPA 2000). Distinct from the approach taken in the BWHRA Tool, this methodology involves the identification of key toxicologically-significant components of a mixture, and the estimation of risk attributable to each component. Estimates of total risk are developed by assuming additivity of risk from all component carcinogens. Although the approach contained in these USEPA (1986, 2000) guidance documents is typically recommended for relatively simple mixtures with approximately a dozen or fewer components (USEPA 2000), use of this methodology may be appropriate when information is lacking on the health effects of a mixture. Risk assessments of DPM performed using this component type of approach have calculated health risks that were one to two orders of magnitude lower than the risk estimated using OEHHA’s CSF developed from that value (Muller 2002; ENVIRON 2006). Since completion of these analyses, both the USEPA and OEHHA have identified naphthalene, a DPM component, as a carcinogen. Had Muller (2002) and ENVIRON (2006) included naphthalene in the cancer risk calculated using the components-based approach, the difference in estimated risks between that method and that of the OEHHA CSF would likely decrease.

2.2 Uncertainty in the Role of DPM in Health Effects from Exposure to Particulate Matter Pollution

The evidence that links particulate matter (PM) to adverse health effects is substantial; reports have consistently demonstrated a correlation between long-term exposure to either PM₁₀ or PM_{2.5} (PM with aerodynamic diameters of 10 or 2.5 microns or less, respectively) to non-cancer adverse health effects (see review by Pope and Dockery 2006). Documented health effects from chronic exposure to PM include premature mortality (Pope et al. 1995; Krewski et al. 2000; Laden et al. 2006), respiratory disease (Abbey et al. 1995), and impaired lung development in children (Gauderman et al. 2007). A recent review and analysis of PM health effects (ARB 2008a) cited evidence that premature mortality is associated with chronic exposure to PM_{2.5} levels as low as 5 µg/m³, and the World Health Organization (2005) has concluded that adverse health effects from PM_{2.5} can occur from chronic exposure to 3-5 µg/m³ - levels that are at (or just above) background for the US and Europe.

Determining the contribution of DPM to these effects requires identifying the extent to which health effects attributable to PM₁₀ or PM_{2.5} are due to the DPM fraction of PM. This question is the source of significant controversy and uncertainty, due in large part to the fact that there is no currently-available method to measure and attribute DPM's contribution to the PM fractions in ambient air. DPM is emitted from the combustion of diesel fuel by on-road and off-road vehicles and equipment, becoming a component of ambient PM; however, estimates of DPM as a percentage of the PM inventory vary widely. The primary component of DPM, elemental carbon (EC) (USEPA 2002), is often measured as a surrogate for DPM. However, EC is also a combustion product of gasoline-fueled engines, barbeques, fuel wood, and other lesser sources, making it a highly inaccurate surrogate. While there have been efforts to identify specific and quantifiable indicators of DPM as a component of PM, these efforts have not yielded definitive results. Consequently, while DPM emissions contribute to PM levels, and likely contribute to health effects other than cancer, the uncertainties in current estimation methods of these effects (e.g., ARB 2008b) remain substantial.

2.3 Uncertainties in Exposure Assumptions

Consistent with OEHHA and South Coast Air Quality Management District (SCAQMD) guidance (OEHHA 2003; SCAQMD 2005), individual cancer risks were estimated assuming that residents at the receptor points spend 70 years at one location. Use of the 70-year exposure duration in risk assessments is intended to produce a hypothetical estimate of risk that does not underestimate actual risks and that can be viewed as an upper-bound estimate. To illustrate the conservative nature of the 70-year assumption, it is worth noting that the USEPA has estimated that 50% of the U.S. population lives in the same residence for only nine years, while only 10% remain in the same house for 30 years (USEPA 1997). Adults, moreover, spend only 68-73% of their total daily time at home (USEPA 1997), rather than the 100% assumed in the BWHRA Tool. In addition, due to potential filtration provided by building envelopes and ventilation systems, indoor DPM concentrations resulting from Ports operations are likely to be lower than the outdoor concentrations assumed in this analysis (OEHHA 2003). Accordingly,

the actual risks to hypothetical residential receptors are likely to be significantly lower than those calculated in this assessment.

2.4 Uncertainties in Population-weighted Average Risk

The population weighted risk calculations were based on 2000 census data that was applied to both 2005 and 2020. Although this assumption is likely to be reasonably accurate for the 2005 calculations of population-weighted average risk, it introduces uncertainty into the 2020 risk estimates. Notwithstanding that fact, predicting 2020 populations within the modeling domain would have likely introduced greater uncertainty into risk estimates, although the magnitude of that uncertainty cannot be readily quantified.

An additional component of uncertainty in the population-weighted average risk calculations is attributable to the fact that census tracts were divided in order to approximate the population of the receptor grid used to calculate population-weighted risk for each receptor location. This approach likely does not reflect actual population distributions, nor does it address potential changes in population distribution over time.

2.5 Summary

The risks calculated in the BWHRA Tool were estimated using a series of conservative assumptions regarding exposure concentrations, magnitude and duration of exposure, and carcinogenic potency of DPM. These assumptions, applied in a manner consistent with current guidance (OEHHA 2003; ARB 2003), tend to produce upper-bound estimates of risk, ensuring that these values do not underestimate the actual risks posed by DPM emissions from the ports. It is important to note that the risks calculated in the BWHRA Tool do not necessarily represent the actual risks experienced by populations in the modeling domain. By using standardized conservative assumptions in a risk assessment, the USEPA (1989) has noted that:

“These values [risk estimates] are upper-bound estimates of excess cancer risk potentially arising from lifetime exposure to the chemical in question. A number of assumptions have been made in the derivation of these values, many of which are likely to overestimate exposure and toxicity. The actual incidence of cancer is likely to be lower than these estimates and may be zero.”

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