

RESPONSES TO COMMENTS SUBMITTED BY THE AMERICAN TRUCKING ASSOCIATIONS, INC. ("ATA")

I. Method of Prioritization

Comment 1: In determining which substances “may cause infants and children to be especially susceptible to illness,” the Children's Environmental Health Protection Act sets forth the following criteria for OEHHA to consider:

- (A) Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.
- (B) Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.
- (C) The effects on infants and children of exposure to toxic air contaminants and other substances that have a common mechanism of toxicity.
- (D) The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.¹

These criteria necessarily focus upon the differential sensitivity of infants and children to known air pollutants.

OEHHA developed its own criteria for prioritizing the list of TACs and determining which chemicals have disproportionate impacts upon infants and children. OEHHA's criteria do not place as much emphasis upon the differential sensitivity of infants and children, resulting in a prioritization process that is flawed.

Response: OEHHA disagrees with the comment that we did not utilize the criteria in the statute. In fact, these criteria are embedded in those we spelled out in the document. The law requires OEHHA to evaluate the extent of public exposure in developing the list.

¹ California Health and Safety Code § 39660(c).

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Hence, ambient concentration data as well as emissions inventory information from the Air Resources Board programs were part of the prioritization process. As was explained in the introductory section of OEHHA’s document, the criteria for prioritization of the TACs included evidence for differential effects on infants and children wherever such evidence is available. Health protective values based on adult toxicity were only one of several inputs into the process: unfortunately for some TACs these are the only specific data available.

Comment 2: OEHHA began with the entire list of TACs and ranked them according to their toxicity and extent of air emissions in the state. While the 200 TACs may be a reasonable starting point, for purposes of the Children’s Environmental Health Protection Act, OEHHA should have focused upon those chemicals that have a unique impact upon infants and children.² Without such a focus, the exercise of prioritizing chemicals based upon toxicity and extent of air emissions is merely a repeat of the original TAC listing process.

Response: The statute requires OEHHA to develop a list of up to 5 TACs that may cause infants and children to be especially susceptible to illness. It does not exclude any TACs from consideration. In addition, the law requires OEHHA to evaluate the extent of public exposure. Thus, the use of ambient concentration data in the initial prioritization step fulfills one of the requirements of the law. The focus upon those chemicals that have a unique impact upon infants and children is precisely the intent, and the result, of the process used based on the criteria outlined in our draft document.

Comment 3: In determining which TACs pose the most risk to children in California, OEHHA first focused upon the ratio of chronic Reference Exposure Levels (“RELs”)³ to

² We note that the listing of diesel exhaust as a toxic air contaminant in the State of California is currently the subject of litigation. This litigation questions whether the classification of diesel exhaust as a toxic air contaminant is supported adequately by scientific evidence. *See Apodaca v. California Air Resources Board*, (Superior Court Case No. 00CECG10832).

³ A Chronic Reference Exposure Level is an airborne concentration at or below which adverse non-cancer health impacts would not be anticipated. *See Proposed Listing at 3.*

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act ambient air concentration. This provided OEHHA with a ranking of chemicals with the highest non-cancer toxicity combined with the highest exposure potential.⁴ OEHHA also created a ratio for the carcinogens by multiplying the cancer unit risk factor⁵ by the ambient air concentration and then ranking the carcinogens.⁶ These two rankings were then "combined" to produce a chemical ranking based on existing health criteria and ambient air concentration data. Because the method for combining these two lists and the assumptions used by OEHHA for comparing relative risks between the two groups were not described in the Proposed Listing, we are unable to provide informed comments as to OEHHA's ranking methodology.

Response: OEHHA provided an explanation of the prioritization process used in the introductory section of the document, which was intended to provide a sufficient understanding of the procedure. All the exposure estimates, chronic RELs (draft or final), and cancer potencies were obtained from identified published sources, mostly available on the OEHHA and ARB Web sites. The calculation methods used were simple arithmetical ratios which for the noncancer endpoints and products of Unit Risk Factor times ambient concentration for carcinogenic endpoints. However, since several commenters have requested additional details on the values used by OEHHA, the final version of the document will be amended to include additional information to clarify this process.

Comment 4: Using the processes described in the preceding paragraph combined with OEHHA's knowledge of certain toxicological endpoints of the chemicals, OEHHA

⁴ Because OEHHA did not reproduce its work with respect to the creation of these ratios, it is impossible for ATA to offer meaningful comment upon this aspect of OEHHA's prioritization process.

⁵ A Cancer Unit Risk Factor describes the additional risk of cancer associated with inhaling air containing one microgram of a specified carcinogen per cubic meter. *See* Proposed Listing at 3.

⁶ Again, because OEHHA did not reproduce its work with respect to the creation of these ratios, it is impossible for ATA to offer meaningful comment upon this aspect of OEHHA's prioritization process. For instance, we are unsure of the ambient concentrations used for the diesel exhaust and therefore are unable to comment meaningfully upon the correctness of OEHHA's exposure estimates. We note, however, that diesel exhaust represents only a small fraction of ambient particulate matter.

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act narrowed the TAC list from 200 chemicals down to 34 chemicals.⁷ At this stage, OEHHA had not analyzed whether infants and children are more susceptible to adverse health effects from these chemicals. In other words, OEHHA excluded more than 160 chemicals from in depth review, without first determining whether any of these excluded chemicals have a unique impact upon infants and children. The exclusion of these chemicals in the absence of any meaningful analysis renders OEHHA's prioritization process deficient for purposes of implementing the Children's Environmental Health Protection Act.

After narrowing the list of TACs down to 34 chemicals, OEHHA began focused literature reviews in an effort to determine whether infants or children may be more sensitive to those chemicals than adults. OEHHA created the following criteria to help it further narrow the list of TACs:

1. Any evidence indicating that infants and children may be more susceptible to the toxicological effects associated with that TAC than adults. The strength of this evidence was weighted heavily in this initial selection of eleven TACs that disproportionately impact children.
2. The nature and severity of the effect(s), especially irreversible effects.
3. Any evidence indicating that based on current risk assessment methodology, the existing health criteria may not be adequately protective of infants and children.
4. Any potential difference in susceptibility of infants and children relative to adults to carcinogenesis based on known information or plausible mechanisms.

⁷ OEHHA also considered emissions data in picking the TACs for focused literature reviews; however, OEHHA does not explain how this data was used in determining whether to include or exclude a particular TAC for further review.

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5. Extent of exposure and/or the magnitude of risk estimated to occur at concentrations typical of California urban ambient air, and any indication that infants and children may be more heavily exposed to materials contaminated by airborne particles (e.g., housedust).⁸

The focused literature reviews and application of the criteria listed above, led to a further narrowing of the TAC list and resulted in 11 potential candidates for the initial listing of up to five TACs that disproportionately impact children. OEHHA, however, provided no information on either its methodology for selecting the 11 chemical candidates or the outcome of its literature reviews. The only clue OEHHA provided as to the relative importance of various endpoints it examined is a brief statement that its “decision was heavily influenced by the toxicity of the compounds and less so by the estimated exposures to the compounds.”⁹ As such, ATA is unable to comment on the OEHHA prioritization process, which narrowed a field of over 200 TACs that may cause infants and children to be especially susceptible to illness down to 11 TACs.

Response: The description of the process in the introductory section of OEHHA's document, explains that general population exposure and toxicity were properly considered as part of the available data, but were not the exclusive input into the prioritization. In particular, these general data were used in the early stages of screening to determine which compounds might be relevant, in terms of endpoint and/or exposures in California, for further, more detailed consideration. The statute specifically requires consideration of exposure. Thus, ambient concentration data and emissions inventory data were used in the prioritization process. In some cases, we had no evidence from either measurements in air or emissions inventories that indicated exposures to the general public. Thus, many chemicals were eliminated from further consideration on this basis alone. It should be noted that the list will be updated and as new information on

⁸ Proposed Listing at 5.

⁹ Proposed listing at 4.

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act exposure (and toxicity) becomes available, some of these chemicals may increase in priority.

The use of general toxicity and exposure data was necessitated by the fact that for many of the less well-studied TACs data specific to infants and children or in immature experimental animals were not available. In addition to this prioritization using general toxicity data, OEHHA specifically considered evidence of differential toxicity to infants and children in the few cases where these data were available. Having assembled the general toxicological data, OEHHA was specifically interested in toxicological endpoints which might reasonably be expected to have differential impacts on children. (Further explanation of the use of general toxicity and exposure data in the initial stages of prioritization is given in the response to comments 1 - 4 from the Chemical Industry Council of California, on pages 1-3 of those responses.)

II. Diesel Exhaust Analysis

Introductory comment: OEHHA included diesel exhaust among the 11 TACs that were investigated as potential candidates for the initial listing of chemicals that disproportionately impact children. OEHHA sets forth two arguments for including diesel exhaust: (1) diesel exhaust disproportionately exacerbates asthma in children and (2) diesel exhaust contains polycyclic aromatic hydrocarbons (PAHs), which may disproportionately impact children. We respond to each of these arguments below. In addition, we discuss the difficulty in distinguishing between diesel exhaust and ambient particulate matter, the difference between occupational exposure and ambient exposure, and some of the more recent regulatory initiatives and their impact upon exposure to diesel exhaust.

Response: See below for OEHHA's specific responses. The commenter should take note of the fact that the prioritization phase currently presented is a hazard identification process, where evidence of current exposure and (principally) toxicological information were considered. Questions relating to specific sources of emissions, and trends in exposures over time, were not addressed as part of this process. We defer to the

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California Air Resources Board regarding the need for additional Air Toxic Control Measures, which they will address later.

Comment 1: Diesel Exhaust Has Not Been Shown to Exacerbate Respiratory Illness in Infants or Children.

Some studies have reported that diesel exhaust can exacerbate already-existing respiratory disorders such as asthma. However, the effect reported is an acute, reversible irritability and inflammation of the airways, usually presenting as increased phlegm and cough. Studies do not consistently show a chronic effect on pulmonary function or lung disease from diesel exhaust.¹⁰ Studies have not shown that diesel exhaust is a cause of non-cancerous respiratory disease. Though most of the studies represent exposure in adults, many represent occupational exposures that are likely at a higher dose for a longer duration than ambient air levels responsible for exposure in children.¹¹ Currently, there are relatively few studies on the effects of diesel exhaust exposure in children.

¹⁰ Attfield, MD (11/6-7/78) The Effect of Exposure to Silica and Diesel Exhaust in Underground Metal and Nonmetal Miners. *Industrial hygiene for Mining and Tunneling- Proceeding of an ACGIH Topical Symposium*; Gamble, John and Jones, Williams (1983) Respiratory Effects of Diesel Exhaust in Salt Miners. *Am. Rev. Respir. Dis.*; 129:389-94; Jorgensen H and Svensson A (1970) Studies on Pulmonary Function and Respiratory Tract Symptoms of Workers in an Iron Ore Mine Where Diesel Trucks are Used Underground. *J. Occ. Med.* 12 (9): 348-54; Ames R, Reger R and Hall D. (1984) Chronic Respiratory Effects of Exposure to Diesel Emissions in Coal Mines. *Arch. of Environ. Health* 39(6): 389-93; Ulfvarson U, Alexandersson R, Dahlqvist M, Ekholm U, Bergstrom B. (1991) Pulmonary Function in Workers Exposed to Diesel Exhausts: The effect of control measures. *Am. J. of Indus. Med.* 19:283-89; Ulfvarson U, Alexandersson R, Aringer L, Svensson E, Hedenstierna G, Hogstedt C, Holmberg B, Rosen G, Sors, M. (1987) Effects of exposure to vehicle exhaust on health Scand. *J. Work Environ. Health* 13: 505-12; Rudell B, Ledin MC, Hammarstrom U, Stjernberg N, Lundback B, Sandstrom T. (1996) Effects on symptoms and lung function in humans experimentally exposed to diesel exhaust. *Occ. and Environ. Med.* 53:658-62; Battigelli M. Effects of Diesel Exhaust (1965) *Arch. Environ. Health* 10:165-67; Salvi S, Blomberg A, Rudell B, Kelly F, Sandstrom T, Holgate S, Frew, A. (1999) Acute Inflammatory Responses in the Airways and Peripheral Blood After Short-Term Exposure to Diesel Exhaust in Health Human Volunteers. *Am. J. Respir. Crit. Care Med* 159:702-9.

¹¹ Two cross-sectional studies of children also failed to find an association between exposure, measured by traffic density, and prevalence of asthma. See English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L (1999) Examining Associations between Childhood Asthma and Traffic Flow Using a Geographic Information System *Environ Health Perspectives* 107:9; Wjst M, Reitmeir P, Dold S, Wulff A, Nicolai T, Freifrau von Loeffelholz-Colberg E, von Mutius E (1993) Road traffic and adverse effects on respiratory health in children. *BMJ* 307:596-600.

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OEHHA bases its conclusion that diesel exhaust places children at risk for exacerbation of respiratory illnesses on studies that find an association between traffic density and increased respiratory symptoms in children. These studies are in contrast to studies of adults that find no effect of diesel exhaust on adult pulmonary function. OEHHA echoes the comment in the paper by Oosterlee, et al. (1996) that the lack of effects in adults relative to the children may indicate a difference in susceptibility to ambient traffic related air pollution between children and adults.¹² However, the inconsistency is more likely due to differences in study design.

In the studies of children, the investigators used traffic density as a surrogate measure of exposure. Such an indirect measure does not accurately measure exposure to diesel exhaust, and fails to control for confounding variables. Additionally, several studies measured both traffic density and respiratory symptoms by self-reported questionnaires.¹³ For example, OEHHA cites Ciccone et al. (1998) as “clearly” establishing the association of adverse respiratory health impacts with heavy vehicular (diesel powered) traffic.¹⁴ Traffic density in the study was measured by self-report. Additionally, the investigators found an absence, overall, of significant associations between reported traffic density and respiratory symptoms. They went on, however, to analyze the relationship between reported frequency of “lorry” traffic (described as heavy vehicular traffic) and found a positive relationship with respiratory symptoms of children in *metropolitan* areas, but not in *urban* areas. A reasonable conclusion would be that some other confounding variable

¹² Office of Environmental Health Hazard Assessment California Protection Agency “Prioritization of Toxic Air Contaminants Under the Children's Environment Health Protection Act,” Appendix Diesel Exhaust Particulate Matter, pg. 10, March 2001 (*citing* Oosterlee, A, Drijver, M, Lebre E, Brunekreef B (1996). Chronic respiratory symptoms in children and adults living along streets with high traffic density. *Occ. Environ. Med.*, 53:241-47).

¹³ *See e.g.* Oosterlee, et al. *supra*; Duhme H, Weiland S, Keil U, Kraemer B, Schmid M, Stender M, Chambless L (1996) The Association between Self-Reported Symptoms of Asthma and Allergic Rhinitis and Self-Reported Traffic Density on Street of Residence in Adolescents *Epidemiology* 7(6): 578-82; Weiland S, Mundt K, Ruckmann A, Keil U. (1994) Self-Reported Wheezing and Allergic Rhinitis in Children and Traffic Density on Street of Residence. *AEP* 4(3):243-7.

¹⁴ OEHHA, Appendix Diesel Exhaust Particulate Matter, pg. 9 (*citing* Ciccone G, Forastiere F, Agabiti N, Biggeri A, Bisanti L, Chellini E, Corbo G, Dell'Orco V, Dalmasso P, Fatur Volante T, Galassi C, Piffer S, Renzoni E, Rusconi F, Sistini P, Biegi G and the SIDRIA collaborative group (1998) Road traffic and adverse respiratory effects in children” *Occup. Environ. Med.*, 55:771-78).

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exists in the metropolitan area that is not present in the urban area. A simple, yet plausible, explanation is provided in the Duhme, et al. (1996) study, also cited by OEHHA. Acknowledging a weakness of their study (similarly designed to measure the association between self-reported traffic density and respiratory symptoms), the investigators note:

Furthermore, the observed association may not be a direct, but rather an indirect, effect of motor vehicle emissions. Those who live near busy roads may more often close their windows to keep the disturbing motor vehicle emissions like exhaust fumes and traffic noise out of their home. This practice may, as a consequence, change the indoor climate and may have an effect on indoor allergen concentration.¹⁵

The Ciccone study, in fact, comments that information about traffic of heavy vehicles was available for 26,234 children living in houses with windows facing the street from a sample size of 39,275.

In contrast to these studies, those finding no or transient effects of diesel exhaust on adult respiratory function measured either results of direct exposure on volunteers under experimental laboratory conditions, or the prevalence or incidence in occupations with known high exposure, such as miners exposed underground to exhaust from diesel equipment.¹⁶ Such study designs inherently provide a more reliable measure of actual exposure to diesel exhaust and control for confounding variables. Additionally, the adult studies use objective pulmonary function testing as an indicator of lung function as well as subjective reports of symptoms. This, again, provides a more reliable measure of the effect of diesel exhaust on respiratory function.

¹⁵ Duhme, et al. at 581.

¹⁶ Ref. Note 12, *supra*.

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The available studies that measure the effect on children of diesel exhaust exposure are limited, both in number and in design. The studies that measure the non-cancerous effects of diesel exhaust exposure in adults, however, consistently show no effect or no acute transient effect on respiratory status. Additionally, the ambient particulate matter levels that may contribute to respiratory illnesses are comprised of various emission sources, of which diesel exhaust is a small fraction. Current regulations have reduced, and will continue to reduce diesel exhaust emissions.

OEHHA mentions that diesel exhaust is a candidate to move to Tier 1 of the priority list of Toxic Air Contaminants. However, given the above points, and in the absence of scientific evidence showing diesel exhaust may place children at an increased risk for respiratory illness, there is no acceptable basis upon which the OEHHA can decide to list diesel exhaust as one of the five highest priority air toxins.

Response: Studies cited in the diesel exhaust prioritization document (Diaz-Sanchez *et al.*, 1994, 1996, 1997; Terada *et al.*, 1997, Takenaka *et al.*, 1995) indicate that the exacerbation of asthma by diesel exhaust is due specifically to a modulation of the immune system, and not because of a general irritant effect. Since the prevalence of asthma is much higher among children than among adults (CDC, 1996a,b), exacerbation of asthma by diesel exhaust will put more children at higher risk of adverse health effects than adults. Thus, on a population-wide basis, children are disproportionately impacted by airborne substances which exacerbate asthma. In addition, as noted in the draft OEHHA document, the smaller airway diameter of children predisposes to more severe sequelae of asthma attacks. Indeed, hospitalization rates of children 0 to 4 years of age for asthma are much higher than any other age grouping (CDC, 1996a).

The commenters note at the outset that: "Some studies have reported that diesel exhaust can exacerbate already-existing respiratory disorders such as asthma. However, the effect reported is an acute, reversible irritability and inflammation of the airways, usually presenting as increased phlegm and cough. " In this context it should be noted that in the U.S. asthma is the most common chronic disease among children. Exacerbations of

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asthma result in significant school absenteeism, and in more serious cases, emergency room visits and hospitalizations. Thus, even without considering potential chronic effects of exposure to diesel, effects of acute exposure among children represent an enormous public health burden.

It is true that there are challenging issues involved in estimating the direct impact of diesel exhaust on the respiratory health of children. Most of the studies cited in this comment are cross-sectional in nature and use indirect estimates of diesel exposure. There are several studies, however, that use a superior prospective cohort design to examine this issue. In these studies, subjects report daily respiratory symptoms over a period of several months or more and both exposure and response are classified more precisely. The studies have used either PM₁₀ or PM_{2.5} as their exposure metric and find strong and consistent associations between particulate matter and several adverse outcomes in children including increases in lower respiratory symptoms and asthma exacerbation, and decreases in lung function (US EPA, 1996; Pope, 2000). In their policy review of the PM studies, the U.S. EPA cites children as a sensitive subpopulation stating:

Increased community morbidity, decreased lung function, and increased respiratory symptoms have been reported to be associated with PM exposure in children, both as a general group and in individuals with respiratory illness. Children have the potential to be inherently more susceptible to the effects of PM as they show a greater incidence of respiratory and other illness, suggesting decreased immunological protection, and higher deposition of particles than adults...Infants in particular have been hypothesized to be a sensitive subpopulation for PM effects as exposure may increase the incidence or severity of acute respiratory infection including bronchitis, bronchiolitis, and pneumonia (page V-35).

Several additional studies, which appeared since the report by US EPA (1996) or were not considered therein, have reported associations between PM and severe outcomes in infants. Analyses involving both cross-sectional and time-series study designs have

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act demonstrated associations between ambient PM and neonatal or infant mortality , low birthweight or higher rates of prematurity. For example, in Rio de Janeiro (Penna and Duchigie, 1991), the Czech Republic (Bobak and Leon, 1992), Sao Paulo (Pereira *et al.*, 1998) and the United States (Woodruff *et al.*, 1997) cross-sectional associations have been reported between measures of PM and neo-natal or infant mortality. Daily time-series studies have reported associations between changes in PM and infant or child mortality in Mexico City (Loomis *et al.*, 1999) and Bangkok (Ostro *et al.*, 1999). Finally, Ritz *et al.* (2000) reported associations between PM and both low birth weight and premature delivery.

Therefore, there are many studies that indicate that exposure to fine particulate matter, which includes diesel exhaust, are associated with a wide range of adverse health outcomes in children. We agree with the comment that diesel is only one of several sources of PM that may be responsible for these outcomes, but as a general contributor to PM, diesel particulate is likely to have significant effects on children.

Comment 2: Diesel exhaust's polycyclic aromatic hydrocarbon content does not warrant the listing of diesel exhaust as potentially having a disproportionate impact upon children.

OEHHA has listed PAHs in Tier I and will designate those substances as causing infants and children to be especially susceptible to illness. OEHHA states that diesel exhaust contains PAHs and for this reason it has been included in Tier 2. PAHs already are proposed for designation as a Tier 1 contaminant. As such, listing diesel exhaust based upon its PAH content is merely duplicative. Furthermore, no information has been provided as to the percentage of PAHs contained in diesel exhaust. Without this information, it is difficult to determine whether the PAH content of diesel exhaust rises to the level of regulatory concern.

Response: OEHHA did not prioritize diesel exhaust only on the basis of its PAH content, but found this to be a supporting factor among several. The adverse health effects of

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act diesel exhaust are unlikely to be only due to PAHs and particulates; diesel exhaust contains a variety of toxicants, including (but not limited to) the carcinogens benzene, 1,3-butadiene and formaldehyde. Additionally, as noted in the OEHHA draft report, diesel exhaust has been demonstrated to specifically exacerbate asthma and allergic rhinitis. Therefore, the listing of diesel exhaust is not duplicative of the PAH listing.

Appendix A of part A (Exposure Assessment) of the diesel exhaust TAC identification document (CARB, 1998) discusses diesel exhaust composition, and lists ten pages of identified diesel exhaust components, many of which are PAHs and related compounds such as nitroarenes. Therefore, we disagree with the comment "it is difficult to determine whether the PAH content of diesel exhaust rises to the level of regulatory concern".

Comment 3: The discussion of diesel exhaust contained a number of inconsistent propositions, which OEHHA appears to have overlooked. In addition, OEHHA relies upon research that has been supplemented and modified by newer research. For example, in section II, entitled "Overview", the authors report that in June, 1993 the United States EPA "determined" an inhalation Reference Concentration (RfC) of 5ug/m³ for chronic non-cancer effects of diesel effects. As the authors of this document presumably are aware, the EPA assessment report was reviewed on five occasions following the June, 1993 draft and was found to be deficient by the Clean Air Scientific Advisory Committee (CASAC). The most recent Health Assessment Report dated July 25, 2000, which CASAC reviewed and reported on in December, 2000, set a RfC of 14 µg/m³ for non-cancer health effects of diesel exhaust based upon criticisms of the analysis in earlier drafts.¹⁷ Thus, the current level proposed by the EPA Science Advisory Board is almost three times higher than that adopted by OEHHA.

Response: OEHHA is familiar with the various iterations of the US EPA's evaluation process for diesel exhaust. As noted in the description of methodology used, the selection of reference health protective values for prioritization purposes used adopted

¹⁷ EPA draft Health Assessment Document for Diesel Exhaust, EPA 600/8-90/057E, 7/25/00 at 1-4.

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California values in preference to draft or adopted numbers from any other source. OEHHA has no mandate (or intention) to revise currently adopted health protective values at this stage of the process. The validity and applicability of the diesel exhaust cancer unit risk factor (URF) and noncancer chronic Reference Exposure Level (REL) have been thoroughly documented in the diesel exhaust Toxic Air Contaminant (TAC) document and will not be discussed further in these responses.

Comment 4: An example of the selection of inconsistent scientific data appears in the section II "Overview" subsection entitled "Summary of Potential Differential Effects." The authors first argue that studies have demonstrated that diesel exhaust may exacerbate allergic rhinitis and asthma in children. In the next paragraph the authors express concern with the fact that PAHs have been demonstrated in animal experimentation to have immunosuppressive effects. Yet, this finding is inconsistent with the discussion in section III entitled "Summary of Key Human Studies" depicting evidence that diesel exhaust enhances allergic responses to pollen and other allergens.

Response: The reports that immunosuppressive effects as a result of PAH exposure have been reported in animals exposed in utero does not necessarily contradict the data indicating that diesel exhaust specifically exacerbates asthma and allergic rhinitis. Not all asthmatic children will have been exposed to a dose of PAHs in utero sufficient to cause immune suppression. Additionally, many of the animal immunosuppressive effects noted after in utero PAH exposure are the result of altered T-cell lymphocyte production; the exacerbation of asthma and allergic rhinitis appears to be due to changes in cytokine production and IgE response, which are functions of B-cell response. Finally, it would be entirely possible that an asthmatic child with a compromised immune system because of in utero PAH exposure could still react to a sufficient challenge by diesel exhaust.

Comment 5: In both of the section II "Overview" subsections, "Summary of Potential for Differential Effects" and "Principle Sources of Exposure", the authors refer to diesel exhaust as a component of PM₁₀. Elsewhere in this section, diesel exhaust is described as, "an important source of air pollutants..." Diesel exhaust, however, is only one

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act component of PM₁₀. Additionally, recent EPA national assessment data reports diesel exhaust (from both on-road and non-road sources) contributed only 1.3% of total emitted PM₁₀, and 4.9% of total emitted PM_{2.5}.¹⁸ Thus, it is misleading to characterize diesel exhaust as a significant source of PM₁₀.

OEHHA also states that diesel exhaust particulate contains PAH..."and thus diesel exhaust particulate impacts infants and children and is a candidate for moving into Tier 1." OEHHA goes on to list diesel exhaust particulate as "a major source of ambient PAHs". In fact, there is currently no measure of the contribution of diesel exhaust to ambient PAH. However, it is known that PAH compounds comprise only 1% or less of airborne particulate matter mass.¹⁹ The fractional contribution of diesel exhaust to ambient PAH, therefore, should not be classified as major. Furthermore, the OEHHA recognizes that PM₁₀ emissions from on-road diesel exhaust is expected to decline even further as a result of standards and regulations adopted by CARB.²⁰

Response: As stated in the diesel exhaust SB25 prioritization data summary document, based on the ARB 1990 emissions inventory, approximately 58,000 tons of diesel exhaust PM₁₀ from all sources are emitted into California air each year (ARB, 1997). The statewide population-weighted average diesel exhaust PM concentration is estimated to be 3.2 µg/m³. An ARB study to determine the PM₁₀ concentrations due to the primary emissions from diesel engine exhaust near the Long Beach Freeway indicated that near-roadway concentrations of diesel exhaust PM₁₀ may be as high as 8 µg/m³ above ambient concentrations for one 24-hour period (ARB, 1996). This is notable in light of the fact

¹⁸ See National Air Pollutant Emission Trends, 1900-1998, p. 3-13, EPA-454/R-00-002, March 2000.

¹⁹ Benner BA, Gordon GE, Wise SA (1989) Mobile sources of atmospheric PAH: a roadway tunnel study. *Environ Sci Technol* 23:1311-19; Lowenthal DH, Zielinska B, Chow JC, Watson JG, Gautam M, Ferguson DH, Neuroth GR, Stevens KD (1994) Characterization of heavy-duty diesel vehicle emissions. *Atmos Environ* 28:731-43; Rogge WF, Heldemann LM, Mazurek MA (1993) Sources of fine organic aerosol. 2. Noncatalyst and catalyst-equipped automobiles and HD diesel trucks. *Environ Sci Technol* 27:636-51. (As cited in EPA 600/8-90/057E, 7/25/00).

²⁰ Office of Environmental Health Hazard Assessment California Protection Agency "Prioritization of Toxic Air Contaminants Under the Children's Environment Health Protection Act" Appendix Diesel Exhaust Particulate Matter, pg. 4, (March 2001).

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children’s Environmental Health Protection Act that the chronic Reference Exposure Level (REL) for diesel exhaust is $5 \mu\text{g}/\text{m}^3$. After considering these facts along with the cancer and noncancer health effects data for diesel exhaust, it is clear that diesel exhaust is in fact an important source of air pollutants.

The commentors are correct in stating “there is currently no (*direct*) measure of the contribution of diesel exhaust to ambient PAH”. However, it should be noted that ARB staff have estimated that emissions from diesel exhaust contribute about 3 and 8 percent of the total California PM_{10} and $\text{PM}_{2.5}$ inventories, respectively (ARB, 1997). The exposure assessment section of the diesel TAC identification document also observes:

“These total exposures estimates are believed to underestimate, to an unknown extent, Californians’ actual exposures to diesel exhaust particles. This is because insufficient data are available for concentrations inside vehicles and along roadways to allow such near-source, elevated exposures to be estimated for the population”. (ARB, 1998, page A-57)

Zeilinska (1991, cited by ARB, 1998) found that motor vehicle exhaust was the second highest contributor to wintertime PM_{10} , and that diesel-fueled motor vehicle exhaust was responsible for at least half of the motor vehicle derived PM_{10} . Also, as described by ARB (1998), PAHs are generally associated with the particles composed of elemental carbon (EC), rather than the mineral particles of geological or atmospheric origin. ARB found that

“... diesel emissions were responsible for approximately 67 percent of the fine EC mass in the Los Angeles atmosphere, and that the exhaust particles averaged about 64 percent EC.” (ARB, 1998, page A-47)

The entry for diesel exhaust in Table 1 of the TAC prioritization document will be modified to clarify the fact that diesel exhaust is a source of ambient PAHs and also of PM, particularly the finer and EC-containing fractions.

Comment 6: In section III entitled, “Summary of Key Human Studies,” the report argues that evidence for causation of lung cancer as determined by OEHHA in its 1998 report entitled “TAC Health Risk Assessment for Diesel Exhaust” was found “convincing using criteria for causal inference.” However, when the data is stripped of regulatory default

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children’s Environmental Health Protection Act assumptions, the epidemiological data falls far short of providing a “convincing” case of causality between diesel exhaust and human lung cancer.

Epidemiological studies finding an association between diesel exhaust exposure and cancer involve occupational exposure, where diesel exhaust exposure is likely higher than ambient air exposure.²¹ In fact, EPA acknowledged in its Draft Health Assessment Document for Diesel Exhaust that human evidence, plagued by confounding effects of smoking and the lack of actual diesel exhaust exposure data for the workers, is not sufficient to conclude that diesel exhaust exposure is causally associated with lung cancer.²²

Response: OEHHA, the Scientific Review Panel on Air Toxics, the California Air Resources Board, the US EPA, and various expert bodies including the International Agency for Research on Cancer, have concluded that diesel exhaust (either determined as the particulate fraction, or considering the whole exhaust) is probably carcinogenic to humans. OEHHA has also concluded that the epidemiology data are consistent with the conclusion that exposure to diesel exhaust is causally associated with lung cancer. The carcinogenicity of diesel exhaust was debated at length during the identification phase of the toxic air contaminant program. In addition, the comment ignores the fact that U.S.EPA considers diesel exhaust a “likely human carcinogen”. There are many studies of the association of occupational exposure to diesel exhaust that corrected for the effects of cigarette smoking and still found significantly elevated risk of lung cancer. A recent paper (Larkin *et al.*, 2000; Garshick is a co-author) reanalyzed the data in the Garshick 1988 cohort study and found elevated lung cancer risk in diesel exhaust exposed workers after correction for smoking. The meta-analysis conducted by OEHHA and recently published (Lipsett *et al.*, 1999) showed that for smoking-adjusted studies, the combined relative risk was 1.43 (1.31-1.57).

²¹ See Section III.B.5, *infra*.

²² EPA 600/8-90/057E, 7/25/00 at pg. 1-4.

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Comment 7: OEHHA references a number of studies with respect to non-cancer allergic and respiratory effects of diesel exhaust. Concerns are based upon several studies that have looked at the degree to which diesel exhaust acts as an immuno-adjuvant. The most recent study cited by OEHHA is that by Diaz-Sanchez, 2000. In this study, the investigators demonstrated that diesel exhaust has no effect in inducing histamine release when exposed to a murine model without co-administration of allergen. Moreover, the authors determined that the carbon black particle when stripped of the organic chemicals on it has no immuno-adjuvant effect. Thus, the authors conclude that although diesel exhaust can act as an adjuvant to enhance the allergic response in an organism that is pre-sensitized to an allergen when co-administered with an allergen, that it is the soluble organic chemicals on diesel exhaust that mediate the reaction.

If, however, diesel exhaust, because of its surface organic chemicals, can act as an immuno adjuvant, it clearly cannot also act simultaneously as an immuno suppressant as suggested by the authors of the Proposed Listing document with regard to the animal experimentation on PAH. In other words, one cannot have this biological effect both ways. PAH at levels that are consistent with that adsorbed to carbon core particles cannot both in the same species act as an immuno-suppressant while at the same time acting as an immuno adjuvant to enhance allergic response. In this instance, the authors of the Proposed Listing document have made unsound assumptions or conclusions based upon a variety of animal studies of which the dose quantities used bear little resemblance to actual environmental exposure of PAH from diesel exhaust.

OEHHA has not investigated whether the diesel exhaust particles bind or release PAHs in the presence of bodily fluids. Prior to listing diesel exhaust as a contaminant that may disproportionately impact the health of children based upon its PAH content, it is important to know whether PAHs are absorbed into the body. It is theoretically possible for PAHs to remain bound to diesel exhaust particulates, thereby having no adverse health effect once absorbed into the body. OEHHA cites to no studies attempting to describe how the body metabolizes diesel exhaust. In the absence of such evidence, the

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mere presence of PAHs in diesel exhaust is insufficient to form a conclusion of adverse health effects.

The chemical makeup of diesel exhaust likely will vary depending upon the type of engine, the quality of the fuel used, and any add-on emission control devices. These variables likely would impact the PAH content of diesel exhaust actually released to the environment. New regulations, such as EPA's 2007 rule for heavy-duty diesel engines, will result in a dramatic reduction of particulate matter.²³ As such, the amount of PAHs present in today's diesel exhaust may be significantly less than the PAH content emitted from older diesel engines.

Response: The reports that immunosuppressive effects as a result of PAH exposure have been reported in animals exposed in utero does not necessarily contradict the data indicating that diesel exhaust specifically exacerbates asthma and allergic rhinitis. Not all asthmatic children will have been exposed to a dose of PAHs in utero sufficient to cause immune suppression. Additionally, many of the animal immunosuppressive effects noted after in utero PAH exposure are the result of altered T-cell lymphocyte production; the exacerbation of asthma and allergic rhinitis appears to be due to changes in cytokine production and IgE response, which are functions of B-cell response. Finally, it would be entirely possible that an asthmatic child with a compromised immune system because of in utero PAH exposure could still react to a sufficient challenge by diesel exhaust.

The bioavailability of PAHs contained in diesel exhaust was thoroughly reviewed in the diesel exhaust TAC document (OEHHA, 1998). The studies reviewed clearly indicated that the PAHs in diesel exhaust were bioavailable upon inhalation exposure. Additionally, a recent study by Sato *et al.* (2000) indicated that rats exposed to diesel exhaust by inhalation demonstrated increased mutations in a reporter gene and covalent DNA adducts, additional evidence suggesting PAH bioavailability.

²³ See Section III.B.4, *infra*.

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Future diesel engine emissions controls could theoretically reduce the PAH content of diesel exhaust. However, in light of the lack of empirical data on this subject, the possibility of reduced PAH content in the exhaust of diesel engines produced in the future remains speculative. In addition, an ARB-funded study by the College of Engineering, Center for Environmental Research and Technology (CE-CERT) at the University of California, Riverside [*Evaluation of Factors that Affect Diesel Exhaust Toxicity* (draft final report), ARB Contract No. 94-312 (CE-CERT, 1998)] found that genotoxicity of diesel exhaust from engines using California reformulated diesel fuel in bacterial mutation assays was not significantly different from that of engines using pre-1993 fuel. This issue was debated thoroughly during the identification phase of the TAC process. The reader is referred to the TAC identification documentation: *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant*, Appendix III, Part C (ARB, 1998b) for more information on this issue.

Comment 8: Distinguishing Diesel Exhaust from Particulate Matter

Diesel exhaust particulate is ubiquitous and almost impossible to distinguish from ambient particulate matter. OEHHA states that diesel exhaust contributes to ambient particulate matter and then notes that ambient particulate matter has been shown to exacerbate asthma and has been associated with low birth weight and decreased lung function. Based upon this evidence, OEHHA concludes that diesel exhaust, as a component of particulate matter, is a substance of concern. However, OEHHA cites no scientific evidence directly linking diesel exhaust to these adverse effects or demonstrating that exposure to diesel exhaust triggers an adverse health effect different from exposure to particulate matter generally.²⁴ Moreover, OEHHA’s characterization of diesel exhaust as a major source of airborne particulate matter is contrary to fact.

OEHHA describes diesel exhaust as a “major source of” and “significant contributor to” ambient particulate levels. Diesel exhaust, however, is only one component of airborne particulate matter. Indeed, EPA’s recent report entitled “*National Air Pollutant Emission*

²⁴ See Section III.B.1, *supra*.

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children’s Environmental Health Protection Act *Trends, 1900-1998,*” indicates that diesel exhaust (from both on-road and non-road sources) comprises only 1.3% of total emitted PM₁₀, and 4.9% of total emitted PM_{2.5}.²⁵ These levels do not represent a “major source” of ambient particulate matter. In addition, OEHHA recognizes that the PM₁₀ emissions from on-road diesel exhaust is expected to decline even further as a result of standards and regulations adopted by the California Air Resources Board and other environmental regulatory agencies.²⁶ Thus, it is disingenuous to argue that diesel exhaust constitutes a significant source of airborne particulate matter.

Response: As noted in the response to Comment 3, ARB staff have estimated that emissions from diesel exhaust contribute about 3 and 8 percent of the total California PM₁₀ and PM_{2.5} inventories, respectively (ARB, 1997). In urban environments, especially near roadways, the contribution is higher. Diesel exhaust is a major contributor to fine elemental carbon containing particles. As noted previously, the entry for diesel exhaust in Table 1 of the TAC prioritization document will be modified to clarify the fact that diesel exhaust is a source of ambient PM, particularly the finer (PM_{2.5}) and EC-containing fractions.

Diesel exhaust particulate demonstrates immune system effects resulting in adverse health outcomes (e.g. exacerbation of asthma and allergic rhinitis) (Diaz-Sanchez et al., 2000) that are not shared by other model particulates such as carbon black and crystalline silica (van Zijverden et al., 2000). This suggests that diesel exhaust particulate has additional unique toxicological properties above and beyond the cardiopulmonary toxic effects of PM₁₀.

Comment 9: Regulation of Airborne Particulate Matter Obviates the Need for Separate Regulation of Diesel Exhaust.

²⁵ See National Air Pollutant Emission Trends, 1900-1998, p. 3-13, EPA-454/R-00-002, March 2000.

²⁶ Office of Environmental Health Hazard Assessment California Protection Agency “Prioritization of Toxic Air Contaminants Under the Children’s Environment Health Protection Act,” Appendix Diesel Exhaust Particulate Matter, pg. 4 (March 2001).

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One of the principal purposes of the Children's Environmental Health Protection Act is to identify toxic air contaminants that may cause infants and children to be especially susceptible to illness and then to ensure that these harmful contaminants are regulated appropriately.

Within two years of the establishment of the list required pursuant to subdivision (a), the state board shall review and, as appropriate, revise any control measures adopted for the toxic air contaminants identified on the list, to reduce exposure to those toxic air contaminants . . . to protect public health, and particularly infants and children.²⁷

Thus, it is reasonable to expect that substances identified by the OEHHA under the Children's Environmental Health Protection Act may be subject to additional regulation. In the case of diesel exhaust, however, further reductions of air toxics are not technologically feasible and existing regulations already press the envelope on the amount of particulate that can be removed from diesel exhaust emissions.

Many of the programs designed to reduce ambient concentrations of the criteria pollutants also aid in reducing air toxics. For example, U.S. EPA's final rule regulating emissions from heavy-duty diesel engines will result in a 90 percent further reduction in particulate matter from today's standards.²⁸ This rule will be effective in 2007. EPA itself, in the context of its recently finalized rule on the control of emissions of hazardous air pollutants from mobile sources, acknowledges that the 2007 model year standards

²⁷ California Health and Safety Code, Section 39669.5.

²⁸ See 66 *Federal Register* 5002 (January 18, 2001). The 2007 emission standards for heavy-duty engines reduce the allowable level of particulate matter from 0.7 g/bhp-hr to 0.01 g/bhp-hr, a 98.6% reduction. See 66 *Federal Register* 17231, 17243-44 (March 29, 2001).

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children’s Environmental Health Protection Act represent “the greatest degree of emission reduction achievable through the application of technology that will be available considering costs and other relevant factors.”²⁹

Even if one could completely eliminate diesel exhaust from the ambient air, the impact of particulate matter upon infants and children likely would remain. As such, control of diesel exhaust as a separate air contaminant will not solve the problem of exposure to ambient particulate matter. OEHHA should focus its attention upon air toxics for which additional regulation will result in a tangible health benefit for infants and children.

Response: The Children’s Environmental Health Protection Act (SB 25, Escutia; chaptered 1999) requires OEHHA to evaluate available information on the TACs and develop a list of up to five TACs that “may cause infants and children to be especially susceptible to illness” by July 1, 2001. SB 25 does not mandate that OEHHA be involved in the risk management process. In the case of diesel exhaust, risk management is the responsibility of the ARB. The ARB is fully aware that diesel exhaust particulate is being considered for listing under SB 25. ARB is in the process of evaluating risk management options for diesel engine emissions. As is customary, ARB evaluates both cost considerations and technical feasibility in developing risk management measures.

Comment 10: The Increased Respiration Rate of Children Does Not Correlate to an Increased Health Risk from Exposure to Diesel Exhaust.

One of the critical assumptions underlying OEHHA’s decision to classify diesel exhaust in Tier 2, is that children experience an increased exposure to diesel exhaust compared to adults.

Because children inhale a greater volume of air per unit time and body weight than adults they receive higher doses of airborne contaminants.³⁰

²⁹ 66 *Federal Register* at 17244.

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In estimating the health risk associated with diesel exhaust particulate matter, OEHHA relies heavily on studies based upon human occupational exposure. This is inappropriate for the limited purpose of analyzing the increased risk to infants and children.

Infants and children spend very little, if any, time in an occupational setting such as a coal mine using diesel generators or a construction site laden with heavy duty diesel construction equipment. Accordingly, one would expect that most children receive a smaller dose of diesel exhaust than individuals exposed to diesel exhaust in an occupational setting. As such OEHHA's extrapolation of data from studies that were based upon occupational exposure is inappropriate for measuring the health risk to infants and children. OEHHA's conclusion that increased respiratory rates result in increased exposure may be true for other TACs, but does not apply in the context of exposure to diesel exhaust.

Response: As stated in the response to Comment 2, the validity and applicability of the diesel exhaust cancer unit risk factor (URF) and noncancer chronic Reference Exposure Level (REL) have been thoroughly documented in the diesel exhaust Toxic Air Contaminant (TAC) document part B (OEHHA, 1998); the commenter should refer to this source for further information. High to low-dose risk extrapolation has attendant uncertainties, but is nonetheless a generally accepted part of scientific risk assessment. The extrapolation of exposures in occupational studies to the general public is standard risk assessment practice that accounts for the differences in exposures. In the same environment, infants and children will inhale more contaminants than adults on a body weight basis just by virtue of their higher breathing rates per unit body weight. This is applicable to all toxicants across the board, including diesel exhaust. In fact, there is more, not less, concern for particle phase toxicants because, as noted in our draft document, young children receive higher particle loadings (number of particles

³⁰ Proposed Listing at 11.

Draft Responses to Comments on the March 2001 Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act impacting) per lung surface area than adults as the result of their higher breathing rates. It is, therefore, unclear why the comment states that the fact that children inhale a greater volume of air per unit time and body weight than adults and therefore receive higher doses of airborne contaminants should be inappropriate for use in the determination of potential health risks to infants and children exposed to diesel exhaust.

Comment 11: Conclusion

We agree with OEHHA's conclusion that diesel exhaust does not belong in the Tier 1 group of air contaminants proposed for initial listing under the Children's Environmental Health Protection Act. We do, however, have concerns with OEHHA's prioritization process, which has not been adequately explained and which resulted in OEHHA's decision not to conduct focused literature reviews of over two thirds of the California list of TACs. We also do not agree with OEHHA's characterization of diesel exhaust. Specifically, we do not believe that diesel exhaust has been shown to exacerbate respiratory illness in infants and children.

For the reasons set forth above, we believe that OEHHA should remove diesel exhaust from the Tier 2 list and focus upon those chemicals that may pose an increased health risk to infants and children, as required by the Children's Environmental Health Protection Act.

Response: The prioritization of TACs in Tier 1 and 2 will be finalized following our response to the public comments and following peer review by the state's Scientific Review Panel on Toxic Air Contaminants.