RESPONSES TO COMMENTS SUBMITTED BY INTERNATIONAL TRUCK AND ENGINE CORPORATION (ITEC)

Comment 1A: Diesel Particulate Should Not Be Listed on SB 25.

OEHHA cites to two reasons for the listing of diesel particulate: its contributions to ambient loadings of PM and the existence of PAHs in diesel exhaust.[1] For the reasons described below, neither rationale is a sufficient basis for including diesel particulate on the SB 25 list. Moreover, the goal of the SB 25 list is to provide the most additional protection to children and to address the largest sources of harmful byproducts. If the placement of a TAC on the SB 25 list will not contribute to improved protections for children's health, then that TAC should not even be placed in Tier 2. Yet children will not receive any additional benefit from the listing of diesel particulate on the SB 25 list. Since inclusion of diesel particulate on SB 25 list will *not* advance the goals of the statute, it should not be listed on SB 25.

A. Diesel Particulate's Contribution to PM Loadings Is Not a Sufficient Basis for Including it on the SB 25 List.

OEHHA's first reason for including diesel particulate on the SB 25 list is that diesel exhaust contributes to PM.[2] The fact that diesel particulate contributes to PM loadings does not warrant listing it on the SB 25 list. First, an extensive regulatory scheme already exists that addresses the effects of PM on children's health. (This is contrary to the Children's Environmental Health Protection Act section for listing TACs, which does not encompass criteria pollutants.) Second, the available data indicates that diesel particulate is not the only contributor to PM loading in California. Perhaps more significantly, diesel particulate is not even the primary contributor of PM loading in California. Therefore, International believes that OEHHA has not provided a sufficient basis to support inclusion of diesel particulate on the SB 25 list and OEHHA should remove diesel particulate from this listing.

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[1] *Prioritization* at 8.

[2] *Id.*

Response 1A: The statute requires OEHHA to establish a list of up to five TACs that may cause infants and children to be especially susceptible to illness. The statute does not exempt any TAC from evaluation for listing under SB 25. The fact that PM_{10} is a criteria air pollutant does not exempt diesel exhaust particulate matter from evaluation under the TAC portion of the statute.

Comment 1B.An Extensive Regulatory Scheme That Addresses Children's Health Issues Already Exists for PM

"PM is not a TAC. Rather, PM is a "criteria" pollutant". The comment goes on to describe what a criteria air pollutant is and what a state implementation plan is.

"OEHHA instead should focus on the TACs that may be dangerous to children's health and that are *not* covered by other regulatory schemes. Given that an entirely separate and extensive regulatory scheme already addresses the impacts of PM on children's health, PM – whether from diesel particulate or some other source – simply is not an appropriate focus of the SB 25 list. OEHHA should use SB 25 to fill in the gaps that exist in children's protection, not to be redundant in targeting stricter control measures for TACs that are already being addressed elsewhere.

The comment goes on to state "Specifically, OEHHA, in consultation with ARB, is reviewing PM_{10} standards "to determine whether, based on public health, scientific literature, and exposure pattern data, the standards adequately protect the health of the public, including infants and children, with an adequate margin of safety."[3]

In contrast, the only mention of criteria pollutants in the TAC listing section (§ 39669.5) is when it addresses the assessment of public exposure to TACs. The statute states that "the office shall take into account public exposures to toxic air contaminants, whether by themselves or interacting with other

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[3] OEHHA Staff Report Resulting from the ARB Board Meeting of December 7-8, 2000. Executive Summary and Report. http://www.arb.ca.gov/ch/ceh/airstandards.htm citing CAL. HEALTH AND SAFETY CODE § 39606(d)(1).

toxic air contaminants or criteria pollutants…"[4] The statute also refers OEHHA to subdivision (c) of Section 39660 to consider "the interaction of multiple air pollutants on infants and children, including the interaction between criteria pollutants and toxic air contaminants." In both cases, the emphasis is on the *interaction* of the TAC with criteria pollutants, not the *use* of the criteria pollutant to justify the TAC's listing. The SB 25 list only applies to TACs. PM is not a TAC. Thus, claims about the health effects of PM have no place in deciding which compounds get included on the SB 25 list.

Clearly, PM's effect on children is being addressed elsewhere. Additionally, the statute expressly limits OEHHA's use of criteria pollutants in its evaluation of TACs for listing on SB 25. In attempting to broaden its statutory authority to include a consideration of a criteria pollutant on the SB 25 list, OEHHA has exceeded the authority delegated to them by the California Legislature. To remain within its statutory authority, OEHHA cannot rely on diesel particulate's contributions to PM loadings as a rationale for listing it on SB 25.

Response 1B: The comment is correct in so far as the work required by the statute for criteria air pollutants. PM_{10} was prioritized as the highest priority criteria air pollutant for review due to inadequacy of the standard to protect public health including infants and children. However, listing diesel exhaust particulate is complementary to this process, not duplicative. Diesel exhaust particulate is but one source of fine particulate. In addition, diesel exhaust particulate has unique noncancer effects that are above and beyond the cardiopulmonary toxic effects of PM_{10}. Finally, the inclusion of diesel exhaust particulate in considering the establishment of a list under SB 25 is not solely based on its particulate nature. As noted in the draft document, OEHHA is concerned about enhanced allergenicity which appears to be unique to diesel exhaust particulate and not a general effect of PM10. In addition, the PAH content of diesel exhaust particulate (as well as numerous other air toxics) is cited as another factor that should be considered in establishing the list under SB 25.

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[4] CAL. HEALTH AND SAFETY CODE § 39669.5(a)(1).

Comment 1C: Inclusion of Diesel Particulate on the SB 25 List Will Have No Impact On Ambient Concentrations of PM

Not only has OEHHA chosen the wrong regulatory framework for addressing PM, but the mechanism it has chosen – listing diesel particulate on SB 25 – will be notably ineffectual. One of OEHHA's two primary concerns about the effect of diesel particulate on children's health is its contribution to California PM levels. OEHHA states that "[p]articulate matter 10 microns or smaller (PM_{10}) has been associated in numerous studies with adverse respiratory health effects in children including exacerbation of asthma, bronchitis, cough and wheeze."[5] If OEHHA is concerned about PM_{10}, however, then OEHHA should focus on the largest contributors of PM_{10} in California.

Emission of PM is not an issue that is unique to diesel exhaust. Thousands of other sources also emit particulate matter. Fine particles ("$PM_{2.5}$") are emitted not only from diesel engines, but also from fuel combustion in other motor vehicles (including gasoline and natural gas vehicles), power plants, and industrial facilities, as well as from residential fireplaces and wood stoves.[6] Coarse particles ("PM_{10}") are generally emitted from sources such as vehicles traveling on unpaved roads, materials handling, crushing and grinding operations, and windblown dust.[7]

Additionally, some research suggests that while gasoline and natural gas vehicles may have very low PM mass emission rates, they may emit more nanoparticles (or "ultrafine particulate matter") than diesel engines.[8] This conclusion is supported by independent findings where "large concentrations of nanoparticles were found over Minnesota roadways even in the absence of significant diesel traffic."[9]

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5 *Prioritization*: Appendix B: Diesel at 3.

6 EPA, Office of Air Quality Planning and Standards. *Latest Findings on National Air Quality: 1999 Status and Trends*. p.9 (August 2000), available online at http://www.epa.gov/airtrends.

7 *Id*.

8 Gautam, M., N.N. Clark, and D.W. Lyons, *Particulate Matter and NO_x Emissions From In-Use Heavy-Duty Vehicles*. National Research Center for Alternative Transportation Fuels, Engines and Emissions. West Virginia University. College of Engineering and Mineral Resources.

9 D.B. Kittelson, *Nanoparticle Emissions from Diesel and Spark Ignition Engines. Center for Diesel Research*, Department of Mechanical Engineering, University of Minnesota. Presented at the World Truck Conference (March 5, 2001).

Because nanoparticles have a greater ability to penetrate into the deep lung, many scientists believe that these "ultrafine particles" may have more adverse health effects than either coarse or fine PM.[10] Thus, there appears to be no basis for including diesel exhaust on the SB 25 list based on non-cancer health effects, but not including other contributors to PM.

Not only is the emission of PM an issue shared by many other source categories in California, but diesel particulate is not even the major contributor of PM in California. Indeed, relative to other sources of PM in California, diesel particulate is a relatively minimal contributor. According to the 2001 California Almanac of Emission and Air Quality, statewide PM_{10} emissions from *all sources* were calculated to be 2313 tons/day, annual average for 2000.[11] Therefore, the total annual PM_{10} emissions for California from *all sources* are 844,245 tons/year.[12] On-road diesel vehicles contribute only 18 tons per day out of this total California PM_{10} loading, or 0.8%.[13] "Other Mobile" source PM emissions are 69 tons per day (3.0%). Even assuming that that entire category is diesel, diesel's contribution to total PM_{10} in the State of California would increase to only 3.8%.[14] Compared to area sources (748,615 tons/year), which includes only a minimal contribution from stationary diesel sources and contribute 89% of the California PM, it is arbitrary for OEHHA to not focus its attention on the largest contributor to California PM_{10} – area sources.[15]

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10 *See, e.g.*, Elder, A.C.P., Gelein, R., Finkelstein, J.N., Cox, C. and Oberdorster, G. (2000). "Endotoxin priming affects the lung response to ultrafine particles and ozone in young and old rats." *Inhal. Toxicol.* 12, Supp. 1, 85-98; Ferin, J., Oberdorster, G., and Penny, D.P. (1992). "Pulmonary retention of ultrafine particles in rats." *Am. J. Respir. Cell Mol. Biol.* 6: 535-542.

11 California Air Resources Board ("CARB"). *The 2001 California Almanac of Emissions and Air Quality*. Table 3-4 (2001), available online at http://www.arb.ca.gov/aqd/almanac01/toc01.htm.

12 This assumes a constant daily emission rate year-round.

13 California Air Resources Board. *The 2001 California Almanac of Emissions and Air Quality*. Table 3-4.

14 Since virtually all diesel particulates are due to mobile sources, stationary sources' contribution is not included in this calculation.

15 California Air Resources Board. *The 2001 California Almanac of Emissions and Air Quality*. Table 3-4. In fact, CARB's Risk Reduction Plan estimates that stationary diesel sources in 2000 emitted only 558 tons of PM per year out of a total of 28,000 total tons of PM per year, or 2.0%. California Air Resources Board. *Risk Reduction Plan to Reduce Particulate Matter Emission from Diesel-Fueled Engines and Vehicles.* p.12 (October 2000).

International further notes that OEHHA's quantitative statistics about California PM emissions are outdated and overestimate the effect of diesel PM on California children. OEHHA cites to 1990 data points to estimate that the total amount of diesel PM from *all sources* in California is 58,000 tons per year.[16] The California Air Resources Board ("CARB"), however, cites to 46,400 tons of diesel PM per year for 1990 and 28,000 tons of diesel PM annually in 2000.[17] The impact of diesel particulate on children in California therefore is even less than OEHHA anticipates – yet even OEHHA's estimate would place diesel particulate at a paltry 7% of total state-wide PM emissions.

It is an erroneous strategy to attempt to ensure that children are not disproportionately affected by PM by requiring a reevaluation of diesel particulate. Much more direct means exist for OEHHA to address their concerns with PM's impact on children. For one, PM should be targeted directly so that any control revisions will apply to *all* source categories of PM, not just diesel particulates. Of course, this is exactly what the criteria pollutant program does. Given diesel particulate's relatively small contribution to statewide PM emissions, coupled with the existence of a criteria pollutant program designed specifically to address the concerns OEHHA has articulated about PM, it is arbitrary for OEHHA to include diesel particulate as a candidate for the SB 25 list because it is a contributor to PM.

Response 1C: The listing of diesel exhaust particulate matter under SB25 was not solely because of its contribution to $PM_{10.}$ ARB staff have estimated that emissions from diesel exhaust contribute about 3 and 8 percent of the total California PM_{10} and $PM_{2.5}$ inventories, respectively (ARB, 1997); and it is likely higher in congested urban areas. Diesel exhaust particulate matter demonstrates specific toxic effects (carcinogenicity, chronic pulmonary effects) that were part of the basis of its listing as a TAC. Additionally, 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

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16 *Prioritization*: Appendix B: Diesel at 4.

17 OEHHA also cites that "on-road diesel vehicles contribute approximately 59 percent of California's diesel exhaust." *Prioritization:* Appendix B: Diesel at 4; CARB's own Risk Reduction Plan, however, estimates that "[o]n-road engines account for about 27 percent of the [particulate matter] emissions [from diesel-fueled vehicles and engines]." California Air Resources Board. *Risk Reduction Plan to Reduce Particulate Matter Emission from Diesel-Fueled Engines and Vehicles*. p.12 (October 2000).

shared by other model particulates such as carbon black and crystalline silica (van Zijverden *et al*., 2000). This suggests that diesel exhaust exhibits noncancer health effects that are unique over and above the cardiopulmonary toxic effects of exposure to ambient general particulate matter. 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. In addition the smaller airway of children predisposes to more severe sequelae of asthma attacks. Children in the age group 0 to 4 years are hospitalized for asthma much more frequently than any other age grouping (CDC, 1996a). Since diesel exhaust is a TAC, not a criteria pollutant, these data make it eligible and suitable for listing under SB25.

As regards the comment that "because nanoparticles have a greater ability to penetrate into the deep lung, many scientists believe that ultrafine particles may have more adverse health effects than either coarse or fine PM", the science is incomplete with regard to which fraction is the worst actor. Furthermore, the deposition of particles in the lung is not linear with diameter as the comment seems to imply; it is more complicated. Total deposition in the lung increases as you fall below 10 µm in diameter but then decreases as the very small particles do not impact but are exhaled. Furthermore, deposition varies by size with region of the lung and level of activity. It is not possible to say at this point that the particles from combustion of one fuel type are deposited more or less than particles created during combustion of another fuel type. The particulate size fraction has not even been characterized from different motors burning a variety of fuels.

The comment implies that strategies to reduce PM_{10} are sufficient to cover any concerns about diesel exhaust particulate. Strategies to reduce PM_{10} that may occur following the re-evaluation of PM_{10} ambient air quality standard may be different than those that ARB will seek in the risk management of diesel exhaust emissions. Thus, while there may be overlap, the effort is complementary and not duplicative.

It should also be noted that while SB 25 does specify OEHHA's involvement in the risk assessment portion of the legislative mandate, it does not mandate that OEHHA be involved in the risk management process. Risk management responsibilities under SB25 are the responsibility of the ARB. SB25

requires OEHHA to consider in its health effects assessments and recommendations: (1) exposure patterns among infants and children that result in disproportionately high exposure; (2) special susceptibility of infants and children; (3) effects of simultaneous exposures to compounds with the same mechanism of action; and (4) any interactions of air pollutants. OEHHA was not directed to consider present or potential risk management programs during the prioritization process.

Finally, in the last paragraph, the comment indicates we are "re-evaluating" diesel exhaust particulate. The OEHHA draft document provides information pertinent to the question of whether children are more affected than adults. As such, it is a hazard identification document, not a reassessment of the risk assessment recently conducted for diesel exhaust under the TAC program.

Comment 2: The Existence of Tightly bound PAHs on the Diesel Particulate Carbon Core Does Not Warrant Listing Diesel Particulate on the SB 25 List.

OEHHA's second reason for including diesel particulate on the SB 25 list is that diesel exhaust contains PAHs.[18] The existence of PAHs in the diesel exhaust does not warrant listing diesel particulate on the SB 25 list. First, PAHs are already included in Tier 1 of the SB 25 list. The additional listing of diesel particulate will not add any further benefit, as sources of PAHs can already be targeted for risk management activities through the inclusion of PAHs on the SB 25 list. Perhaps more importantly, the available data amply demonstrates that diesel particulate is not a significant source of PAHs. The U.S. EPA itself has concluded that while PAHs may be an important risk driver in some regions of the country, emissions from mobile sources – including diesel – make only a negligible contribution to that risk. Moreover, the PAHs in diesel exhaust are tightly bound to the carbon core, and thus have limited bioavailability. Therefore, International believes that OEHHA has failed to provide a sufficient basis to support the inclusion of diesel particulate on the SB 25 list and OEHHA should remove diesel particulate from this listing.

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[18] *Prioritization* at 8.

1. PAHs Are Already Listed on the SB 25

Any disproportionate effects on children from PAHs are also adequately addressed without adding diesel particulate to the SB 25 list. Namely, OEHHA has also identified PAHs as a candidate for inclusion in Tier 1 of the SB 25 list.[19] This is a much more understandable position than listing diesel particulate since all the reasons that are indirect links for diesel are direct links for PAHs themselves. From a policy standpoint, it just makes more sense to target the harmful pollutant, instead of one of many source categories. Significantly, OEHHA provides no distinctive reason why diesel particulates should also be targeted as a source category *in addition* to their targeting of PAHs generally. There is no marginal benefit from listing diesel particulates on the SB 25 list as well.

2. PAHs in Diesel Exhaust Are Tightly Bound To The Carbon Core and Have Limited Bioavailability

"OEHHA expresses concern about the PAHs associated with diesel exhaust particles. However, PAHs can be recovered from diesel exhaust particles only after treatment with heated hydrocarbon solvents. Such organic extracts of diesel-exhaust particles are not relevant to human inhalation exposures of whole diesel exhaust, because the chemicals in the extracts are not bioavailable."…

For the *in vitro* studies that report mutagenicity, diesel exhaust particles were subjected to extensive extraction procedures in organic solvents in order to remove the adsorbed organic materials, which were then concentrated and used in the various assays.[20] However, experiments with whole diesel exhaust show minimal dissolution of diesel-particulate organic compounds from diesel exhaust particles

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[19] *Id*.

[20] Austin, A.C., Claxton, L.D., and Lewtas, J. 1985. Mutagenicity of the fractionated organic emissions from diesel, cigarette smoke condensate, coke oven, and roofing tar in the Ames assay. *Environ. Mutagen.* 7:471–487; Lewtas, J., Bradow, R.L., Jungers, R.H., Harris, B.D., Zweidinger, R.B., Cushing K.M., Gill, B.E., and Albert R.E. 1981. Mutagenic and carcinogenic potency of extracts of diesel and related environmental emissions: Study design, sample generation, collection, and preparation. *Environ. Intl.* 5:383–387; Shirname-More, L. 1995. Genotoxicity of diesel emissions, Part I: Mutagenicity and other genetic effects. In *Diesel Exhaust: A Critical Analysis of Emissions, Exposure, and Health Effects*. pp. 221–242. Health Effects Institute, 955 Massachusetts Avenue, Cambridge, MA.

by physiological fluids.[21] That is, lung-tissue fluids are ineffective in releasing adsorbed PAHs from diesel exhaust particles. Thus, when diesel particulate is inhaled and deposited onto lung surfaces, the adsorbed organic material is not bioavailable to the target cells, that is, lung epithelial cells. This critical issue of PAH bioavailability is not mentioned by OEHHA.

Furthermore, even if all the organic material absorbed to diesel exhaust particulate were totally bioavailable (which it is not), there is an insufficient quantity of mutagenic material to contribute significantly to any tumorigenicity. For example, Pepelko and Chen have estimated that the concentration of the best known carcinogenic PAH (benzo[a]pyrene) is no more than 0.1 µg/mg of diesel particulate matter, and these authors have concluded that it is unlikely that such low concentrations could be responsible for tumorigenic responses.[22] Studies with rats, the only species that shows tumorigenicity at highly elevated diesel-exhaust concentrations, indicate that the organic compounds on diesel exhaust are not active in the lung-tumor induction in rats.[23]

Some authors have attempted to ascribe urinary mutagens to diesel exhaust. Kanoh *et al.* (1993) conducted a short-term rat study to assess the use of urinary 1-hydroxypyrene as a marker of PAH exposure.[24] For the calculation of inhaled PAH, the authors used the airborne concentration of diesel

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21 Brooks, A.L., Wolff, R.K., Royer, R.E., Clark, C.R., Sanchez, A., and McClellan, R.O. (1981). Deposition and biological availability of diesel particles and their associated mutagenic chemicals. *Environ Intl* 5:263-267; King, L.C., Kohan, M.J., Austin, A.C., Claxton, L.D., and Huising, J.L. 1981. Evaluation of the release of mutagens from diesel particles in the presence of physiological fluids. *Environ Mutagen* 3:109-121; Li, A.P., 1981. Antagonistic effects of animal sera, lung and liver cytosols and sulfhydryl compounds on the cytotoxicity of diesel exhaust particle extracts. *Toxicol. Appl. Pharmacol*. 57:55-62; Siak, J.S., Chan, T.L., and Lee, P.S. 1981. Diesel particulate extracts in bacterial test systems. *Environ Intl* 5:243-248; Vostal, J.J. 1983. Bioavailability and biotransformation of the mutagenic component of particulate emissions present in motor exhaust samples. *Environ. Health Perspect*., 47:269-281.

22 Pepelko, W.E., and Chen, C. (1993). Quantitative assessment of cancer risk from exposure to diesel engine emissions. *Regul Toxicol Pharmacol* 17:52-65.

23 Reviewed by: Rosenkranz, H.S. 1993. Revisiting the role of mutagenesis in the induction of lung cancers on rats by diesel emissions. *Mut Res* 303:91-95; Health Effects Institute. 1995. *Diesel Exhaust: Critical Analysis of Emissions, Exposure, and Health Effects*. Cambridge, MA: Health Effects Institute; Watson, A.Y., and Valberg, P.A. 1996. Particle-induced lung tumors in rats: Evidence for species specificity in mechanisms. *Inhal Toxicol* 8: 227-257; Valberg, P.A., and A.Y. Watson. 1999. Comparative mutagenic dose of ambient diesel-engine exhaust. *Inhalation Toxicology* 11:215-228.

24 Kanoh, T., Fukuda, M., Onozuka, H., Kinouchi, T., and Ohnishi, Y. 1993. Urinary 1-hydroxypyrene as a marker of exposure to polycyclic aromatic hydrocarbons in environment. *Environ. Res*. 62:230-241.

particulate and not the deposition fraction. The actual deposited amount of pyrene was only about 3 to 5 ng. For the calculation of ingested PAH, the authors implied that the two groups of rats consumed the same amount of food, but it does not appear that the authors measured food consumption. Even if all the pyrene adsorbed to diesel particles were bioavailable, diesel exhaust-derived pyrene only accounted for about 2-3 % of the daily pyrene dose, and consequently, urinary 1-hydroxypyrene is very insensitive as an indicator of diesel-exhaust PAH bioavailability.

Studies with workers having potential exposure to diesel exhaust have reported on DNA adduct levels in blood and urine samples. Hemminki *et al*. (1994), Hou *et al*. (1995), and Nielsen *et al*. (1996) investigated DNA adduct levels in peripheral blood cells from healthy, non- smoking males.[25] The subjects were employed as bus garage workers, bus mechanics, or truck terminal workers in Sweden. However, information on diesel exhaust exposure was not available for these studies and dermal exposure to diesel fuel and lubricating oil also occurred. These are extremely important caveats, which severely limit implicating diesel-engine exhaust as the source of DNA adducts.

Schenker *et al*. (1992) showed that urinary mutagenicity was not correlated with exposure to diesel exhaust in 87 railroad workers.[26] The authors obtained measurements of RSP, using personal monitors, and corrected these values for exposure to environmental tobacco smoke. These negative results support an absence of PAH bioavailability.

Scheepers *et al*. (1994) measured the concentration of urinary 1-aminopyrene in 3 diesel train-engine mechanics and 2 office clerks.[27] Although some differences in urinary concentrations were reported, it

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[25] Hemminki, K., Soderling, J., Ericson, P., Norbeck, H.E., and Segerback, D. 1994. DNA adducts among personnel servicing and loading diesel vehicles. *Carcinogen*. 15:767-769; Hou, S., Lambert, B., and Hemminki, K. 1995. Relationship between hprt mutant frequency, aromatic DNA adducts and genotypes for GSTM1 and NAT2 in bus maintenance workers. *Carcinogen*. 16:1913-1917; Nielsen, P.S., Andreassen, A., Farmer, P.B., Ovrebo, S., and Autrup, H. 1996. Biomonitoring of diesel exhaust-exposed workers: DNA and hemoglobin adducts and urinary 1-hydroxypyrene as markers of exposure. *Tox. Lett*. 86:27-37.

[26] Schenker, M.B. Kado, N.Y., Hammond, S.K., Samuels, S.J., Woskie, S.R., and Smith, T.J. 1992. Urinary mutagenic activity in workers exposed to diesel exhaust. *Environ. Res*. 57:133- 148.

[27] Scheepers, P.T.J., Thuis, H.J.T.M., Martins, M.H.J., and Bos, R.P. 1994. Assessment of occupational exposure to diesel exhaust. The use of an immunoassay for the determination of urinary metabolites of nitroarenes and polycyclic aromatic hydrocarbons. *Tox. Lett*. 72:191-198.

was not possible to assign those differences to diesel-exhaust exposure because: (1) There were no differences between the two groups of employees when the authors compared daily excretion levels on a single-day basis. (2) A significant portion (approximately 70%) of the airborne particulate matter was not derived from diesel exhaust. (3) Total suspended particulate matter and respirable suspended particulate matter concentrations did not correlate well with the time and frequency of engine test runs. (4) In the mechanics, the highest 24-hour average of urinary 1-aminopyrene occurred on Monday, when airborne levels of 1-nitropyrene were not detectable. (5) And finally, the authors provided no information on other sources of nitro-PAHs exposure which mechanics and clerks may have encountered both at and away from work. The authors cautioned that this was a preliminary study, and should be treated as such when drawing conclusions about bioavailability.

Qu *et al*. (1997) measured DNA adducts in miners from two diesel-equipped mines and attempted to evaluate differences between pre- and post- occupational exposure differences.[28] Approximately 50% of the workers were active smokers or ex-smokers. In the first mine, linear regression modeling showed a positive association between adduct and smoking status (smokers had 37% higher adducts than non-smokers)and a negative association of adduct formation with the time on job. No significant association was found between adducts and smoking or adducts and job categories in the second mine.

In summary, OEHHA's use of the mere presence of PAHs in organic-solvent extracts of diesel exhaust as persuasive evidence of diesel exhaust carcinogenicity is inconsistent with our current understanding of diesel-exhaust PAH bioavailability.

Response 2A: The issue of bioavailability of PAH on diesel exhaust was thoroughly discussed during the identification phase for diesel exhaust as a toxic air contaminant. For further information and responses to the same comments brought up during the identification phase, the reader should consult the documents: *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Part B.*

Health Effects of Diesel Exhaust (section 5.1.2.6) and *Part C Responses to Comments.*(Volume 3-1, pp.OEHHA 73-86, and elsewhere) (OEHHA, 1998; ARB, 1998b) . 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 showing PAH bioavailability.

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 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 discussed in the response to Comment 1, diesel exhaust also specifically exacerbates asthma and allergic rhinitis. 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. Therefore, the listing of diesel exhaust is not duplicative of the PAH listing.

Comment 2B: Diesel Particulates Are Not a Significant Source of PAHs

In addition to the limited bioavailability of PAHs in diesel particulate and the inclusion of PAHs generally on the SB 25, diesel particulate is not even a major contributor of PAHs in California. If OEHHA wants to use the less efficient source-specific method of dealing with PAHs, it should at least focus on the largest sources of PAHs in California. On the contrary, diesel particulate is not even a significant source of PAHs in California. Thus, OEHHA's concern over PAHs is similarly misdirected towards diesel particulate.

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[28] Qu, S.-X., Leigh, J., Koelmeyer, H., Stacey, N.H. 1997. DNA adducts in coal miners: association with exposures to diesel engine emissions. *Biomarkers*. 2:95-102.

PAHs are a ubiquitous product of combustion from common sources such as motor vehicles and other gas-burning engines, wood-burning stoves and furnaces, cigarette smoke, industrial smoke or soot, and charcoal-broiled foods.[29] Hazardous waste sites can also be a concentrated source of PAHs on a local scale. Examples of such sites are abandoned wood-treatment plants (sources of creosote) and former manufacturer-gas sites (sources of coal tar).[30] Additionally, natural sources of PAH include volcanoes, forest fires, crude oil, and shale oil.[31] "

According to the Agency for Toxic Substances and Disease Registry ("ATSDR"), stationary sources account for approximately 80% of total annual PAH emissions, with the remainder coming from mobile sources (both gasoline and diesel-fueled).[32] The largest single source of PAHs is the burning of wood in homes.[33] ATSDR reports that approximately 36% of total PAHs released into the United States annually come from residential heating, 36% from open burning, 21% from mobile sources (including gasoline and diesel vehicles), 6% from industrial processes, and 1% each from incineration and power generation.[34] Active and passive inhalation of the compounds in tobacco smoke also is a significant source of individual PAH exposure.[35]

Significantly, as part of its National Air Toxics Assessment, U.S. EPA has recently evaluated the risks from exposure to PAHs and concluded that PAHs from mobile sources – including diesel exhaust – make only a negligible contribution to that risk. The National Air Toxics Assessment Program is a

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29 Research Triangle Institute. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons*. (August 1995). Prepared for the U.S. Department of Health and Human Services: Agency for Toxic Substances and Disease Registry, citing IARC, *IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans*. Vol. 32: Polynuclear aromatic compounds: Part 1. Chemical, environmental and experimental data. Lyons, France: World Health Organization, International Agency for Research on Cancer, 155-161, 225-237 (1983).

30 *Toxicological Profile* at 229.

31 *Toxicological Profile* citing Hazardous Substances Data Bank. National Library of Medicine, National Toxicology Program (via TOXNET), Bethesda, MD. (December 1994).

32 *Toxicological Profile* at 232.

33 *Id.* at 229.

34 *Id.* at 232.

35 *Id.* at 230.

combination of activities designed to provide risk-based information to the Agency to assist in development of the next phase of HAP regulations (residual risk, urban air toxics, etc.).[36] It includes five core components: (1) emissions inventory; (2) toxics monitoring; (3) toxics modeling; (4) risk assessment; and (5) research to improve assessment tools. EPA's *Draft National-Scale Air Toxics Assessment for 1996* (January 2001) ("*Assessment*") uses emissions inventory, monitoring and modeling data to determine potential inhalation exposures and health risks associated with selected HAPs. EPA conducted these risk assessments by comparing estimated individual chemical exposures at the census tract level against the health benchmarks for those chemicals. Because of uncertainties in the data, these census tract-level risks were then aggregated to identify average risks at a national level, although the assessment also provides State-level risk information and county-level exposure information (from which risks can be estimated).[37]

EPA has determined that the overall risk of cancer in California from exposure to 7-PAHs is only approximately one in ten million.[38] EPA's analysis further broke down exposure concentrations of 7-PAHs into statewide source sectors. EPA's analysis demonstrates that diesel particulate is not the major source of 7-PAHs in California. In California, the largest source of 7-PAHs are "area" sources, although onroad sources (both gasoline and diesel) constituted a relatively significant source category in

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36 *See*, EPA, *Draft National-Scale Air Toxics Assessment for 1996*, (Jan. 2001).

37 Although this Assessment can provide perspective regarding the relative cancer risks associated with PAHs, some limitations of this study must first be acknowledged. First, significant emission reductions have occurred since 1996, the year that the data for this study was based upon. Second, risk estimates were based on exposure concentrations for the median individual within each census tract. Third, risk estimates included only inhalation exposure. Despite these limitations, the results are still illustrative of the minimal impact of diesel particulates on PAH. EPA, *Draft National-Scale Air Toxics Assessment for 1996*, p.8-9 (Jan. 2001).

38 EPA, *Draft National-Scale Air Toxics Assessment for 1996*, 1996 Modeled Exposure Concentrations POM (7-PAH) – Statewide Concentration Distribution Estimates. (Jan. 2001). "7-PAH" consists of the 7 best-characterized carcinogenic PAHs: chrysene, indeno[1,2,3-cd]pyrene, benzo[b]fluorathene, benzo[k]fluoranthene, dibenz[a,h]anthracene, benzo[a]anthracene and benzo[a]pyrene. *See* EPA, *Draft National-Scale Air Toxics Assessment for 1996*, Appendix H (Jan. 2001). Relevant pages from EPA's *Draft National-Scale Air Toxics Assessment for 1996* are provided at Attachment 1. Additionally, at the request of CARB, researchers also conducted ambient monitoring of several particle-bound PAH and PAH-derivatives in Riverside, California. The study found that "the aggregate risk from ambient exposures to these PAHs and their derivatives is calculated to be *less* than one in a million." Krieger, RK; Wright, JN.

terms of emissions.[39] Significantly, however, non-road sources were found to be only a trivial contributor to total PAHs.[40] Since a higher proportion of non-road vehicles are dieselized (as compared to on-road sources), these data suggest that gasoline vehicles may be contributing more PAHs than diesel vehicles in the on-road sector.

Even more significantly, when EPA evaluated regional risk in its *Assessment*, the risk varied dramatically, depending on the source category. While on-road sources contributed a relatively significant amount of PAH emissions, their contribution to the *risk* from PAHs was trivial. For 7-PAHs emitted from *major* sources, approximately one million people have a one in a million risk of cancer and 100,000 people have a ten in one million risk.[41] This risk characterization drops off steeply when the cancer risk is based on *mobile* sources. In fact, for both PAH emitted from on-road mobile sources and non-road mobile sources, risks are less than one in one million for every region of the United States.[42] (That is, no one exceeds a one in a million risk of cancer from mobile source 7-PAHs anywhere in the country.) On the contrary, the PAH risk increases dramatically when based on *area* source emissions. For 7-PAHs emitted from area sources, there are approximately 3 million people who have a one in one million chance of cancer risk.[43]

The *Assessment* also provided a national risk characterization. According to their findings, 75% of the entire population of the United States have only a one in ten million chance of cancer from *major*

"Ambient monitoring of selected PAHs in California." Air & Waste Management Association 90. Annual Meeting, Air & Waste Management Association, Pittsburgh, PA, 1997 (emphasis added).

39 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Modeled Exposure Concentrations POM (7-PAH) – Statewide Source Sector Contribution Estimates. (Jan. 2001).

40 *Id*.

41 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Population whose 1996 exposure exceeded set cancer risk levels based on major sources (Jan. 2001)

42 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Population whose 1996 exposure exceeded set cancer risk levels based on on-road mobile sources (Jan. 2001) and EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Population whose 1996 exposure exceeded set cancer risk levels based on non-road mobile sources (Jan. 2001).

43 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Population whose 1996 exposure exceeded set cancer risk levels based on area sources. (Jan. 2001).

sources of 7-PAH.[44] Even for the 99th percentile exposure category, the risk was substantially lower than one in one million. However, the risks of cancer are again substantially higher for those individuals exposed to 7-PAHs from *area* sources. Approximately 99 percent of the U.S. population has a one in a million lifetime cancer risk from area source emissions.[45] Alternatively, 50 percent of the U.S. population has only a one in a *hundred* million lifetime risk of cancer from 7-PAHs from on-road mobile sources.[46] Indeed, 90 percent of the population has only a one in a *hundred* million lifetime cancer risk from non-road sources of 7-PAHs.[47] In other words, if OEHHA is concerned about the risks of PAHs, listing diesel exhaust is an ineffective means of addressing that risk.

Therefore, because OEHHA can not cite to a rationale for listing diesel particulate that is unique to diesel particulate, and because their two main concerns related to diesel particulate are addressed through other regulatory schemes, OEHHA should not include diesel particulate on the SB 25 list.

Response 2B: In this comment, it is stated that diesel particulates are not a significant source of PAHs. Data on ambient PAH source apportionment in California are scant; however, the commenter also cites US EPA as stating that in California, "the largest source of 7-PAHs are "area" sources, although on road sources (both gasoline and diesel) constituted a relatively significant source category in terms of emissions". This suggests that diesel exhaust is in fact a significant source of ambient PAHs in California.

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 diesel exhaust are unlikely to be only due to PAHs and particulates; diesel exhaust contains a variety of toxicants, including (but not limited to)

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44 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Distribution of lifetime cancer risk for the US population, based on 1996 exposure to major sources. (Jan. 2001).

45 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Distribution of lifetime cancer risk for the US population, based on 1996 exposure to area sources. (Jan. 2001).

46 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Distribution of lifetime cancer risk for the US population, based on 1996 exposure to on-road sources. (Jan. 2001).

47 EPA, *Draft National-Scale Air Toxics Assessment for 1996,* 1996 Risk Characterization: Distribution of lifetime cancer risk for the US population, based on 1996 exposure to non-road sources. (Jan. 2001).

the carcinogens benzene, 1,3-butadiene and formaldehyde. Additionally, as discussed in the response to Comment 1, diesel exhaust also specifically exacerbates asthma and allergic rhinitis. Therefore, the listing of diesel exhaust is not duplicative of the PAH listing. As noted in our draft document, 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. In addition, hospitalization rate data for asthma show that children 0 to 4 years of age are hospitalized much more frequently than any other age grouping for asthma. Thus, on a population-wide basis, children are more impacted by asthma and substances that exacerbate asthma than adults.

Comment 3: Listing Diesel Particulate on the SB 25 List Will Not Provide any Additional Protections For Children's Health.

"The Children's Environmental Health Protection Act was intended to ensure that state toxic air contaminant standards expressly take into account, and protect, infants and children.[48] OEHHA's directive is to list five TACs "that may cause infants and children to be especially susceptible to illness."[49] As the statute mandates, these five TACs will then be reviewed by ARB to determine whether the control measures for that TAC need to be revised.[50] The statute calls for revisions of any control measures adopted for the TAC listed, "as appropriate…to reduce exposure."[51] Thus, the main purpose of SB 25 is to allow ARB the opportunity to reevaluate control measures for TACs to ensure that children's health effects are addressed….

"Thus, the intent of the SB 25 list is to identify pollutants for which additional control measures may be necessary to protect children's health. If another regulatory vehicle already imposes maximum feasible control measures, then the intent of the statute is satisfied and the pollutant need not be listed to achieve

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48 S.B. 25, 1999 Leg., As Amended in the Assembly Comm. on Nat. Resources, July 8, 1999 (Ca. 1999), available online at http://www.leginfo.ca.gov/pub/99-00/bill/sen/sb_0001-0050/sb_25_bill_19990708_amended_asm.html.

49 CAL. HEALTH AND SAFETY CODE § 39669.5(a)(1).

50 CAL. HEALTH AND SAFETY CODE § 39669.5(b)(1).

51 *Id*.

this purpose. This clearly is the case for diesel particulate. Since children's exposure to diesel particulate will have already been reduced as low as technology will allow, it would not be "appropriate" for CARB to revise the control measures for diesel particulate. "

The comment goes on to discuss U.S.EPA emission standards for heavy-duty vehicles and some efforts underway in California to evaluate ways to reduce diesel exhaust emissions.

"Thus, standards that stretch the limits of feasibility are already required, and revisiting them with an eye to children's health will not make them any more stringent.

Since the intent of this SB 25 TAC listing is to identify pollutants for which additional control measures may be necessary to protect children's health, the intent of the statute would not be served if OEHHA fills one of the five spaces allocated for the initial SB 25 list for a TAC for which emissions already are being reduced to the lowest feasible level. The more appropriate step would be to maximize the effectiveness of the statute by placing a TAC on the list for which significant reductions are needed."

The comment goes on to describe PM10 emissions reductions and industry efforts to use green diesel technology.

"In light of these dramatic upcoming emissions reductions – which are the maximum emissions reductions that can be achieved –adding diesel particulate to the SB 25 list is simply unwarranted.

In contrast, where placement of a TAC on the SB 25 list is more likely to provide additional benefits for the protection of children's health, OEHHA should place that TAC on the SB 25 list before other TACs that will not result in such additional benefits. As we have shown above, the placement of diesel particulate on the SB 25 would provide no additional protection for children's health. However, there likely are other TACs that are known to cause adverse effects on children and whose inclusion on the SB 25 list would provide a benefit for children's health. (Some of these TACs are identified in Section II, below). At a minimum, OEHHA should place such other TACs, whose inclusion on the SB 25 list will provide additional protections for children's health, on the SB 25 list ahead of diesel particulate.

Response 3: The draft OEHHA document is a hazard identification document. It is not a risk assessment document or a risk management document. The statute does not require us to consider any ongoing risk management efforts in establishing a list of TACs that may cause infants and children to be especially susceptible to illness. SB25 requires OEHHA to consider in evaluating TACs for inclusion on the list: (1) exposure patterns among infants and children that result in disproportionately high exposure; (2) special susceptibility of infants and children; (3) effects of simultaneous exposures to compounds with the same mechanism of action; and (4) any interactions of air pollutants. OEHHA was not directed to consider present or potential risk management programs during the prioritization process. Discussing diesel exhaust particulate as a candidate for listing is valid; it is a TAC and has unique toxicity over and above other PM_{10} components. Existing reductions in PM_{10} are laudable but have no impact on this process.

Comment 4: Diesel Particulate Should Not Be Listed in Tier 1.

The difference between the TACs listed on Tier 1 and Tier 2 is the adequacy of direct studies available to support the proposition that the TAC causes "infants and children to be especially susceptible to illness."[52] According to OEHHA, the decision to include a TAC on the SB 25 list is based on the strength of the evidence linking that TAC to adverse effects on children's health.[53] "The strength of this evidence [indicating that infants and children may be more susceptible to the toxicological effects associated with that TAC than adults] was weighted heavily in this initial selection of eleven TACs that disproportionately impact children."[54] Indeed, in its March 19 and 20, 2001 public workshops, OEHHA observed that all chemicals currently listed in Tier 1 have *direct* evidence linking them to adverse children's health effects. In the case of diesel particulate, there is insufficient evidence to prove that it causes adverse effects on children's health. Not only does the evidence linking diesel particulate to adverse children's health effects pale in comparison to that of the five TACs recommended for Tier 1 inclusion, but the lack of human, animal, and child-specific data is also much weaker than for the other

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52 *Prioritization* at 6.

53 *Prioritization* at 5.

Tier 2 candidates, as well as for several other TACs that are not even included in the draft list. Unlike the other chemicals listed in Tier 2, let alone the chemicals listed in Tier 1, there are no direct links between diesel particulate and adverse effects on children's health. Therefore, OEHHA cannot justify listing diesel particulate in Tier 1.

A. Those Compounds Listed In Tier 1 All Have A Direct Link Between the TAC And Adverse Effects on Children's Health

> "The amount of child-specific evidence linking a particular TAC to adverse health effects in children is crucial to whether that TAC will be listed in Tier 1." The comment goes on to describe that differences between children and adults need to be studied on a case-by-case basis.

"The difference between the TACs listed on Tier 1 and Tier 2 is the adequacy of direct studies available to support the proposition that the TAC causes "infants and children to be especially susceptible to illness."[55] The strength of the data indicating that children are especially susceptible to the adverse health effects of the TAC is crucial to a listing in Tier 1. Since the five TACs that OEHHA has proposed for Tier 1 all have direct evidence of their adverse effects on children's health, the evidence supporting a listing of the five TACs is significantly stronger than the evidence supporting a listing of diesel particulate. "

The comment goes on to reiterate why the TACs in Tier 1 were chosen.

"Comparatively, the best OEHHA can do for diesel particulate are blanket statements that overstate the extent of the linkage between diesel particulate and adverse children's effects.

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[54] *Id*.

[55] *Prioritization* at 6.

B. There Is Insufficient Evidence Linking Diesel Particulate to Adverse Effects on Children's Health to Warrant a Listing in Tier 1.

If OEHHA does not have adequate studies linking a TAC to an adverse effect on children's health, OEHHA should not list that TAC in Tier 1. As explained below, the arguments used by OEHHA in support of their recommendation to list diesel particulate on the SB 25 list are, at best, indirect. In contrast, such direct evidence does exist for a number of chemicals which OEHHA has not included on its proposed SB 25 list. At a minimum, these compounds should be a higher priority than diesel particulate for inclusion in Tier 1 of the SB 25 list. Because OEHHA currently does not have adequate evidence of a link between diesel particulate and an adverse effect on children's health, it should not list diesel exhaust in Tier 1 of the SB 25 list.

1. Carcinogenicity

The available data on the potential carcinogenicity of diesel particulate to children is demonstrably inadequate to support placing diesel particulate on the SB 25 list. Diesel particulate is not a known human carcinogen. At most, there may be an increased relative risk in certain occupations that have chemicals and chemical mixtures, including both diesel and gasoline exhaust. Yet even if the data on the potential carcinogenicity of diesel particulate were as strong as OEHHA claims, there still is no animal or human evidence to suggest that exposure to diesel exhaust might be linked to increased carcinogenicity in children. Therefore, carcinogenicity does not provide a ground for listing diesel particulate in Tier 1.

As International and others have explained in previous comments, the data supporting a connection between exposure to diesel exhaust and lung cancer are extremely weak, with conflicting results and limited evidence of causality in the epidemiological database. " The comment goes on to describe the opinions of Dr. Sverre Vedal and Dr. Charles Poole regarding the strength of the evidence of carcinogenicity of diesel exhaust.

"And while OEHHA also relies on a single study of the railroad industry, the consensus of the scientific community – including the Clean Air Science Advisory Committee, the Health Effects Institute and the railroad study author himself – is that the study showed a <u>decreasing</u> risk of lung cancer with <u>increasing</u>

exposure to diesel exhaust. Thus, at best, this study provides limited, if not negative, support for OEHHA's claims about the link between diesel exhaust and lung cancer.

"While the weaknesses in the epidemiological database might be overcome by other evidence of carcinogenicity, such evidence is lacking for diesel exhaust. " The comment goes on to discuss select aspects of the Clean Air Science Advisory Committee discussions. "In sum, the available data falls far short of supporting OEHHA's characterization of diesel exhaust as a "likely human carcinogen."

Yet even if the data on diesel carcinogenicity were as strong as OEHHA claims, there is still no animal or human evidence to suggest a link between diesel particulate and increased cancer risks in children. OEHHA even admits that "[t]he epidemiological studies of the relationship between human exposure to diesel exhaust and lung cancer involve occupational situations that necessarily involve adults but not children, so direct evidence of differential effects on infants and children is not available from this source."[56] Not only do the existing studies on the carcinogenicity of diesel particulate apply only to workers, but they also implicate only *lung* cancer, an endpoint that is not of specific concern to children. Because, OEHHA has no evidence of either diesel particulate's effect on children nor on forms of cancer that are common to children, carcinogenicity cannot provide a basis for including diesel particulate on Tier 1 of the SB 25 list.

In contrast, OEHHA has identified several other TACs that are carcinogenic in adults and *also* have been shown to have demonstrated carcinogenic effects on children. Specifically, OEHHA notes that "[l]eukemias, lymphomas and brain tumors are the most common cancers among children."[57] Furthermore, OEHHA notes that "[e]vidence in experimental animals of increased cancer risk following early life exposure to carcinogens exists for a number of compounds, including urethane, vinyl chloride, DES, tamoxifen, nitrosourea compounds (e.g., methylnitrosourea), and alkenylbenzene compounds (e.g., safrole and estragole)."[58] Of these carcinogens, only vinyl chloride and nitrosourea compounds

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[56] *Prioritization*, Appendix B: Diesel at 7.

[57] *Prioritization* at 38.

[58] *Prioritization* at 40.

are TACs and subject to the SB 25 listing. However, OEHHA only lists vinyl chloride on the SB 25 list. Not only does OEHHA not list nitrosourea compounds in either Tier of the SB 25 list, but it only lists vinyl chloride, a TAC that poses direct carcinogenic concerns to children, in Tier 2. In an effort to remain consistent with their task of placing those TACs *most* harmful to children's health on the SB 25 list, OEHHA obviously should list these TACs before diesel particulate. By placing diesel particulate on the SB 25 list before these other pollutants, OEHHA is acting contrary to its own goal of developing a list of TACs that are *most* harmful to children.[59]

The difference in strength of the evidence linking the proposed Tier 1 TACs to increased cancer rates in children and the strength of the evidence for diesel particulate is striking. The mere assertion that children, in general, can be more vulnerable than adults to cancer is not sufficient to justify the extrapolation from adult to child in the diesel particulate findings. Therefore, the lack of concrete data to link diesel particulate to carcinogenicity in adults, combined with the even more tenuous attempt to link diesel particulate to carcinogenicity in children, are insufficient reasons to justify a listing of diesel particulate in Tier 1 based on increased carcinogenicity in children.

Response 4: OEHHA agrees with the comment that there are no data directly linking diesel exhaust exposure to a disproportionate lung cancer risk in infants and children compared to adults. However, the fact that diesel exhaust is a lung carcinogen is not the primary reason for considering diesel exhaust particulate as a candidate for the SB 25 list. Other perhaps more important evidence that caused OEHHA to place diesel exhaust in Tier 2 includes: 1) evidence that infants and children are potentially more susceptible than adults to genotoxicity and cancer induced by known diesel exhaust components (PAHs); 2) fine particulate (e.g., diesel exhaust) exacerbates asthma, (a respiratory disease which disproportionately impacts children) and adversely impacts both lung function and development in children, and 3) diesel exhaust is associated with other respiratory health effects as evidenced in

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[59] *Prioritization* at 3.

occupational studies and in the traffic studies cited in the document, and 4) evidence of developmental effects of PAHs, a constituent of diesel exhaust.

With regard to the diesel exhaust carcinogenicity data, the validity and applicability of the diesel exhaust cancer unit risk factor (URF) have been thoroughly documented in the diesel exhaust Toxic Air Contaminant (TAC) document and will not be discussed further. For further information and responses to the same comments brought up during the identification phase, the reader should consult the documents: *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Appendix III Part B Health Effects of Diesel Exhaust* and *Part C Responses to Comments.* (OEHHA, 1998; ARB, 1998b). The International Agency for Research on Cancer, the National Institute of Occupational Safety and Health, and many other regulatory bodies, including the U.S. Environmental Protection Agency treat diesel engine exhaust as a carcinogen. OEHHA analyzed dozens of studies that associated occupational diesel exhaust exposure with lung cancer including many studies which adjusted for the confounding effects of cigarette smoking. The relative risk estimate from the meta-analysis conducted buy OEHHA and later published (Lipsett and Campleman, 1999) describes a relative risk for lung cancer of 1.43 (1.31-1.57) for all smoking adjusted studies. Contrary to what is indicated in the comment, OEHHA relied on numerous studies in its evaluation of diesel exhaust as a carcinogen, not just the Garshick studies. In addition, our analyses of the dose-response information which was recently published (Dawson and Alexeeff, 2001), relied on both the case-control and cohort studies of railroad workers and obtained positive dose-response relationships. As noted above, all these issues were discussed at length during the identification phase for diesel exhaust as a toxic air contaminants.

Comment 5: Developmental/Reproductive

The available data on reproductive or developmental effects of diesel particulate similarly do not support its listing in Tier 1. There is no direct human data linking diesel particulate to reproductive or developmental effects. In fact, diesel particulate has not even been shown to have adverse developmental or reproductive effects in animals. As the U.S. EPA has recently determined, "exposure

to diesel exhaust would not appear to be a reproductive or developmental hazard."[60] The available data on the developmental and reproductive health effects of diesel particulate thus are inadequate to support a listing on the SB 25 list.

In contrast, OEHHA has data on the adverse effects on development or reproduction of many other TACs.[61] For example, OEHHA has some human evidence of adverse developmental or reproductive effects for the following TACs: arsenic, chlorinated dioxins and dibenzofurans, ethylene glycol monoethyl ether and ethylene glycol monomethyl ether.[62] OEHHA also cites to human studies that link dichlorodiphenyldichloroethylene (DDE) to adverse effects on the endocrine system.[63] Additionally, there is animal data that is at least suggestive of a developmental or reproductive hazard for the following other TACs: carbon tetrachloride, ethylbenzene, ethyl chloride, ethylene glycol monoethyl ether acetate, ethylene glycol monomethyl ether acetate, methyl bromide, and toluene.[64] Moreover, many studies have shown evidence of links between PCBs and cognitive developmental problems in children, detrimental effects to the immune system, and non-Hodgkin's lymphoma.[65] Yet only dioxins and glycol ethers have been proposed for addition to the SB 25 list. OEHHA would be acting arbitrarily and capriciously if it were to list diesel particulate based on reproductive or developmental toxicity, yet not list these other TACs with more direct evidence of adverse developmental or reproductive health effects. Therefore, developmental or reproductive effects cannot provide a ground for listing diesel particulate in Tier 1.

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60 U.S. EPA. *Draft Health Assessment Document for Diesel Exhaust*. p.5-53. (July 25, 2000).

61 OEHHA. All Chronic Reference Exposure Levels Adopted by OEHHA. (January 2001), available online at http://www.oehha.org/air/chronic_rels/AllChrels.html

62 *Id*.

63 *Prioritization* at 35.

64 OEHHA. All Chronic Reference Exposure Levels Adopted by OEHHA. (January 2001), available online at http://www.oehha.org/air/chronic_rels/AllChrels.html

65 Elizabeth Bluemink. "Monsanto Reports Available Online." *The Anniston Star Online News* (March 29, 2001), available online at http://www.annistonstar.com/news/news_20010329_4032.html.

Response 5: No data have been reported describing diesel exhaust-induced human developmental toxicity. However, appropriate animal developmental toxicity data could be sufficient to place a chemical in Tier 1 or 2. Both reproductive and developmental toxicity due to diesel exhaust exposure have been reported in animals. Yoshida *et al*. (1999) reported ultrastructural changes and a reduction in LH receptor mRNA expression in Leydig cells, and a dose-dependent decrease in daily sperm production in diesel exhaust-exposed mice. Watanabe and Kurita (2001) found that the anogenital distance was significantly longer in both male and female fetuses following exposure to diesel exhaust from gestational days 7 to 20. Although exposure resulted in some changes in maternal hormone levels relative to controls, the authors concluded that the effects observed were the result of exposure-induced changes in the fetus and its interaction with the maternal endocrine system, rather than maternal toxicity or adaptation. This last study was a contributing factor in the inclusion of diesel exhaust in Tier 2.

The comment implies that because other TACs are reproductive toxicants, they should be listed rather than diesel exhaust. As noted in the draft document (page 5 and elsewhere), the fact that a TAC is capable of inducing developmental toxic response is in and of itself not enough to list under SB 25. Many of the TACs induce developmental toxicity but at fairly high doses. Exposure information indicates that general ambient exposures would be insufficient to induce developmental toxicity. Some of these chemicals have RELs that are based on developmental toxicity. If the REL were close to measured ambient levels, then that would be of concern. We used this information in our prioritization process.

Comment 6: Asthma/Immunotoxicity

The current data available on the immunotoxicity of diesel particulate is also inadequate to support a listing on the SB 25 list. The increased incidence of asthma, particularly among children, is one of the mysteries of modern health science. It is undisputed that the rate of asthma in children has increased in the United States over the past several decades. It is also undisputed that elevated levels of particulate matter can exacerbate asthma. Yet while the incidence of asthma has been increasing, the levels of PM

in ambient air have been decreasing, as have the levels of diesel particulate.[66] Thus it is far from obvious that ambient PM exposures are responsible for the increased incidence of asthma in children. Nor is it obvious that diesel exhaust is responsible. Indeed, researchers have speculated as to many possible causes, including the increased loadings of ultrafine particulates that may be accompanying the decrease in overall PM levels[67], a *lack* of early exposure to dirt and pollutant that may cause the airways to fail to develop appropriate immune responses[68], and even cockroach excretia.[69] The bottom line is that we do not know why the incidence of asthma is increasing in children. OEHHA therefore cannot scientifically justify a conclusion that diesel particulate warrants placement in Tier 1 – in lieu of many compounds with *known* effects on children – simply because diesel particulate *may* induce asthma, or because diesel

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66 *See, e.g.*, Committee on the Assessment of Asthma and Indoor Air, Division of Health Promotion and Disease Prevention, National Institute of Medicine. *Cleaning the Air: Asthma and Indoor Air Exposures* (National Academy Press, 2000) (reporting that asthma prevalence has increased substantially since 1980); EPA, *Latest Findings on National Air Quality: 1999 Status and Trends*, p.2 (2000) (available online at www.epa.gov/airtrends) (reporting that PM emissions have declined 77% between 1970 and 1999, while PM levels have declined 18 percent since 1990).

67 *See, e.g.*, Elder, A.C.P., Gelein, R., Finkelstein, J.N., Cox, C. and Oberdorster, G. (2000). "Endotoxin priming affects the lung response to ultrafine particles and ozone in young and old rats," *Inhal. Toxicol*. 12, Supp. 1, 85-98; Barrett, T., Barr, E.B., Bice, D.E., Redman, T.K. (1998) "Role of Inhaled Ultrafine Particles In Exacerbating Asthma In Susceptible Individuals," Lovelace Respiratory Research Institute, available online at www.nercenter.org/pilotbice.htm; EPA Research Grant R826781, "Human Health Effects of Exposure to Ultrafine Particles," research conducted by University of Rochester School of Medicine and Dentistry, available online at http://es.epa.gov/ncerqa_abstracts/grants/98/healtheff/frampton.html; EPA Research Grant R826785, "Effects of Inhaled Ultrafine Particles on Asthma," research conducted by Lovelace Respiratory Research Institute, available online at http://es.epa.gov/ncerqa_abstracts/grants/98/healtheff/bice.html.

68 *See, e.g.*, Gereda, J.E., Leung, D.Y.M., Thatayatikom, A., Streib, J.E., Price, M.R., Klinnert, M.D. and Liu, A.H. (2000). "Relation between house-dust endotoxin exposyre, type 1 T-cell development and allergen sensitisation in infants at high risk of asthma." *Lancet* 355 (9216): 1680-1683; Ball, T.M., Castro-Rodriguez, J.A., Griffith, K.A., Holberg, C.J., Martinez, F.D. and Wright, A.L. (2000). "Siblings, day-care attendence, and the risk of asthma and wheezing during childhood." *N. Engl. J. Med*. 343 (8): 538-543; Wickens, K., Pearce, N., Crane, J. and Beasley, R. (1999). "Antibiotic use in early childhood and the development of asthma." *Clin. Exp. Allergy* 29 (6): 766-771.

69 *See, e.g.*, Potera, C. (1997). "Working the bugs out of asthma." *Environ Health Perspect*. 105(11): 1192-1194; Rosenstreich, D.L., Eggleston, P., Kattan, M. et. al. (1997). "The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma." *N. Engl. J. Med*. 336: 1356-1363; Committee on the Assessment of Asthma and Indoor Air, Division of Health Promotion and Disease Prevention, National Institute of Medicine. *Cleaning the Air: Asthma and Indoor Air Exposures* (National Academy Press, 2000); Mayo Clinic, "Cockroach Allergen: An Important Asthma Trigger" (July 1998), available online at http://www2.mayohealth.org/mayo/9807/htm/cockroach.htm.

particulate is a contributor to PM, which in turn *may* be responsible for an increased incidence of childhood asthma.

Response 6: Firstly, OEHHA did not include diesel exhaust as a TAC candidate for listing based on increased incidence of asthma as asserted in the comment. Rather, the listing is based on evidence that PM10 exacerbates asthma, not that it influences prevalence of asthma. 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. In addition, the smaller airways of children predisposes to more severe sequelae of asthma attacks. Indeed, the hospitalization rate for children 0 to 4 years of age for asthma is much higher than for any other age grouping (CDC, 1996a). Thus, on a population-wide basis, children are more impacted by asthma and substances that exacerbate asthma than adults.

Comment 7: Respiratory Effects

Finally, OEHHA does not have sufficient evidence of respiratory effects to warrant listing diesel particulate in Tier 1. As a preliminary matter, International notes that the U.S. EPA in the *Health Assessment Document for Diesel Exhaust* concluded the following about the noncancer health effects of diesel exhaust: "The overall conclusion of these [human] studies is that reversible changes in pulmonary function in humans can occur in relation to diesel exhaust exposure, although it is not possible to relate these changes to specific exposure levels,"[70] and that "Noncancer effects in humans from long-term chronic exposure to DPM [diesel particulate matter] are not evident [although some animal studies] showed pulmonary histopathology and chronic inflammation."[71]

Accordingly, based on pulmonary histopathology and chronic inflammation seen in high dose animal studies, U.S. EPA has established two alternative reference concentrations ("RfCs") (5 and 14 $\mu g/m^3$).

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[70] EPA, *Draft Health Assessment for Diesel Exhaust*, pp. 5-11 – 5-12 (2000).

[71] *Id*. at 5-70.

These RfCs have been approved by CASAC and represent the airborne concentration of a substance to which the general population, *including susceptible individuals*, may be exposed continuously for a lifetime without significant adverse effects.[72] In other words, there is an added uncertainty factor built into the RfC to ensure protection of sensitive subpopulations – which includes children. California has adopted the lower RfC value as its chronic reference exposure level (REL).

Significantly, a comparison of ambient diesel concentrations against these RfCs and REL demonstrate that current levels of diesel exhaust (let alone the reduced levels that are coming as a result of numerous regulatory initiatives) cannot reasonably be expected to cause any adverse respiratory effects – including effects on children. Annual average diesel exhaust exposure levels are well below both RfCs (in the range of 2 ug/m^3) and only a few "hot spot" areas are expected to exceed the RfC.[73]

These results are consistent with monitoring and modeling done by the South Coast Air Quality Management District (SCAQMD) as part of its "MATES-II" assessment of the Los Angeles basin. Even in heavily urbanized areas with substantial diesel traffic, the *highest* measured elemental carbon emissions were 5 µg/m^3 (90 percent confidence interval).[74] Even assuming that *all* elemental carbon is from diesel emissions, which it is not[75], the highest level of emissions in one of the most urbanized areas in the State would not exceed what OEHHA itself has determined is a concentration level that will be *without* adverse respiratory effects if breathed every day for a lifetime.

Moreover, OEHHA has determined that numerous other TACs that are targeted for respiratory effects have chronic inhalation RELs that are much lower than that for diesel exhaust (The comment goes on to list these).

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72 *See* www.epa.gov/iris

73 This exposure estimate is from EPA's *Draft National-Scale Air Toxics Assessment for 1996*. (Jan. 2001).

74 South Coast Air Quality Management District, *Multiple Air Toxics Exposure Study In The South Coast Air Basin*, p. 3-11 (March 2000).

75 For example, SCAQMD estimated that 67 percent of the elemental carbon mass in the basin was from diesel particulate. This percentage is based on a fine EC emission inventory developed for the year 1982, in which the highway vehicle emission factors used appear to be based on testing data from the late 1970s (Pierson

OEHHA acknowledges that their system of placing TACs on the SB 25 list was implemented so as to "determine which [TAC] posed the *most* potential hazard to children in California."[76] Implicit in an attempt to target those TACs that are most harmful to children's health is a comparison of various TACs' effects on children. As shown by OEHHA's own studies, many other TACs have much lower RELs for respiratory effects. Yet, formaldehyde and acrolein are the only TACs mentioned above that are proposed for listing on SB 25. OEHHA should list on SB 25 the TACs with lower RELs (and thus higher likelihood of adverse respiratory effects) before listing diesel particulate. Even if exposure levels are less for some of these TACs, OEHHA itself has acknowledged that their choice of eleven TACs "was heavily influenced by the toxicity of the compounds and less so by the estimated exposures to the compounds."[77] Therefore, the higher toxicity of these other TACs should supercede any potential exposure discrepancies and justify their listing on SB 25 before the listing of diesel particulate.

Response 7: As noted in the response to Comment 1, 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 has unique noncancer health effects over and above the cardiopulmonary toxic effects of general particulate matter. 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. The fact that there are TACs listed with lower chronic Reference Exposure Levels (RELs) than diesel exhaust has no bearing on the question of whether diesel exhaust may disproportionately impact the health of infants and children compared to adults.

The comment notes that many compounds have Reference Exposure Levels (RELs) that are lower than that established for diesel exhaust, and implies that we should be more concerned with those

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1979). Even assuming these data are correct, however, it would mean that actual diesel concentrations in the most heavily polluted area of Los Angeles are only approximately 3.4 ug/m^3 – well below the RfC.

[76] *Prioritization* at 3.

[77] *Prioritization* at 4.

compounds. What the comment misses is that the REL by itself does not indicate hazard; you need to combine the toxicity information with exposure information to determine hazard. In the prioritization process, we used ambient concentration data where available and divided the REL by the concentration in air to ascertain how close existing ambient concentrations are to a level of concern. Both formaldehyde and acrolein come close to or are above existing ambient concentrations. Diesel exhaust concentrations (around 3 $\mu g/m^3$ as a statewide average) are generally a little below the REL(5 $\mu g/m^3$), but much higher concentrations of diesel exhaust have been measured in urban canyons (up to 10 ug/m^3; CARB, 1998). In sum, the REL must be combined with exposure information to determine significance of the chemical in air. Also, the prioritization process evaluated cancer risk by multiplying ambient concentration data by the unit risk factor. When this is done for diesel exhaust particulate, the significance of exposure becomes important.

Comment 8: In addition to a lower REL, there are additional reasons to list acrolein before diesel particulate. Of the 32 hazardous air pollutants assessed by the EPA in its *Draft National-Scale Air Toxics Assessment*, the Agency concluded that "those that appear to pose the greatest health threats to individuals (from inhalation exposure) in all parts of the U.S. are chromium, acrolein, benzene, formaldehyde, and carbon tetrachloride."[78] Additionally, EPA used a non-cancer hazard quotient (HQ) for 27 of the pollutants that compared the RfC to the median exposure concentrations. If the current exposure level is exactly at the RfC (*i.e.*, the known safe dose), the HQ is 1. For those where the exposure levels exceed the RfC, the HQ is greater than 1. With respect to acrolein, EPA found,

> "For at least 50% of the population, the inhalation HQ associated with a single pollutant – acrolein—was approximately 4. The HQ for the most exposed 1% of the population

[78] EPA, *Draft National-Scale Air Toxics Assessment for 1996*, 124.

> was approximately 20. No other pollutants approached within an order of magnitude of acrolein's HQ distribution."[79]

The *Assessment* thus found that at least 50 percent of the population is being exposed to acrolein in levels that are four times the known safe concentration. The Assessment further noted that "one pollutant, acrolein, presented an HQ exceeding 10 to more than 20 million adults and 4 million *children*. Virtually all adults and children in the US population lived in census tracts were the median HQ exceeded 1.0."[80] Additionally, three more pollutants—formaldehyde, acetaldehyde, and manganese—showed HQs exceeding 0.1 for some of the U.S. population.[81] The remaining pollutants in the national-scale assessment were found "not to contribute HQs exceeding 0.1 for 99% of the population."[82]

A fact common to all of OEHHA's concerns is the enormity of data gaps connecting diesel particulate to adverse health effects on children. Given the absence of data linking diesel particulates to adverse children's health effects, OEHHA should simply remove diesel particulate from the SB 25 list. The evidence for diesel particulate is simply too tenuous to justify its listing above other TACs with a more plausible connection. At a minimum, however, it would be arbitrary and capricious for OEHHA to elevate diesel particulate to Tier 1 given the paucity of data suggesting diesel particulate could cause infants and children "to be especially susceptible to illness," as compared to numerous other compounds not listed by OEHHA.[83] Mere placement of a TAC on the SB 25 list is not a substitute for more conclusive data.

Response 8: OEHHA thanks the commenter for supporting our concern about acrolein.

The position of diesel exhaust in Tier 1 or 2 will be decided after responding to public comment and undergoing peer review by the state's Scientific Review Panel on Toxic Air Contaminants.

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79 *Id*. at 98.

80 *Id*. at 99 (emphasis added).

81 *Id*. at 98.

82 *Id*.

83 *Prioritization* at 6.