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Dr. Pingkuan Di
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PO Box 2815
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Via Email: pdi@arb.ca.gov

Dear Dr. Di:

This letter provides my comments on CARB's report "Diesel Particulate Matter Exposure Assessment Study for the Ports of Los Angeles and Long Beach," October 2005 Draft. I begin with some general comments, followed by comments on specific pages in the Draft Report.

CARB's conclusions about the health effects of diesel particulate matter (PM) are based on epidemiological studies reporting small statistical associations between PM and various health outcomes. When these studies report a statistically significant increase in risk, the relative risks typically range from a few tenths of a percent to a few percent across the range of ambient levels. However, CARB fails to acknowledge the evidence that epidemiological studies are probably incapable providing reliable information on risks this small, including whether the risks even exist. Air pollution epidemiology studies in particular have also been shown to suffer from data-mining, and publication bias making it likely that the risks claimed for PM at current ambient levels are not real.[1]

[1] H. Anderson, R. Atkinson, J. Peacock et al., *Meta-Analysis of Time-Series Studies and Panel Studies of Particulate Matter (PM) and Ozone* (World Health Organization, 2004), www.euro.who.int/document/e82792.pdf; M. L. Bell, F. Dominici and J. M. Samet, "A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study," *Epidemiology* 16 (2005): 436-45; S. N. Goodman, "The Methodologic Ozone Effect," *Epidemiology* 16 (2005): 430-5; G. Koop and L. Tole, "Measuring the Health Effects of Air Pollution: To What Extent Can We Really Say That People Are Dying from Bad Air?" *Journal of Environmental Economics and Management* 47 (2004): 30-54; T. Lumley and L. Sheppard, "Time Series Analyses of Air Pollution and Health: Straining at Gnats and Swallowing Camels?" *Epidemiology* 14 (2003): 13-4; S. H. Moolgavkar, "A Review and Critique of the EPA's Rationale for a Fine Particle Standard," *Regulatory Toxicology and Pharmacology* 42 (2005): 123-44; J. Schwartz, *Particulate Air Pollution: Weighing the Risks* (Washington, DC: Competitive Enterprise Institute, April 2003), http://www.cei.org/pdf/3452.pdf.

The case of hormone replacement therapy should add a dose of humility to CARB's improbably precise estimates of the port's health effects. Based on epidemiological studies, researchers concluded that not being on HRT increased a woman's risk of heart disease by 100% (i.e., a factor of 2).[2] An influential meta-analysis of these studies, published in 1991, helped make HRT one of the most prescribed therapies in the United States. But more recently, randomized controlled trials, which eliminate the possibility of confounding by unobserved factors that affect health, showed that HRT does not reduce heart disease risk and might even increase risk.

Thus, in the HRT case, even a 100% increase in risk based on epidemiological studies turned out to be spurious once all confounding effects were genuinely eliminated through a randomized, controlled trial. The putative risks that air pollution studies are attempting to pick out are tiny by comparison—at most a few tenths of a percent to a few percent. Furthermore, the effects of air pollution need to be separated out from a much larger array of potential confounding factors than in the case of the HRT studies. In other words, even risks substantially larger than the putative risks of air pollution can't be reliably assessed with observational data. Indeed, some epidemiologists have suggested that epidemiological studies are inherently unreliable for assessing the existence of such small risks.[3]

Evidence from toxicology studies also provides little support for CARB's PM health effects claims. Toxicology studies have clearly shown that nitrate PM is not toxic, even at concentrations of hundreds of micrograms per cubic meter.[4] Toxicology studies also provide little evidence for acute health effects from ambient concentrations of diesel PM or PM in general.[5]

For example, a Health Effects Institute (HEI) study exposed both healthy and asthmatic volunteers to 100 ug/m3 of diesel particulate matter for 2 hours while they exercised intermittently on a stationary bicycle.[6] The researchers found little evidence an inflammatory response and the healthy subjects exhibited more evidence of inflammation than the asthmatics. In fact, according to the project summary, the study "did not find inflammatory changes in asthmatic participants after controlled exposure to diesel

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[2] See discussion of HRT and references to the literature in Moolgavkar, "A Review and Critique of the EPA's Rationale for a Fine Particle Standard."

[3] G. Taubes, "Epidemiology Faces Its Limits," *Science* 269 (1995): 164-69.

[4] L. C. Green and S. R. Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives," *Regulatory Toxicology and Pharmacology* 38 (2003): 326-35; M. T. Kleinman, W. S. Linn, R. M. Bailey et al., "Effect of Ammonium Nitrate Aerosol on Human Respiratory Function and Symptoms," *Environmental Research* 21 (1980): 317-26; M. J. Utell, A. J. Swinburne, R. W. Hyde et al., "Airway Reactivity to Nitrates in Normal and Mild Asthmatic Subjects," *Journal of Applied Physiology* 46 (1979): 189-96.

[5] Green and Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives."; Moolgavkar, "A Review and Critique of the EPA's Rationale for a Fine Particle Standard."

[6] S. T. Holgate, T. Sandstrom, A. J. Frew et al., "Health Effects of Acute Exposure to Air Pollution. Part I: Healthy and Asthmatic Subjects Exposed to Diesel Exhaust," *Research Report / Health Effects Institute* (2003): 1-30; discussion 51-67.

exhaust." Furthermore, it is unlikely that residents near the port would ever be exposed to diesel PM at levels approaching 100 ug/m3.

Another HEI study exposed healthy and asthmatic volunteers to 200 ug/m3 of concentrated ambient PM2.5 collected in the Los Angeles area for 2 hours with intermittent exercise.[7] There were no changes in symptoms or lung function in either the healthy or asthmatic subjects, and little evidence of inflammatory responses. Since many inflammatory markers were measured, and only a few changed, the authors pointed out that these changes could be due to chance. 200 ug/m3 is much higher than peak PM2.5 levels in the Los Angeles area (or anywhere else in the U.S.).

Diesel exhaust has been shown to cause premature death in animal studies, but not until concentrations reach levels tens to hundreds of times greater than would ever be experienced in ambient air.[8]

The epidemiological studies are not consistent with the virtual lack of PM effects in toxicology studies, even though the toxicology studies assessed PM concentrations much higher than are ever experienced in Los Angeles. This suggests that the report greatly overstates non-cancer health effects from port pollution.

CARB estimates the cancer risk for people living near the port ranges from about 50 to 1,000 in a million. To put it in more familiar and intuitive terms, CARB's estimate implies a risk of developing cancer ranging from 1-in-1,000 to 1-in-20,000 over a lifetime. These risks are greater than the arbitrary cancer-risk standard set by regulators, but they are tiny nevertheless. But even these small cancer risks are inflated for two reasons. First, following standard practice in regulatory risk assessment, CARB made assumptions that intentionally create an upward bias in the predicted cancer risks from port pollution. Even within the conventional regulatory risk assessment framework, the actual cancer risks from port pollution are probably at least several times lower and may be more than an order of magnitude lower.

Second, conventional regulatory risk assessment exaggerate cancer risks by mistakenly assuming that the risks from very high pollution exposures from either animal studies or mid-20th Century worker cohorts can be linearly extrapolated down to the much lower ambient exposures experienced today.[9] Diesel PM causes tumors in laboratory rats, but the mechanism involves particle overload and ensuing persistent inflammation.[10] This requires diesel PM levels hundreds of times greater than ever occur in ambient air.

There is no doubt that diesel exhaust at high enough concentrations is an unpleasant and aggravating nuisance. But this is a far cry from CARB's claim that thousands of people

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[7] H. Gong, Jr., C. Sioutas and W. S. Linn, "Controlled Exposures of Healthy and Asthmatic Volunteers to Concentrated Ambient Particles in Metropolitan Los Angeles," *Research Report / Health Effects Institute* (2003): 1-36; discussion 37-47.

[8] Green and Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives."

[9] B. N. Ames and L. S. Gold, "The Causes and Prevention of Cancer: Gaining Perspective," *Environmental Health Perspectives* 105 Suppl 4 (1997): 865-73; B. N. Ames and L. S. Gold, "Paracelsus to Parascience: The Environmental Cancer Distraction," *Mutation Research* 447 (2000): 3-13.

[10] Green and Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives."

are experiencing serious harm each year from port pollution. The report should be revised to reflect the fact that the health effects of port pollution are both far lower and less certain than CARB claims.

The remainder of this letter includes comments on specific portions of the report.

1. On page 1, the report states "Diesel PM emissions from the ports are a major contributor to diesel PM in the South Coast Air Basin. The combined diesel PM emissions from the ports are estimated to be about 1,760 tons per year in 2002." Without context, it's hard to know what this means. What is the ports' *percentage* contribution to total diesel PM emissions in the area affected by the port? More importantly, of total diesel PM and total PM2.5 in the air around the ports, what fraction is contributed by port emissions? The Executive Summary should clearly state these values and their uncertainty ranges. This discussion should also separate out the carbonaceous (soot) and nitrate components of the ports' contribution to total PM.
2. On page 2, the report states that cancer risk levels near the port range from about 50 to more than 500 in a million over a lifetime. Assuming the peak risk for residents near the ports is 1,000 in a million, this means that a person living near the port has a lifetime chance of between one-in-1,000 and one-in-20,000 of getting cancer due to port pollution. As discussed in my general comments, even these low risks are inflated. But I want to point out here that the standard regulatory practice of reporting risk on a cancers-per-million basis creates the appearance of large risk, when in fact the risk is tiny. The average reader of this report would find a "one-in-X chances" description of probability far more familiar and intuitive. Wherever the report includes an "X-per-million" it should also always include a "one-in-X chances" risk description.
3. On page 3, the report states "Diesel particulate matter is a major component of particulate matter in many cities." Rather than the vague "major component" phrasing, the report should just say what percent of PM2.5 is diesel PM in the area affected by the port, and how much of the total diesel component actually comes from the port. Furthermore, diesel PM should be separated into the carbonaceous and nitrate components. This separation is important for two reasons: First, although some nitrate PM is due to NOx emissions from diesels, calling nitrate PM "diesel PM" is misleading. The special concern about diesel PM isn't due to nitrates, but to carbonaceous particles (i.e., soot). Second, as already noted, toxicology studies have clearly shown that nitrate PM is not toxic, even at concentrations of hundreds of micrograms per cubic meter.[11] Whatever health effects CARB is attributing to nitrate PM from port emissions, these health effects should be removed from the risk tallies in the report.
4. On page 4, the report states, "These studies [of PM health effects] have found an increase of one to two percent in daily mortality associated with each 10 ug/m3

[11] Ibid; Kleinman, Linn, Bailey et al., "Effect of Ammonium Nitrate Aerosol on Human Respiratory Function and Symptoms."; Utell, Swinburne, Hyde et al., "Airway Reactivity to Nitrates in Normal and Mild Asthmatic Subjects."

increase in PM10 exposure." This dose response is about a decade out of date. The report should use 0.2% to 0.3% per 10 ug/m3, which is the latest PM10 dose-response from NMMAPS, or some other value that reflects the weight of the evidence from the recent literature.[12] As I noted above, I don't believe these studies are identifying real causal effects, but CARB should at least be using numbers that reflect the numbers being published in the recent scientific literature. As the epidemiological methods have been refined, the dose response curve from epidemiological studies has been getting flatter.

5. On page 11, the report states "These estimates [of PM health effects] are based on a well-established methodology for calculating changes in health endpoints due to changes in air pollution levels." This statement creates the false impression that we have evidence of direct causal links between given air pollution levels and given people's health or medical status. The report should remove this sentence and instead make it clear that all of the health claims in the report are based on small statistical correlations between air pollution levels and health endpoints, and that the causal relationship CARB is claiming between air pollution and health outcomes is inferred, not measured. Furthermore, the report should note that toxicology studies do not support CARB's claims about the health effects of port pollution.

6. The report discusses some aspects of the uncertainty of its estimates (see pp. 11-12), however the discussion is both incomplete and misleading. The discussion is incomplete, because it does not discuss the large uncertainties in the actual emissions of diesel PM from port-related activities. Emissions inventories are notoriously uncertain, and they are most uncertain for specialized non-road engines.

   The uncertainty discussion is also misleading, because it conflates uncertainty with bias. Bias occurs when errors are not random. As CARB notes "risk assessment is intentionally designed to avoid underprediction." In other words, following standard regulatory practice, CARB is intentionally erring on the side of overstating the cancer risks from port pollution. Because of all the safety factors implicitly built into risk assessments, this overstatement could be anywhere from a factor of several, to a factor of 10 or more.

   I stress that the risk overstatement discussed here has nothing to do with the validity of the standard regulatory risk assessment paradigm of extrapolating estimates of high-exposure risks linearly down to ambient exposures. Taking that framework as given, the report still greatly overstates port cancer risks. The report should make clear that this is intentional overestimation, rather than uncertainty, and should clearly state that the real cancer risks from port pollution are substantially lower than the numbers from the risk assessment would lead one to believe.

   The heading of the uncertainty discussion should be changed to "What are the uncertainties *and biases* associated with *this* risk assessment" and CARB should discuss the magnitude of these uncertainties and biases in quantitative detail in the Executive Summary.

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[12] Moolgavkar, "A Review and Critique of the EPA's Rationale for a Fine Particle Standard."

7. CARB seems to have derived all its health effects estimates from models. Have there been any efforts at real-world validation of the emission rates or ambient PM levels output by these models, either for the ports or in other parts of the country? The reliability and uncertainty in the models' output should be discussed in the Executive Summary, including specific quantitative details. If the reliability and uncertainty of the model's output is unknown, the report should clearly say so.

In my specific comments, I've focused on the Executive Summary, because that is what most people who look at the report will actually read. However, the rest of the report should also be revised as necessary to reflect my comments.

Thank you for the opportunity to comment on CARB's port pollution risk assessment and regulatory activities. If you have any questions or would like additional information, you can reach me at 916.203.6309 or joel@joelschwartz.com.

Sincerely,

/Joel Schwartz/

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