School Buses and Diesel Fuel

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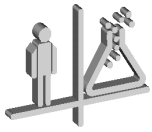
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EXECUTIVE SUMMARY

Diesel exhaust—we all know the smell and sight, whether coming from an 18-wheeler's exhaust stack or the tailpipe of a diesel-powered school bus. But what do we know about the risks that diesel exhaust (DE) may pose to human health? The authors of *No Breathing in the Aisles*, a report released by the Natural Resources Defense Council (NRDC) and the Coalition for Clean Air (CCA), claim that children riding diesel-powered school buses are at a significantly increased risk of developing cancer from exposure to DE. This is a serious claim, if true, and one that merits analysis from a scientific perspective. The American Council on Science and Health (ACSH), committed to sound public health policies and the protection of human health, has reviewed the NRDC/CCA report and concludes the following.

- The monitoring study, which forms the basis of the report and from which the NRDC/CCA based its risk estimates for children, is not described in sufficient detail, nor are enough data presented to allow for a thorough and informed critique of its validity. Thus, the conclusions regarding cancer risk to children from DE exposure cannot be confirmed.

- The estimated DE exposures for children riding a diesel-fueled school bus have not been confirmed through other independent studies. Quantitative

human exposure data are rarely available for DE, particularly for non-occupational settings, and thus, comparisons to the NRDC/CCA findings are not possible.

- DE has been associated with a small increase in lung cancer risk in some epidemiological studies involving occupational exposures. Typically, occupational DE exposures are far greater than ambient exposures to which children and the public would potentially be exposed. No published studies are known that link ambient or non-occupational exposure to DE with increased cancer risk.

- The estimated cancer risk for children calculated by the NRDC/CCA uses a unit risk factor that has not received general acceptance by the scientific community and is not currently used by the U.S. Environmental Protection Agency (EPA).

- No case reports or studies are known to link childhood exposure to DE, either on buses or in any other exposure scenario, with an increased risk of developing cancer.

INTRODUCTION

Recently, the National Resources Defense Council and the Coalition for Clean Air released a report entitled *No Breathing in the Aisles* (NRDC, 2001), which focused on diesel exhaust (DE) from diesel-powered school buses and the potential health risk it poses to children. The main conclusion of the report is that children who ride diesel-powered buses are at higher risk for developing cancer at some time in their lives than are those without such exposures. The American Council on Science and Health (ACSH), because of its orientation and focus on presenting balanced, scientifically sound analyses of current health topics, felt compelled to weigh in on this current concern. Children's health is a prominent topic in the news and, given the various risk factors that are often

identified (and misidentified) as important to children's health (e.g., environmental chemical exposure, pesticides, lead, diesel exhaust, drugs, violence, and teen pregnancy), it is critical to emphasize those most significant to children's health. Misallocation of attention and resources to the "wrong" risk factors can do more harm than good, in this respect. With this spirit in mind, ACSH undertook a review of the NRDC/CCA report to evaluate its scientific merit and the basis behind its conclusions.

REVIEW OF THE BUS MONITORING STUDY

The monitoring study conducted by the NRDC/CCA has numerous shortcomings, which prevents a thorough analysis and precludes confirmation of the report's conclusions. The methodological and statistical details are not presented in sufficient detail to allow independent evaluation and validation of the results. Thus, many of the statements in the report concerning exposure and risk to children cannot be substantiated. For example, the document states that "comparison of the Aethalometer readings in the car and the bus allowed a very accurate continuous measurement of the difference in black carbon particles between the two vehicles and a very good estimate of the diesel smoke attributable to the school bus." However, the report fails to provide numerical averages for either vehicle. In short, there is insufficient data and information provided to support the position that DE is a risk to children who ride diesel-powered school buses.

Appendix C of the NRDC/CCA report contains general specifications for the Aethalometer (an instrument designed to measure carbon particles, or soot), while Appendix D contains general specifications for the DataRam (which measures particulate matter). For clarification, measurements of soot and particulates are both surrogates for the presence of DE, which cannot be directly quantified. However, information pertaining to calibration, positioning of the equipment in the buses, and collection of data are not provided. Instrument cali-

bration is very important to the accurate quantification of DE concentrations in any environment. The Aethalometer is calibrated by performing quantitative chemical analysis for black carbon on the filter, removed from the instrument at the end of the sampling period (Hansen et al., 1984), yet there is no indication that this was performed. Direct-reading instruments such as the Aethalometer are highly dependent upon the particle size, opacity, and composition of the material deposited on the filter and, therefore, its use in this manner is not considered a scientifically established analytical technique for particle mass concentration (ACGIH, 1995).

Failure to follow standard scientific protocols and a lack of attention to methodological detail is illustrated by the following excerpts.

- "When some windows on the bus are open, the levels of diesel exhaust go down, and when all of the windows are closed, the levels rise." How many windows are open? By how much do diesel levels rise and fall?

- "The only times when the levels of diesel exhaust particles in the car briefly exceeded the levels in the bus were when the car was behind the bus for a few minutes idling at a school . . . ." For how many minutes?

- "In Figure 3, all of the concentrations represent average levels measured in the bus, minus the average ambient levels measured in the car or outdoors." Which was subtracted—the car levels or the levels measured outdoors, and when? Moreover, does Figure 3 represent all of the data for all of the buses, or selected data points?

- "There was significant variability among individual buses sampled. Even two buses from the same model year had different levels of diesel exhaust." Do the authors mean statistical or practical significance here? In addition, it would be important to know not only what differences existed between buses, but also cur-

rent average DE concentrations in rural and urban Los Angeles vicinities as well as statewide variability in DE concentrations.

The report concludes: "[t]hus, for the time a child is in a school bus, his or her exposure to diesel exhaust may be up to 8½ times the average statewide air levels." That may be an interesting statistic, but it is not the relevant comparison. More central to the topic is the average difference between DE exposure on a diesel-powered bus ride and exposure in other ambient environments (e.g., riding to school in a car or walking to school in an urban area). It would also be helpful to know what the current ambient background concentrations of DE are in Los Angeles and California, as the NRDC/CCA report cites ambient concentrations for 1995, somewhat dated.

Based on Figure 2 ("Real-Time Bus and Car Comparisons") in the report, it appears that the average DE concentration on the bus is 3–4 times the average concentration in the car. But Figure 2 is difficult to interpret, as the numerical gradations on the x-axis vary between one and four minutes per gradation, and there is no explanation for this variance. It is also interesting that a leading (i.e., ahead of the bus), not a following, car was used in the study for comparison purposes. The study should have included both upstream (leading car) and downstream (following car) measurements, as a car trailing a diesel-powered bus is likely to have greater DE exposure than one in front.

The report states that four buses were monitored with collective monitoring time totaling 20 hours. However, Figure 2 showing DE concentrations seems to be based on the worst-emitting bus. Comparing the average concentrations on the other buses, with the respective car average, the differences are much less, ranging from approximately 1.2 to 2. Figures 4 and 5 in the report also appear to be representations of worst-case emissions, not average emissions from all four buses. Additional questions pertain to whether the buses were selected at random, were they representative of the bus fleet, and were enough selected and used to provide sta-

tistical significance. Frequently, in comparison studies such as these, statistical significance can be meaningful and important to the interpretation of the results, yet there is no discussion as to whether statistical analysis for any of the data collected was performed. In summary, the report does not provide enough information and data to allow an independent assessment of its validity, and there are unanswered questions that prevent one from concluding that DE exposure in buses poses a health risk to children.

REPORT CHAPTER 2 ("THE SERIOUS HEALTH IMPACTS OF DIESEL EXHAUST") EVALUATION

Chapter 2 of the report purports to characterize the risk associated with DE, yet the discussion does not include much of what we know about DE and its potential effects in humans. Many statements of fact are either not supported with appropriate references, or they cite information derived largely from California State agencies. It is not essential that peer-reviewed literature be cited, but the degree of credibility afforded such a report increases substantially if supportive citations are provided. More problematic for independent reviewers of this report is the inability to have access to many of the internal communications and other nonpublished sources that are cited.

In contrast to the literature reviewed in the NRDC/CCA report, numerous peer-reviewed studies exist which contribute to our knowledge base concerning the potential risk that DE poses. For example, the Draft EPA Health Assessment Document for Diesel Emissions, released in July 2000 (EPA, 2000a), contains thousands of primary references related to peer-reviewed studies on DE and related effects on human health.

DE has been associated with a small increased cancer risk in some occupational studies, which is discussed in the report, but the focus of this chapter should have been on the weight of scientific evidence for DE, so that an informed assessment of the potential risk to children

from bus-derived DE exposure could be made. Data from occupational studies should not be directly extrapolated to ambient, non-occupational exposures for children and the report fails to make this point. The exposures are different and the risks may well be different. The authors should focus attention on what we know about DE and the hazard it may pose to children. Many statements included in the chapter—the two repeated below, for example—are not supported by appropriate citations and focus primarily on occupational studies.

- "Diesel exhaust has been demonstrated in more than 30 human epidemiological studies to increase cancer risk." Such a statement of broad implication should be supported with references.

- "Over two dozen well-designed occupational studies have demonstrated that long-term exposure to diesel exhaust significantly increases the human incidence of long-term lung cancer [*sic*] and possibly of bladder cancer."

More germane to the current report should be a focus on what evidence and data exist for ambient exposures to DE, particularly for children. Occupational studies, while providing information related to an agent's hazard potential, frequently are not appropriate surrogates for ambient or environmental exposures.

Much of the chapter focuses on broad topics such as air pollution as a risk factor in disease, exacerbation of asthma related to DE, and the susceptibility of children—a common topic these days when discussing environmental hazards. Many of these points—as of the excerpts below—are not supported with citations from the peer-reviewed scientific literature and, thus, cannot be construed as fact, but must be regarded as author opinion.

- "Premature death due to long-term exposure to particulate matter and other air pollution has also been a subject of much research." No citation.

- "Diesel exhaust is also believed to exacerbate asthmatic conditions." No citation.

- "Children are among those most susceptible to the health effects of diesel exhaust." No citation.

This last assertion is largely unfounded, as there are no known comparative studies that have evaluated child and adult response to DE exposure. Aside from assumptions about a child's developing body, narrower airways, faster metabolism, and increased breathing rates, no studies are known to exist that support the contention of greater child susceptibility. In fact, the opposite argument could be made—that differential physiological characteristics in children (i.e., narrower airways) result in reduced penetration of DE particulates to the deep lung, and that faster metabolism or lymphatic clearance may result in increased clearance of exogenous particulate matter. Mauderly et al. (1987) reported that DE did not affect the developing rodent lung more severely than the adult rat lung and that particulate clearance was faster in the younger lung. We know that live and kidney clearance of some substances reaches and exceeds adult capacities during the first years of life and that children thus have the ability to metabolize certain chemicals, drugs, and other substances more readily than adults—a reversal of the presumption that children are always more sensitive (Bruckner, 2000).

Much of the chapter's discussion concerns speculation based on alleged associations between environmental hazards and health effects in humans. This is illustrated by the discussion in the last three paragraphs of Chapter 2:

- The authors report that there is some type of correlation or association between air pollutants, including DE, and asthma attacks.

- The authors then associate an increase in asthma-related hospital admissions and emergency-room visits by low-income children with periods of persistently high ozone levels, despite the question of whether

socioeconomic status is related to physiological susceptibility.

- DE is brought back into the picture as a result of ozone being created when various air contaminants react in sunlight.

- Finally, this discussion concludes: "[S]tudies have also shown that the proximity of a child's school or home to major roads is linked to asthma and the severity . . . of symptoms increases with proximity to truck traffic."

In this discussion, some sort of relationship is alleged to involve DE, ozone, hospital admissions, asthma, proximity to roads, and severity of symptoms. The report goes on to assert that asthma is the leading cause of school absenteeism attributed to chronic illness conditions, and that asthma has been associated with poorer academic performance, more activity limitations, and increased dropout rate, in addition to its causing affected children to require special education programs. It is unclear how this discussion of affected children with asthma relates to the primary purpose of the study, i.e., that of measuring DE concentrations in school buses. The authors fail to point out that the rise in asthma incidence (Clark et al., 1999), regardless of whether DE is an influencing factor, has occurred during a period of general improvement in outdoor air quality (EPA, 2000b; Nelson, 1998), which represents a non-correlation and argues against a causal relationship between ambient pollution and asthma incidence. Other studies described in the report that address air pollution and effects on humans cannot be used as a proxy for the role that DE might play in eliciting health effects, as the contribution of DE in such studies is rarely measured or reported. While fine particulates from DE are implicated as a causative agent in human disease, we should stress that there are many other sources of fine particulates.

DIESEL EXHAUST AND ALTERNATIVE FUELS: COMPARATIVE ANALYSIS

The NRDC/CCA report compares the potential risks from DE exposure with that from other alternative fuels, and such an analysis presents only part of the picture. While on the surface it may be reasonable to compare diesel exhaust with other alternative fuels, this type of comparison is valid only based on the hazard associated with each type of emission or exhaust. For example, the principal concern associated with DE exposure appears to be cancer risk while an alternative fuel's primary hazard or concern may be some other health endpoint. When comparing the potential risk associated with DE on a diesel-powered bus to other scenarios and other fuels, one needs to be clear-cut on what health endpoints are compared.

The statement that DE exposure from diesel-powered buses is greater than from non–diesel-powered buses assumes that we have adequate information for such a comparison to be made. In other words, this assumes that we have measured DE concentrations on non–diesel-powered buses and that the levels are lower. The premise that non–diesel-powered buses present lower health risks also assumes that we have adequate information and data about the types of emissions that these buses produce and about the potential health risks (i.e., presumably lower) associated with these alternative fuels. These are assumptions that have not been addressed in the report.

Finally, there are different types of risks associated with alternative fuels, and it is important to evaluate all risks that accompany a fuel type. One that comes to mind concerning compressed natural gas is explosivity. With any flammable fuel, diesel included, it is important to compare all potential risks, not just the risk (i.e., chemical hazard) associated with inhalation of fugitive emissions. All potential risks—including flammability, explosivity, and toxicity of alternative fuels—should be compared to diesel fuel to present a more complete picture of potential risk.

HEALTH HAZARDS OF DIESEL EXHAUST

The NRDC/CCA report concludes that DE poses a significant risk to the health of children, although no direct evidence or studies exist to support this statement. In EPA's Draft Health Assessment Document for DE (EPA, 2000a), this statement is made: "Caution is warranted in the interpretation of results from the epidemiological studies that have addressed noncarcinogenic health effects from exposure to diesel exhaust. These investigations suffer from myriad methodological problems, including (1) incomplete information on the extent of exposure to diesel exhaust, necessitating in some studies, estimations of exposures from job titles and resultant misclassification; (2) the presence of confounding variables such as smoking or occupational exposure to other toxic substances (e.g., mine dusts); and (3) the short duration and low intensity of exposures. These limitations restrict drawing definitive conclusions as to the cause of any noncarcinogenic diesel-exhaust effect, observed or reported."

Interest in DE toxicity has been prominent for some time because of DE's complex chemistry, because DE has individual constituents that by themselves have been shown to be carcinogenic in laboratory studies, and because it is visible and possesses a distinctive odor. These last two characteristics are perhaps more important contributors to impressions of its perceived toxicity than the actual toxicological data would suggest. A brief review of the toxicological profile for DE is warranted.

Noncancer Effects

Some of the more commonly reported acute effects of DE include subjective complaints of eye, throat, and bronchial irritation, and symptoms such as headache, lightheadedness, nausea, vomiting, and numbness/tingling of the extremities (Rudell et al., 1990, 1994). One of the NRDC/CCA's concerns over acute effects on children riding school buses is whether short-term exposure to DE might result in acute decrements in ventilatory function and whether the frequent repetition of such acute

respiratory effects could result in chronic lung function impairment. One way to study reduced lung capacity is to monitor differences in pulmonary function in occupationally exposed workers (a group whose exposure to DE is higher than that of children) at the beginning and end of a work shift. In studies of underground miners, locomotive repairmen, and bus-garage workers, increasing respiratory symptoms (cough, phlegm, and dyspnea) and decreases in lung function over the course of a work shift were generally found to be minimal and not statistically significant (Reger, 1979; Battigelli et al., 1964; Ames et al., 1982; Jorgensen and Svensson, 1970). In a study of acute respiratory responses in diesel bus-garage workers (Gamble et al., 1987), there was an increased reporting of cough, labored breathing, chest tightness, and wheezing, but no reductions in pulmonary function were associated with exposure to DE. Thus, given the minimal impact upon occupationally exposed individuals, there appears to be little basis for concern for non-occupationally exposed individuals.

Much of what is known about acute effects of DE comes from case reports that lack quantitative exposure information. There is a consensus that reversible changes in pulmonary function in humans can occur after DE exposure but that it is not possible to relate these changes to specific exposure levels. Taking the available data in total, the absence of reported noncancerous human health effects—except infrequently occurring effects related to respiratory symptoms and pulmonary function changes—is notable (EPA, 2000a).

Cancer

The primary concern related to human exposure to DE is that of its carcinogenic potential, a concern based on some evidence that shows a small increase in cancer risk from long-term occupational exposure (EPA, 2000a). There are a number of epidemiological studies on DE and cancer in humans, and to assess the totality of the evidence, a meta-analysis (a review that looks at the overall results of the collective studies) is often appropriate. Taken together, the epidemiologic data show a weak asso-

ciation between occupational DE exposure and lung cancer (Cohen and Higgins, 1995). The evidence suggests that there is approximately a 1.2–1.5-fold increase in the relative risk of lung cancer compared with workers classified as unexposed (Cohen and Higgins, 1995; Bhatia et al., 1998). A risk ratio this low (i.e., contrast with a risk ratio of > 20 associated with cigarette smoking) limits our ability to make confident statements relative to how occupational DE exposure impacts cancer risk.

The most significant shortcoming of the available human occupational studies is their failure to quantify exposure. Most occupational studies have evaluated exposure on the basis of work histories reported by subjects or their next of kin, or by retirement records. Because of the absence of exposure information, this key factor limits interpretation of the epidemiologic findings and consequently their utility in making quantitative estimates of cancer risk.

The carcinogenicity of DE in experimental animals is inconclusive with respect to its relevance to humans. There is no evidence in rat DE inhalation studies to support carcinogenicity of DE at moderate or low exposures (Mauderly et al., 1987; Heinrich et al., 1995). In rat studies with positive findings, cumulative exposure doses are extremely large and not likely to have any relevance to potential human exposures. Such studies clearly show an association with lung cancer at concentrations higher (measured in mg/m^3, or exposure 1,000 times greater) than the ug/m^3 levels that humans might experience. Mouse, hamster, and monkey studies did not yield positive results and, thus, the rat may be unique in its carcinogenic response to DE (EPA, 2000a).

OVERVIEW OF THE NRDC REPORT

The basis for the NRDC/CCA claim that children are at increased cancer risk from exposure to DE is a monitoring study that measured DE concentrations in empty buses during a "typical" run to and from school. With this as a starting point, the authors of the report dis-

cuss a number of topics, including estimates of the cancer risk to children, recommendations for less hazardous fuels, cleaner modes of public transportation, and guidance on written appeals to public officials concerning funding for replacement of diesel buses. This is a broad scope for one report, one predicated largely on a single monitoring study using 4 buses. A closer look at the NRDC/CCA report reveals what follows.

The Crux of the Report—Cancer Risk to Children

Although the report authors suggest that children are at significant risk of cancer from exposure to DE on school buses, they fail to provide a sound scientific basis for that assertion. A standard risk assessment of diesel exhaust would incorporate (*a*) peer-reviewed exposure data showing distributional estimates of DE exposure for the general population, and (*b*) a hazard benchmark (i.e., a cancer unit risk factor—a defined number used to estimate how many excess human cancers will result from a given exposure) that has been scientifically derived and based on the weight of the evidence for DE.

For exposure estimates, the authors use limited and selective exposure data, which is inadequate for a robust assessment of cancer risk. The monitoring study did not measure actual child exposures but used analytical instruments inside an empty school bus as a surrogate indicator. Given that children commonly ride buses, the NRDC's study would more accurately quantify exposure if personal monitoring had been employed. There are very few exposure data for DE, and given the importance of this variable in the risk equation (i.e., risk is the probability of an occurrence that depends on both the hazard and exposure), the report's quantitative estimates of cancer risk must be viewed with considerable uncertainty.

For DE, there appear to be only two occupational studies that possess quantitative exposure-response estimates for lung cancer, and neither of these is considered suitable for a risk assessment (HEI, 1999). It is believed that the unit risk number used in the NRDC/CCA calculation was based on a study of railroad workers, and this study is considered inappropriate for estimating cancer

risk for the reasons stated below.

- The exposure data on individuals was inadequate (NCEA, 1999).

- Evidence for a positive association was dependent on risk differences by job categories; within each category the risk decreased as years of work increased—which suggests that there was a negative exposure-response trend (NCEA, 1999).

- There are confounding factors that could influence any exposure-response trend, including bias due to exposure misclassification, incomplete ascertainment of lung cancer deaths by job category, other uncontrolled exposures (e.g., smoking), and analysis of relative versus absolute risks. The salient point of this discussion is that the EPA and their Advisory Board (CASAC) concluded that it would be unproductive at this time to "engage in more dose-response analyses" of the railroad worker study (NCEA, 1999). Therefore, the use of the unit risk number by the NRDC/CCA report in its calculation of cancer risk to children is unprecedented and not recommended by other regulatory groups.

Conservative Assumptions and Calculations

In contrast to the cancer risk estimates reported, an assessment of the variability and uncertainty associated with estimating cancer risks should be discussed. Specifically, a probabilistic risk assessment (distribution risk estimate) is preferable to a deterministic estimate (single point risk estimate). This is to say that risk ranges should be presented that show the variability that exists, depending on the selection of various input factors. For example, several of the input parameters chosen were worst-case values instead of average values. The authors selected 180 days/year as the number of days per year a child typically rides the bus to school, although this is more likely the maximum number of days that school is in session and therefore, more closely represents the max-

imum number of days a child may ride a bus. The same point pertains to the number of hours per day and the number of years that a child spends riding a diesel-powered school bus. The risk estimates appear to represent those for a maximally exposed individual and should not be construed to represent the risk to all children or to the general population.

In addition, the calculated cancer risk estimates appear to be based on the average DE concentration from the worst-emitting bus. Through visual estimation of Figure 3 (since the actual data were not reported), the average DE concentration attributable to the school buses appears to be 6 ug/m^3. The authors used 13.8 ug/m^3 based on the worst-case bus; if the average value had been used, the potential theoretical excess cancer risk would be 10–20 per million individuals, not 23–46 as reported.

Communication of Cancer Risk

It is common to express the cancer risk that exposure to a certain chemical may pose in terms of excess human cancers per million persons. This is predicated on the long-standing EPA policy of considering anything less than a one-in-a-million risk as an acceptable risk whereas anything greater is not acceptable. With this in mind, what follows should be understood.

Estimated cancer risks per million persons exposed are theoretical risks based on (*a*) estimated exposures and (*b*) unit cancer risk values frequently derived from laboratory animals studies—they do not predict cancers that will necessarily develop. This is also the case with the cancer risk estimates developed by the authors of the NRDC/CCA report.

Projected excess cancer risks, expressed per one million persons exposed, are not a meaningful way to express an individual's added risk from exposure to a particular chemical, including DE. A preferred description is one that involves incremental cancer, or the added risk imposed by a particular agent. For example, it is generally recognized that women have a 1-in-3 risk of developing cancer at some point during their lifetime (for men it is 1

in 2). (ACS, 2000). Thus, for women, the lifetime risk of developing cancer is approximately 30 percent, or 0.3. If one were now to add the additional risk that the authors estimate results from childhood exposure to DE from riding a school bus, a child's (female in this case) cancer risk increases from 0.3 to 0.300003 (using the average of the risk estimates). In other words, exposure to DE on a bus for 1–2 hours/day, 180 days per year, for 10 years increases a child's lifetime incremental risk of developing cancer from 30 percent to 30.0003 percent, a percent increase that is vanishingly small.

SUMMARY

The NRDC/CCA diesel-exhaust monitoring study, which constitutes the basis for the report, fails to provide the necessary data and information required for a thorough independent evaluation. Thus, the authors' conclusions pertaining to childhood cancer risk from DE exposure cannot be supported from a scientific perspective. The report discusses a variety of peripheral topics unrelated to DE health risk to children, including alternative fuels, the rise in asthma, and funding mechanisms for diesel-powered bus replacement. Collectively, this range of topics departs from the central purpose of the study—that of evaluating child exposures to DE, an important component when evaluating health risk.

Some occupational studies indicate a weak association between DE and lung cancer under high occupational exposure conditions; however, in other studies, no significant differences were seen between exposed and comparison groups. Health effects reported in occupational settings should not be directly compared or extrapolated to ambient settings, such as exposures on school buses, because the exposure concentrations and durations are significantly different. There are no known studies or case reports that link DE exposure from buses with increased cancer incidence or cancer risk in humans. The conclusions presented in the NRDC/CCA report should be viewed as opinions based on limited data and risk esti-

mations, as scientific evidence is currently lacking to support the claim that school bus exposures are associated with an increased risk of cancer to children.

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