APPENDIX IV

ARB Responses to Comments of the Scientific Peer Review Committee

Report to the California Legislature
Indoor Air Pollution in California
California Air Resources Board
November 2004

The ARB wishes to extend its deep appreciation to the scientific peer review committee for their extensive review of the November 2004 draft Report. The members of the committee included Drs. S. Katharine Hammond, William W. Nazaroff, and Kirk R. Smith, all from the University of California at Berkeley, and Mr. Richard Sextro, from the Lawrence Berkeley National Laboratory. We especially thank Dr. Smith for serving as the Chair of the committee. The committee's comments have improved the Report notably, and will help guide future assessments and actions that may be taken as a result of the report.

COMMENTS:

I. To begin, we commend the California Legislature for commissioning this work. Indoor air quality is an important issue for the health and well being of Californians and the state government can play a significant role in helping to ensure good indoor air quality in both public and private spaces. The topic is not currently well addressed by governments at the national, state, or local level. Consequently, an effort to summarize the state of knowledge and to explore the potential for governmental action is appropriate. California has a proud tradition of leading the country on matters of environmental health, including in the area of indoor air quality.

II. The Air Resources Board has prepared a good draft report. The document assembles a large and diverse body of literature and presents it in a coherent, well-organized, and well-written manner. Particularly impressive are the extensive scope of the pollutant-by-pollutant summaries and the compilation of existing regulations and guidelines.

III. As is generally the case in a scientific peer review, this commentary focuses on aspects of the report that could be improved, rather than discussing the portions that are already in good shape.

IV. In looking over our complete review, below, however, we are concerned that it may be a rather large effort by ARB staff to fully respond. We worry that this would lead to a significant delay in moving forward with efforts in the state to deal with the aspects of indoor air pollution that are well-documented and worthy of action. We recommend, therefore, that the state consider dividing indoor air issues into phases or tiers. In this way, appropriate policy actions can proceed with a first tier, i.e., those pollutants and indoor environments where the evidence is already adequate as a basis for action. Further analysis, needed measurements and other research can be initiated for

contaminants and environments in a second tier. Such division could be made according to judgments of the importance of remaining uncertainties in 1) exposure-response relationships, 2) exposure patterns in California, and 3) effectiveness of available control measures. We would be happy to work with ARB staff in making such judgments or reviewing those made by others.

> **Response:** We agree with the Committee's intent to avoid any considerable delay in moving forward. Consequently, our responses fall into three general categories: 1) immediate changes were made (or will be made in the final version of the report), 2) future research is needed, and 3) comment requires intensive analysis that will not change the report recommendation, but should be carried out in the future.

We start by discussing each of the major pollutants, principally with regard to presentations in Chapter 2 and 3 and then comment on other issues in the executive summary and chapter by chapter. Minor points are collected at the end.

Radon

For the most part, the report relies on the various surveys conducted in CA to assess exposures and to estimate the annual number of lung cancer deaths associated with radon exposure. The overall health effect estimates for CA (p. 90) are done in proportion to the U.S. EPA estimates, adjusting for lower average radon concentrations in the state compared with the US average and correcting for the prevalence of smoking in CA vs. the US as a whole. This latter 'correction' must be done with some care, as the BEIR VI report on which the EPA risk assessment is based only provided risk estimates for two smoking categories - never smokers and ever smokers. Depending upon how many current non-smokers in CA are former smokers, the validity of this adjustment is unclear.

> **Response:** ARB staff consulted with the Department of Health Services, the lead state agency for addressing radon, in preparing these responses to comments on radon. The California radon risk estimate was characterized as preliminary and order-of-magnitude. It was based on an approximate adjustment for different smoking rates in California in recent years. The text was revised to stress the importance of the smoking-radon interaction, and that a truly more accurate assessment would require resources and data not currently available.

In the same paragraph on p. 90, there is a discussion about the estimates being 'worst-case' because "elevated radon regions in California are less populated". The distribution of indoor radon concentrations is irrelevant here, as the risk assessment is based on the average radon concentration, not the underlying concentration distribution. On the other hand, given that only 0.8% of the measured indoor concentrations are above 4 pCi/L in CA, compared with 7% nationwide, the task of determining which regions (and thus populations) are at potentially greatest risk is tractable. To some extent, this has already been done by virtue of the studies in the Sierra Nevada foothills and the Santa Barbara-Ventura counties Rincon.

> **Response:** The text concerning "worst-case" was removed. However, explanation was added to clarify that this estimate is nonetheless very likely an

overestimate, due to more recent data showing substantially lower levels in the Sierras (3% now vs. 25% previously estimated as above the 4 piC/l recommended mitigation level), and other factors as discussed in the revised text.

The report also notes, at p. 90 (3rd para.), that radon in drinking water contributes to the overall risk. However, the report should note that the estimated 168 annual cancer deaths is for the nation as a whole. Furthermore, based on the assessments of radon-in-drinking-water concentrations in CA, one would expect very few of these cases to occur in CA due to the low dissolved Rn concentrations. Note also that these cases are based on public water systems, not wells serving individual homes.

Response: Clarification was added that the 168 is a national estimate.

It is interesting to note that if the overall risk assessment approach were applied to average outdoor concentrations (0.4 pCi/L – which represents the 'lower limit' to radon risk reduction), the expected annual cancer death rate is still ~800 for CA (~600 if the smoking prevalence adjustment is made). According to the EPA radon risk assessment report (EPA 2003c in the ref. List), the estimated lung cancer deaths among never-smokers is 2900 nationwide or ~350 in CA. Adjusting this for the outdoor air concentration (0.4/1.25), the number of lung cancer deaths per annum in CA among never-smokers due to exposures equivalent to those outdoors is ~100.

Response: The text was revised to include an estimate of deaths if indoor radon levels were reduced to outdoor radon levels. It illustrates that, because radon is naturally-occurring and occurs indoors and out, there is a substantial background risk that cannot reasonably be mitigated, other than through reduction of exposure to tobacco smoke.

Finally, with respect to radon risk estimates, there are several places in the report narrative (p. 5, para. 2; p. 28, para. 1; p II-4) that state that radon risks have been reduced and that it is closely associated with smoking. The EPA risk assessment has, if anything, increased the risks associated with radon exposures. The previous risk assessment (circa 1992) yielded a central estimate of ~13,000 lung cancer deaths per year – the current estimate is ~21,000 and the relative risk estimate for never smokers has increased (see Pawel and Puskin 2004). With respect to never smokers, the estimated radon-related lung cancer death rate is 350 per year for CA, which is on par with the estimates for ETS-associated lung cancer. Finally, while there is uncertainty in all risk estimates for exposures to contaminants, radon is the least uncertain. There have been statewide and regional surveys, so the distribution of exposures can be reasonably estimated. The dose-response information is largely based on human exposures – uranium and hard-rock miners at high exposure levels and residential studies at the low end.

Response: As indicated above, ARB consulted with DHS on the responses to comments on radon. The NRC estimates for radon potency (and estimated deaths), on which EPA bases their estimates, have oscillated over the years. Between 1980 and 1987 (NRC's BEIR IV and BEIR V reports), the estimated risk and numbers of deaths were reduced, based on the NRC's reconsideration of differences between uranium miners and the general public, and other factors.

Subsequently, estimates increased, for various reasons. The point is that, despite the agreement that radon is a potent carcinogen, the most knowledgeable scientists have decreased and increased their estimates for radon over the years. Additionally, the estimates for number of deaths in the U.S. has also increased because of improved exposure estimates in high radon states, which is where EPA has focused much of its attention…but California has much lower levels of radon. EPA has changed their interpretation of the BEIR results over the years as well. It is important to note, though, that the majority of the risk accrues to active smokers; it is not spread across the population evenly. Thus, the risk numbers must be considered in light of other factors, such as the inseparability of the risk between smoking and radon, as noted by the NRC in its various BEIR reports. As the panel correctly notes, the risk to never smokers in California is much lower: 350 cancer deaths based on EPA's estimates. Most recently, the NRC (p. 19, BEIR VI report, Health Effects of Exposure to Radon, 1999a) stated that:

1) the deaths from radon-attributable lung cancer in smokers could most efficiently be reduced through tobacco-control measures, in that most of the radon-related deaths among smokers would not have occurred if the victims had not smoked;
2) the committee's…approach to assessing lung-cancer risks posed by indoor radon and cigarette smoking are subject to considerable uncertainty because of gaps in our scientific knowledge of effects at low levels of exposure; and
3) the attributable risk for smoking, the leading cause of lung cancer, is far greater than for radon, the second leading cause.

Specific comments

1. p. 5, middle paragraph. Better to refer to "radium-containing" rock and soil as the source of radon. (Uranium is the ultimate progenitor, but geochemistry can cause separation between uranium and radium, the immediate parent.)

Response: The text was revised.

2. p.20. The cancer risk from lifetime radon exposure, even at 1 pCi/L is remarkably high in comparison with the 1/100,000 used as the NSRL for Prop 65.

Response: Comment noted.

3. p.20. The State of California has frequently developed its own environmental quality standards and guidelines, and these are often more stringent than the federal ones. It is curious that the federal 4 pCi/L guideline has not been seriously scrutinized by CA agencies.

Response: Comment noted**.**

4. §2.3.9. Radon-222 emanates from the decay of radium-226 (not uranium-238). Radon is not directly a carcinogenic hazard; rather it is its short-lived radioactive decay products that accumulate in the lungs and irradiate epithelial cells.

Response: Text changed.

5. Based on the discussion above, the characterization of the risk estimate as "preliminary" on p 160 is unwarranted.

Response: The text was revised to characterize the estimate as a rough, order-of-magnitude estimate.

6. The radon discussion in Appendix II (p. II-4) is inconsistent with the exposure and risk estimates performed elsewhere in the report.

Response: The Appendix was revised.

Volatile organic chemicals

Formaldehyde

Appendix III presents a method for estimating HCHO concentrations in the current building stock. For manufactured homes, an overall reduction factor of 49% is applied to the concentrations measured in the 1980s, on the grounds that manufacturing processes have reduced average emission rates by this amount. However, this reduction applies to new manufactured homes and not to the existing manufactured home stock. A better (more defensible) basis for estimating the changes in indoor HCHO concentrations in these residences would be to estimate the annual rate of new manufactured home construction since the Sexton, et al. survey and combine these with the concentrations in the existing stock. The 49% likely didn't occur all at once, so some sort 'phased' reduction factor should be derived. Some accounting for the removal of older manufactured housing is also necessary, assuming the data are available on which to base an estimate.

The application of the 49% reduction to the peak concentration doesn't appear to be legitimate. Given that the 49% reduction applies to new construction, it is still possible to have high concentrations in older manufactured homes. In addition, since the 49% is an average, there are still likely to be new homes in which low-emitting products were not always used, etc. A better way to estimate the 90 or 95th percentile peak concentrations (not the highest) would be to use the concentration distribution given by the data from Sexton (GM and GSD) and add in an estimated distribution for new manufactured homes. This could be done year by year with the GM adjusted downward to account for the emission changes. As a first estimate, keeping the GSD the same is reasonable. In the end, there will be a new distribution of HCHO concentrations updated to 2000, from which one can derive an estimate for the 90 or 95th percentile peak concentration.

Response: New homes, not older homes, are the primary concern for exposure to formaldehyde. A more complex calculation that takes into consideration reductions associated with the percentage of older homes in California is not considered necessary, because most building materials have completed their majority of off-gassing after about five years. The changes in composite wood manufacturing processes occurred in the early 1980s; as a result, decreased initial off-gassing rates did occur substantially at one time. Manufacturers have

continued to change their processes to a lesser extent, so emissions have declined a small amount since the 1980s, but not enough to warrant a more complex mathematical approach associated with varying ages of housing stock. For the same reasons, a 49% reduction of the peak values measured by Sexton should be representative of today's high-end formaldehyde concentrations. An older house will not have high concentrations of formaldehyde as a result of composite wood materials that had a high initial emission rate 20 years ago.

One issue not addressed by the report directly is how much HCHO concentrations decline with building age due to out-gassing, etc. Some discussion of the aging effect is necessary, hopefully with some quantitative estimate of the decline in HCHO with age. The discussion on p. III-1, para. 6 – that human activities generating HCHO offset the aging effect – is incorrect on its face, unless there has been an increase in such activities with time. Besides, if such activities are important, they deserve their own set of exposure estimates.

Response: A paragraph has been added to describe the observed decrease in home formaldehyde levels during the first five years in new manufactured homes (Sexton *et al*., 1986). An old (1980s) analysis of a small number of Oak Ridge, TN homes showed a distinct drop from years 1-5, relative to formaldehyde levels in older homes; however, while we have the slide based on that study, we could not locate the reference, and so have not yet included that study in the revised report. The report also contains other references to chamber studies and measurements in test homes taken eight to ten months after an initial measurement.

As building materials age and formaldehyde emissions decrease, people's activities have a more significant impact on indoor formaldehyde concentrations. For example, cigarette smoking, use of combustion appliances, the introduction of new clothing and fabrics, and the result of indoor air chemistry will become the major formaldehyde sources (not the building materials). Older homes can also experience substantial increases in their formaldehyde levels when new bookcases or furnishings are purchased, or when remodeling occurs.

Appendix III also presents an estimate for HCHO concentrations in conventional homes, which is based on a sample-size weighted average of two studies. Given the different natures of the two studies, this averaging is not justified. The report notes the limitations of the study conducted in southern CA – limited by the fact that it was summer only with potentially high ventilation rates due to doors and windows being open. The only legitimate comparison that can be made with these data are with data collected under similar conditions in the AZ study – either as a reality check or possibly to add the data from the two studies together for the same seasonal conditions. The AZ study also has its limitations with respect to its application to CA housing and these need to be discussed. While it appears reasonable to argue that construction practices and ventilation conditions may be similar (enough), a probability based sample for AZ will be heavily biased toward houses in Phoenix and Tucson, whose climates may be similar to some areas in southern CA, but not likely the coastal population centers like San Diego, LA and especially the SF Bay Area. Given that both temperature and humidity affect HCHO emission rates, these issues deserve further discussion in the report – perhaps with some emission-rate-based adjustments.

Response: In essence, this comment highlights the lack of recent California data for this topic. Reasons for including the two studies follow. The Arizona study is a recent study, and it is weighted to be representative of the entire Arizona population. To the extent new homes are included, the study includes accurate indoor concentrations for newly constructed homes. It is possible that differences in temperature, humidity, and ventilation considerations relative to California would lead to different indoor formaldehyde levels in California. However, those values are certainly within the same order of magnitude, and probably are within a factor of 2 of the Arizona homes. As far as we know, the study conducted in Southern California is the most recent California study. We felt it should be included, despite its shortcomings as to older housing stock and only summer sampling. As you mention, these facts are explained in the appendix, then a weighted combined estimate is calculated.

With respect to the peak concentration, there is certainly no good basis for adjusting the peak measurement from the AZ study. However, the report is inconsistent in its derivation and use of peak concentrations/exposures throughout. In those cases where sufficient measurements have been made, it is best to use a 90 or 95th percentile approach, which can be estimated directly from the data (if there are sufficient numbers of measurements) or from a cumulative distribution plot. Such an approach reduces the impact of an extreme measurement and provides a more statistically sound means of describing elevated concentrations.

Response: Your comment is well taken. We could leave the Arizona peak result unadjusted. However, not knowing the conditions surrounding that measurement, for consistency we adjusted it slightly downward by a weighted measurement from southern California. The net effect is that we report a peak of 232 ppb instead of 331 ppb (which industry has commented is much too high). Peak concentrations are used in Figure 2.4 to illustrate maximum levels that have been measured; they are used in Figure 2.4 because 90th or 95th percentile values are not available for most of the categories shown. In Table 2.7, on the other hand, 90th percentile concentrations for VOCs were available and are presented. Each is clearly identified.

In the end, given the potential importance of HCHO to indoor contaminant exposures and risks, a well-defined statewide survey of HCHO concentrations would be the best way to resolve these questions.

Response: Although we agree that a well-defined statewide survey of formaldehyde concentrations would provide the best results, such an effort is outside the scope of this report. The California Portable Classroom Study was recently completed, and provides a recent, statewide, comprehensive study of indoor formaldehyde levels. The results are disturbing and compelling: using just the field study results, which were the most conservative measurements, we found that over 4% of the classrooms had formaldehyde levels above OEHHA's acute REL for irritant effects, and all classrooms exceeded the acceptable cancer risk guideline. While a new residential statewide survey of formaldehyde concentrations would undoubtedly be enlightening, the body of knowledge associated with formaldehyde, along with cancer risk estimates, indicate that

formaldehyde is a chemical of major concern in the indoor environment and warrants prompt reduction.

Other VOC/TAC

The review of VOC and TAC is quite extensive in the report. In many respects, however, it presents an undiscriminating treatment of VOCs, even though it is clear that the authors recognize the strongly varying potency among different compounds. In addition to being careful to discriminate among the different compounds that *have been* measured, it is also essential to point out that many potentially important compounds for human health and comfort are not routinely measured. Carslaw (2003) presents a nice (brief) summary of this issue. Weschler (2004) is also a good source for the latest information on indoor air chemistry and its relationship to human health and comfort. (He has referred to this issue under the heading of "stealth chemicals.") Overall, the sections on sources, emissions and concentrations of various VOCs need context, which is provided by health information, and the levels at which health effects occur. In some cases there are reports of emissions that have been measured on VOCs for which no toxicologic or health data are reported; the report should either restrict itself to compounds for which adverse health effects are known or strongly suspected, or at least clearly separate these from compounds with known health effects, so the reader is clear.

Response: The VOC section is complex due to the many pollutants, sources, and health effects that are included within the category. Statements have been added to clarify that many compounds in indoor air are not routinely measured, and reactive chemistry is a new field of research. The report states that by definition there is no safe exposure level to carcinogens; it discusses the concept of exposure and dose, and refers the reader to the OEHHA website for specific information. The report quotes work by Hodgson and Levin (2003) stating that in most cases indoor levels of most VOCs are below health benchmarks such as chronic Reference Exposure Levels, odor thresholds, and irritancy levels (Section 2.3.2.1). However, as noted, some are frequently above acceptable cancer risk levels, and as noted by the panel members, there are many potentially harmful chemicals that are not routinely measured.

The risk estimates for these materials include HCHO, which is already discussed above. For the remaining chemicals, the concentrations and exposures are considerably less certain. The report notes, for example, that with the changes in smoking habits, some of these exposures (e.g., benzene) may be much smaller than that found in the 1992 study (Sheldon et al., 1992a). The overall risk estimate (annual cancer deaths) presented in Table 3.2 (p. 99) of 115 has large uncertainties associated with it (as acknowledged in the report) yet no estimates are provided for either the low or high case – implying a much greater precision on the central estimate than is warranted. At a minimum, one could use formaldehyde exposure as the limiting case (assuming these exposures are more ubiquitous than are exposures to other chemicals).

Response: As indicated in the report and Appendix II, the risk estimates are taken from the Comparative Risk Project. That document did not present the full breadth of information developed during the project, including confidence intervals or other measures of variability for each carcinogen, although such uncertainty was stressed in the text of that report. However, the panel's comment

is noted, and caveats were added in the report and the appendix to emphasize that the values used are estimates with potentially substantial uncertainty.

Specific comments

1. p 20, "architectural coatings." The "low VOC" products were developed to minimize ambient ozone forming potential. This is a very different goal than minimizing toxicity associated with exposure to primary or secondary emissions indoors. The presumption in this document is that these low VOC products have yielded benefits in improved indoor air quality. That presumption requires scientific support, or else it should be presented as speculation or inference.

   Response: Specific information was added regarding ARB restrictions against using certain toxic air contaminants in consumer products.

2. Missing from the discussion in this section are two source categories that seem important: outdoor air pollution and attached garages (and their contents). The only place attached garages are mentioned is on p. 65, second paragraph.

   Response: A discussion of information regarding attached garages was added for both the CO and VOC sections of the report. Some additional information regarding outdoor pollution was added as well; however, the focus of this report is on indoor pollution.

3. Health Effects of Formaldehyde (pp. 58-59). Given the large number of people exposed over the guidelines, as given later, and OEHHA calculations, compare the estimate the number of cancer deaths from formaldehyde with the actual # of nasopharyngeal cancer deaths in CA (shouldn't exceed)

   Response: Between 1998-2002 there was an average of 98 nasopharyngeal cancer deaths per year in California. Our estimate of 62 cancer deaths due to formaldehyde per year compares very well with this figure. The remaining cancers are most likely attributable to occupational formaldehyde exposures. We will add this information to the final version of the report.

4. Indoor Formaldehyde Concentrations (pp 60-61). End of first paragraph: Classrooms and offices should be of nearly equal concern as homes, given the distributions shown in Figure 2.4.

   Response: Text was adjusted in the report to indicate that classrooms and conventional homes also are a concern. However, offices have lower formaldehyde levels as would be expected, due to the different materials used.

5. Page 60, first bullet in 2nd list. Should not equate "emissions" with "concentrations." What does it mean to have "carpet emissions were generally below the limit of detection of 1 $\mu g/m^3$?"

   Response: Text was changed, thank you.

6. p. 67, last paragraph What levels of 2-methoxyethanol and 2-ethoxyethanol were found in these cleaning products? These are the solvents associated with

spontaneous abortion in the semiconductor health study (Swan et al., 1995; Eskenazi et al., 1995); these effects were observed at quite low levels, well under 1 ppm (Hammond et al., 1996). Note that 2-ethoxyethanol is mentioned again on p.70, 3rd bullet; once again, given its demonstrated toxicity, report levels

Response: Only 2-butoxyethanol was found in the cleaning products. Text was clarified and the concentration in the cleaning products was added. On page 70, the compound is 2-ethoxyethylacetate. The reader should refer to the citation for additional information.

Biological Agents

The treatment of "biological agents" in the Executive Summary effectively states the main points. Here are a few items that should be considered in revision:

- The list of biological contaminants (p 9) should also include microbial VOCs and SVOCs, which are often associated with unpleasant odors. (Although the link between odor and health is not strongly established, odor has well-accepted historical legitimacy as a basis for air pollution control measures.)

  Response: Although firm data associating indoor microbial VOC exposures with human health effects is not available, the reviewer's comments relative to malodors, especially considering individual variation in sensitivity to odors, are reasonable. MVOCs will be added to the list of biological contaminants. However, evidence for potential health effects from microbial SVOCs is too preliminary to include in this document.

- Transmission of infectious disease and exposure to pathogens from poorly maintained ventilation systems merit attention, as noted. In addition to the points made in the executive summary (p 9), it might be worth noting concerns about indoor air transmission of emerging infectious agents, as evidenced by the recent SARS outbreak. The broader relationship to security concerns and possible exposure to biological agents (e.g. anthrax spores) may also be worth mentioning.

  Response: Added SARS as an example of an emerging infectious agent. A broader discussion of indoor exposure to bioterrorism agents is beyond the scope of this document.

- Table ES-2 (p 11) lists the total costs associated with "ETS: asthma episodes" as 0.001 billion $/y and associated with "mold and moisture: asthma and allergies" as 0.22 billion $/y. It is not credible that the cost of mold and moisture-induced asthma is 200 times that of ETS-induced asthma. See further discussion below.

  Response: The medical costs for ETS-induced asthma were revised upward. Additional detail will be added in the future to include the explanation discussed below regarding potential underestimates.

  Because of differences in the types of data available, the original cost estimates in Section 3 and Table ES-2 for asthma due to ETS exposure and to mold-moisture exposure were calculated using different methods. The medical cost estimates for ETS used a unit cost for a doctor's visit ($42) multiplied by the

number of children with an asthma episode due to ETS exposure. The ETS estimate does not include adults, and does not include mortality, because such data are not available. Mortality cost estimates were not made for ETS-induced asthma because the ARB/OEHHA (2004-2005) risk assessment did not include premature deaths in its summary table for health. For the mold-moisture estimates, the medical and mortality costs were derived by taking a fraction of the total U.S. costs (direct and indirect) for asthma, and multiplying that by the fractions of asthma attributed to mold and moisture.

In the revised estimate, the medical cost estimates for ETS-induced asthma were revised upward using a unit cost of $640 (1990 dollars), the per capita cost of asthma in the U.S. (direct and indirect) cited by Weiss and Sullivan (2001). The mold-moisture estimate is now 10 times greater than the cost of ETS-induced asthma episodes. This remaining difference between the ETS and mold-moisture total estimates for the medical cost of asthma is due to the following:

- The attributable fraction of asthma due to mold-moisture is higher than that for asthma due to ETS (13% vs. 3.5%). The ETS value is taken from ARB/OEHHA (2004-2005).
- The ETS estimate is for children's cases only, so it does not include the substantial indirect costs for lost work days, as shown in the cost estimates by Weiss and Sullivan (2001).
- The direct cost for treating children is not fully reflected in the ETS estimate; the unit cost is based on the population average cost in the U.S., but children may require more medical care than adults.
- The unit cost used for the ETS estimate has not been adjusted from 1990 to 2000 dollars.

Biological Agents: Chapter 2

- In Table 2.1 (p 33), consider adding “building occupants” to the “major indoor sources” column for “biological agents.” Certainly this is the major source category for infectious disease transmission. Another important source is “infestations” by cockroaches.

  Response: The recommended additions were made.

- The underlying reason for the increase in asthma is not well understood. It is misleading to suggest that indoor and outdoor air pollution are understood to be important causes. The opening paragraph on p 34 creates an inaccurate impression.

  Response: No change was made relative to this comment. The sentence in question (last sentence in the paragraph) was worded carefully, is already qualified, and is well-supported by literature citations. We believe the panel may have misinterpreted the statement, which reads “…indoor and outdoor air pollution have been identified as potentially important contributors to the increase of asthma.” Nonetheless, we will clarify this statement in the final version.

- Care should be taken to distinguish information on asthma initiation (becoming an asthmatic) from information on asthma exacerbation (attacks in asthmatics). Different pollutants have different impacts, which in turn have different implications

for age distributions, burdens, and policy. If a study being examined does not provide information allowing us to tell whether the effect was on initiation or exacerbation, it should be so stated in the discussion.

Response: We agree with the panel's preference, and that is why both Tables 2.2 and 2.3 (from IOM 2000) are included in the report. Due to time constraints, we were not able to revisit all papers throughout the report that discuss asthma, but will make edits accordingly in the final version of the report.

- The specific reference to Tables 2.2 and 2.3 from the IOM report is appropriate. However, it is important to note that the IOM report only found sufficient evidence of a causal relationship for several factors: house dust mite (for development and exacerbation), ETS (for exacerbation among preschoolers), and cat and cockroach (exacerbation). The text on p 34 overstates the case of causality: "The committee found that, in addition to the known biological asthma triggers such as mold, house dust mites, and animal dander, chemicals such as … *can exacerbate* asthma in sensitive individuals." (Emphasis added here.) This sentence strongly suggests causality, but for molds scientific evidence only supported a finding of an association, rather than causality.

  Response: The original sentence combined the results of several categories in a summary sentence, as the first sentence in a paragraph, followed by a specific discussion of each trigger, which is very explicit. The first sentence has been revised. Molds were included in the introductory sentence because the focus of that clause was biologicals vs. chemicals.

- In addition to the IOM, a large review was recently published in the EU related to this topic (Bornehag et al., 2004). A key conclusion: "Dampness in buildings is a risk factor for health effects among atopics and non-atopics both in domestic and in public environments. However, *the literature is not conclusive* in respect of causative agents, e.g. mites, microbiological agents and organic chemicals from degraded building materials." (Emphasis added here.) Given the existence of authoritative reviews by scholarly committees such as this EU review and also the IOM reports in 2000 and 2004, it seems inappropriate to give comparable weight to the review findings of a single scientist (Delfino, 2002) (p 35-36).

  Response: Reference to the Bornehag review was added, including the absence of an identified specific causative agent in damp buildings. The EuroExpo review (basis for the Bornehag et al. article, 2004) specifically sought studies that evaluated building dampness and health effects. Thus their conclusions that damp buildings increased the risk for respiratory symptoms in non-atopics and asthma exacerbation, but the underlying agent in <u>damp</u> was not identified. The Delfino review included a different population of studies looking at the relationship between indoor airborne chemicals and asthma regardless of moisture problems in the building.

- The discussion of Sick Building Syndrome (SBS) relies on relatively old literature of limited scope (p 41). More recent investigations that add substantially to the literature include Mendell et al. (2002), Seppanen and Fisk (2002), and Wargocki et al. (2002). While the causal connections with pollutants have not been elucidated,

Mendell et al. summarize the state of understanding for biologically plausible connections. All three of these papers make a strong case for associations between ventilation system deficiencies and SBS symptoms. The present review should incorporate some of this more recent evidence.

Response: The suggested references were added to the discussion of Sick Building Syndrome.

- Section 2.3.4 begins by listing biological contaminants (p 74). Properly, one should refer to fragments or excreta from house dust mites and cockroaches, rather than the entire organism. (Also applies at bottom of p 75.)

  Response: This additional detail has been added.

- The discussion of health effects of biological contaminants (p 74-81) relies heavily and appropriately on the recent IOM (2004) review. Overall, this section makes its major points effectively and accurately. It could be further improved in some specific details, as described here:
  - ? It is stated (p 75) that "colds are more often transmitted by direct contact." In our reading of the literature, the mode of transmission of rhinovirus is not well known. The statement should be supported by a reference to an authoritative source, or removed.

    Response: Text was changed to acknowledge that the mode of rhinovirus transmission is not fully understood.

  - ? A conference paper by Myatt et al (2002) is cited in relation to communicable disease transmission (p 75). A recent peer-reviewed journal article by the same investigators is a more scientifically compelling source (Myatt et al., 2004).

    Response: Updated reference included.

  - ? In discussing toxic responses to "damp or moldy" buildings (p 75), endotoxins and mycotoxins are listed as possible causative agents. This presentation leaves the impression that the problem is clearly of biological origin. The literature leaves open the possibility that the underlying cause of health problems in damp buildings is chemical, rather than biological. This point should not be lost in the presentation of evidence. (This is done well in a paragraph on p 77.)

    Response: The text states that exposure to toxic bacterial and/or fungal elements in indoor environments is controversial and not proven to be linked to human health effects.

  - ? Section 2.3.4.2 (p 75-77) discusses "sources" of biological contaminants. The subsection on house dust mites should note that the allergens are carried on excreta and body fragments that may become resuspended by simple indoor activities such as walking and cleaning. The discussion of cockroaches does not present information about sources. The review should

briefly explain what is allergenic about cockroaches and how exposure might occur. Similarly, the discussion of viruses does not say anything about sources of viruses.

Response: Clarification of mite excreta and body fragments as source of allergen has been added. Comment on potential for mite allergen resuspension is not included due to the rapid resettling rate and the large size of resuspended particles (IOM, 2000, p 139). Additions to text were made to augment sections on cockroach and viral exposure.

? On p 79, a statement is made that "healthy children in damp or moldy buildings sometimes report having more respiratory infections…." This statement requires a reference to an appropriate study; otherwise, it is inappropriate in a scientific review.

Response: Sentence revised and literature citation added.

? Unless you really think pulmonary hemorrhage is a concern (in which case more support is needed), drop the sentence appearing at the end of the 3rd paragraph, p 79.

Response: The sentence will be dropped or additional support added in the final version of the report.

- Section 2.3.4.3 (p 77) opens with the statement that indoor mold is experiencing "increasing occurrence at problem levels." This statement requires supporting evidence.

  Response: It is based on the great demand for mold inspection and remediation services that did not exist 10-15 years ago, and the increasing number of calls received from the public by state agencies regarding very serious mold problems (extensive visible or hidden mold) in homes in California. Mold was hardly mentioned at ARB's 1994 indoor air quality symposium, yet at a similar symposium in 2000, it was the unintended focus of discussion. However, we will revise the sentence in the final version.

- The discussion of "mold concentrations" (p 80-81) makes the important point that objective measures of moldy indoor conditions that would pose health problems are lacking. If indoor mold is deemed a high priority issue (as listed in Table 6.1, p 147), then research is likely warranted on methods of diagnosis.

  Response: The need for additional research in mold environmental assessment methods was added to the recommendation for further research.

Biological Agents: Chapter 3

- The review determines that 13% of asthma cases are attributable to residential mold and moisture-problems/dampness. The basis for this determination is an analysis of four large studies, as summarized in Table 3.4 (p 105). This 13% finding is important in the context of this review, as it drives the estimates of cost associated with

mortality (Table 3.2, p 99) and morbidity (Table 3.3, p 103). Several concerns arise that should be considered in revising the draft:

? The estimated asthma costs for ETS is $1.3 million per year, less than 1% of the estimated cost of asthma for mold & moisture ($190 million per year). It simply seems implausible that the true difference in costs could be so large, given these facts. (1) Epidemiology is the basis for both ETS and mold/moisture to be investigated as "causes" of asthma. (2) The prevalence of ETS exposure (e.g. in one's residence) and mold/moisture exposure is comparable in magnitude (~ 10-30%). (3) The odds ratio for asthma to be associated with mold/moisture is small in epidemiological terms (1.3-1.6). The differences should be reviewed and either the estimates revised or the differences explained.

Response: See the response above, under Biological Agents.

? In presenting cost estimates for mold/moisture, the implication is that there is a causal association. (This is what is implied in the term "attributable.") Yet IOM (2000) could only conclude that there is an association between exposures to fungi/mold and exacerbation of asthma (Table 2.2, p 35).

Response: Subsequent studies have strengthened the link between mold/moisture and asthma. Clarification of any causal association will be added to the report at a later date.

? The studies cited in Table 3.4 are not specific to California. Are construction and climate conditions sufficiently similar to justify the direct application of these studies to estimate conditions in California? Even if so, a caution should be added.

Response: The text was modified to explain the applicability of these studies to California conditions.

? The presentation states (p 106) that the estimate "does not include the costs of other indoor allergen sources…" Please confirm that there was careful control in all of these studies on all other allergenic agents. Mold and moisture would tend to indicate poor operation and maintenance and this would likely correlate with the prevalence and levels of other allergenic agents.

Response: Due to time constraints, those studies will be reviewed in the future and the report will be revised if needed to add any necessary clarification.

? At the top of p 106, the figure $24 million should be $240 million.

Response: Thank you, the text was changed.

- The discussion of the potential for control of moisture and mold problems (p. 106) is inappropriately simplistic. Yes, of course, in principle mold and moisture problems can be controlled. We can also build a fleet of motor vehicles that don't emit

excessive pollutants, and we can eliminate smoking-related diseases by having everyone quit smoking. But these feasible solutions are very challenging to implement in part because they rely on informed action by large segments of the population. Any discussion of control should acknowledge the real and substantial challenges. Specifically, the statement that it is "probably feasible to eliminate at least 50% of the particle exposures that contribute to asthma" (p 107) is unsupported speculation and should be removed.

Response: We did not intend to be overly simplistic, but rather, brief. However, we do believe that preventive maintenance and proper attention to building operation and maintenance are more readily achievable than some other resolutions to indoor pollution that require new technologies, for example. The examples cited above…smoking cessation (where people are addicted), and a new fleet of low-emitting cars (where new technologies, and actions by both manufacturers and the public must all be implemented) would in our view be in a different, more long-term category, because they are more difficult to achieve.

- There is not sound scientific evidence to support the claim that biological particles are the dominant source of the adverse health effects associated with dampness and mold. (Furthermore, that statement is not necessary in the discussion here.) (p 104)

  Response: One paragraph was revised to remove reference to biological particles. Other similar references in the document will similarly be revised before the report is finalized.

Environmental Tobacco Smoke

Health Effects of ETS

The report has properly listed some of the most well known effects of ETS, e.g., lung cancer and heart disease, but some of the others are dealt with less systematically; some, e.g. SIDS, asthma induction, are mentioned in one section, but are not included in the calculations of the Costs of Indoor Air Pollution (Chapter 3). The California EPA has produced two excellent reviews of the health effects of ETS, *Health Effects of Exposure to Environmental Tobacco Smoke*, 1997, and the recent update, *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant* SRP Version, October 2004. The information in these reports should be incorporated into this report, at least as a complete summary and with references to the report. We suggest a table of these health effects, including the estimated number of Californians affected by each disease (with upper and lower confidence estimates where available); this has already been done in the Cal/EPA report on Proposed Identification of ETS as a TAC, October, 2004. This document on Indoor Air Pollution should be congruent with the two documents cited above. Note that the effect of ETS on heart disease has a very profound public health impact, which deserves more attention than has been given in this report.

Response: The report summarized and cited the information from the October 2004 Part B report by OEHHA. This information has been updated using the more recent OEHHA estimates, and a table with OEHHA's case estimates attributed to ETS exposure has been added to the ETS Health Effects section.

The text and estimates will be further revised as appropriate after the Scientific Review Panel has approved the ARB/OEHHA ETS document.

Exposure to ETS

Environmental tobacco smoke is a major indoor contaminant/risk factor in CA for which the exposure estimates currently are substantially uncertain. The report notes this, but spends considerable time describing studies conducted before smoking was banned/reduced (pp 71-74). For the most part, these studies have little quantitative relevance today. Not only have workplace and public (e.g., bars and restaurants) ETS exposures been reduced to essentially zero, surveys indicate that smoking behavior in homes is being changed to reduce ETS exposures to non-smoking members of the household. ETS exposures of nonsmokers should also be adjusted to account for the observation that cigarette consumption in CA is about half that of the US.

Response: We concur that smoking is nearly gone from California's public buildings, and that fewer people are allowing smoking in their homes. Nonetheless, there are still significant exposures to ETS in some buildings in California. Older studies and studies of ETS in casinos and bingo parlors were included in the report for completeness, because some California environments continue to allow smoking. Some new casinos in the state allow smoking, and there remains a percentage of homes and other environments where smoking is allowed. Children are especially at risk of being exposed in homes and cars if an adult in their family smokes and does not avoid smoking around the children.

It is unclear how the panel's comment on ETS relates to the pages indicated, which discuss VOC levels in homes, not ETS. However, if the concern is that the VOC levels are outdated, cigarettes are not the primary indoor source of most of the VOCs discussed. Only the benzene measurements (and to a lessor extent xylene and toluene) would be expected to be substantially different in homes now. Additionally, the VOC study indicated was the last large, population-based indoor/personal VOC study to be conducted in the state, and thus warrants discussion. The Energy Commission is funding, and ARB is managing, a study of indoor air quality, including VOCs, in new California homes, which should provide more current information.

ETS: Chapter 3

Table 3.1 of the report lists the Unit Costs for Health Effects; these have been treated unevenly. At the very least, the costs of visits to physicians should be included, as these have been well studied. As an example, Table 6.11 of the document *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant* reports that there are over 50,000 ETS attributable office visits for otitis media for children under the age of 3; even at $100 per office visit this comes to a cost of over $5 million. There are ways to incorporate also the lost work time due to having to take the child to a doctor. Similarly, Table 6.09 lists doctor consultations for respiratory symptoms, and reports a 15% increase if a smoker is in the household, and a 38% increase if two or more smokers are present. More seriously, young children have over twice the risk of developing bronchitis or wheezing if they are exposed to a pack or more a day (Table 6.08).

Response: The text and tables have been revised to include unit costs and total cost estimates for all but one of the health outcomes with estimated case numbers from Table ES-2 of the OEHHA ETS risk assessment. Otitis media (middle ear infection) was added. The unit cost for asthma cases has been increased to $640 per year, based on national per capita averages, as discussed above. Respiratory symptoms and bronchitis are not included in this table, so cost estimates were not developed. SIDS was not included in the cost estimate because cost estimates were not available. Assuming that each case of premature death from SIDS would have a valuation of $6.3 million, the cost would total about $130 million. This amount would not significantly affect the total cost estimate for ETS (over $25 billion in Table 3.6).

The cost of premature delivery was not estimated separately because it overlaps with the costs of low birth weight to a large degree. Caveats were added to the text for the additional costs of premature delivery that could not be quantified currently.

How was the cost for low birth weight children ($118,000 per case) derived? How low birth weight? Most ETS related low birth weights are small decrements in birth weight—are these the number of cases of babies born with a weight under a given weight, or with a statistically significant lower birth weight?

Response: The cost is based on U.S. EPA's Cost of Illness report, as summarized in Section 3. U.S. EPA compared the costs for low birth weight infants (less than 2,500 g) vs. other infants. OEHHA (2004) attributed a 20-100 g decrement in birth weight due to ETS exposure, and a downward shift in the distribution of birth weights. The final report will clarify this discussion.

Parts of Table 3.3 seem inconsistent. For example, the medical cost for ETS asthma episodes is estimated at $42 each, yielding an estimated cost per year of $1.3 million, while asthma costs related to mold and moisture are estimated over 100 times greater--$190 million. Does this really make sense? What about emergency room visits for asthma attacks? Different methodologies may have been used to derive these numbers (see page 106), but they need to be reconciled when they are compared and contrasted, as in this table.

Response: The text and tables have been revised, as discussed above.

ETS risk assessment

The basis for the risk estimate in this report (p. 100) is from OEHHA, based only on spousal smoking (incorrectly referred to as 2004 – should be 2003c), although this has apparently not yet been peer reviewed by the external review panel for OEHHA.

Response: The text and tables have been revised to reflect the latest OEHHA risk assessment, which has been revised in response to the panel comments and is still under review.

The risk estimate given on p. 100 is based on estimated US lung cancer rates, re-scaled for the CA population fraction, corrected to the year 2000. No correction was made for

the differences in smoking rates, although the data are discussed in para. 5 and the same data are used to correct the risk estimates for radon (described earlier). This would reduce the central estimate to ~ 275, based on the estimated number of smokers. An even lower estimate would result from the observation that cigarette consumption in CA is about half that of the US.

> **Response:** Changes, clarifications and caveats will be to added to the report at a later date to reflect the latest OEHHA risk estimates and with the radon approach.

Table 3.2 (p. 99) surprisingly does not provide upper or lower bound estimates for annual lung cancer cases associated with ETS. Clearly such bounds are necessary, as the current estimates are misleading as to the precision of the central estimate. At the lower end, it is possible that behavior modification by smokers in their homes has reduced non-smoker exposures. At the upper end, estimates of ETS exposure based on nicotine measurements may be underestimated, based on recent work reported by Apte, et al (2004). This work demonstrated that for situations where the interior surfaces are not chronically exposed to ETS, nicotine sorbs to these surfaces more rapidly, thus leading to lower measured nicotine concentrations in the air (and hence, lower ETS-RSP exposure estimates).

> **Response:** The text was revised to show the range in the latest OEHHA estimates. OEHHA used the lower bound because it was based on the best study available.

Particles (PM)

We were concerned that the health effects of indoor PM were not discussed in detail. Although PM epi has not been done with indoor-generated PM (IPM) per se, there is little reason to think that IPM is somehow not a health hazard, given its sources (combustion, for example). And, if less hazardous than outdoor PM, it is unlikely to be so by a large factor. Extrapolating from outdoor PM to IPM is less uncertain than extrapolating, e.g., from high dose-rate studies to low dose-rate environmental conditions, or from animal studies to humans.

This raises a variant of the drunk-looking-under-the-streetlamp principle, i.e., it is a bit of an anomaly that we have such excellent and extensive work on outdoor epi. It is simply because, unlike most risk factors, a small number (often just 1) of existing measurement stations can be used to characterize reasonably well the changes in exposure to huge populations with reasonably good health records for important outcomes. The area illuminated by this strong streetlamp, however, should not be confused with the area where the biggest impacts from PM exist (which include different locations, populations, and diseases). With appropriate care, we should attempt to extrapolate the results to the darker parts of the street (e.g., indoor PM, vulnerable populations, and, even, end points not usually examined, childhood pneumonia, for example, as done in the WHO Comparative Risk Assessment (Cohen et al., 2004). Such kinds of extrapolations are already accepted as necessary and useful from occupational settings and animal studies to indoor environments, arguably larger stretches than from outdoor PM to IPM.

If CARB were to extrapolate outdoor PM epi to indoor concentrations from indoor sources, even if there were to be some discounting due to different particle mixes, the results are likely to be much larger than the relatively small contributions to frank health risks from VOCs. Given that it is based on epi with similar populations and exposure levels (and not animals and high-exposure occupational settings), it would likely be more convincing as well to most observers.

> **Response**: We agree that indoor PM assuredly has large impacts on health. Accordingly, the November 2004 report included a full three-page discussion of the health effects of ambient PM (more than any other pollutant in the report), because there is no such body of information for indoor PM. That was followed by a discussion of the likelihood that indoor PM has similar effects. In response to the panel's comments, we have added additional studies and text to bolster those sections of the report. However, as discussed in the report, ARB convened a panel of expert scientists in indoor PM in February 2004 to advise us regarding the conclusions that can be drawn at this time regarding the health impacts of indoor PM. They concluded that there is insufficient information on which to base any type of quantitative estimate of the health impact of indoor PM. Additionally, a European group conducted a similar assessment, and came to a similar conclusion (Schneider et al., 2003). We also consulted with the OEHHA's PM staff, who concurred with those conclusions. Thus, we are not able to include a quantitative estimate in this report. However, ARB is in the process of funding research into the health impacts of indoor-generated PM, beginning with indoor combustion emissions, and we hope to begin to shed some light on this critical area very soon. We encourage others to pursue this area of research as well.

Indoor and Personal PM Concentrations

The report overstates the similarity between indoor and personal PM, especially if one examines CA data as presented in Table 2.5. Thus, the third line of the report inaccurately states that personal exposures "often exceed both indoor and outdoor concentrations." However, examination of Table 2.5 for PM2.5 reveals that while this is true for studies in Boston, Detroit, and Baltimore (Midwest and east coast), it is less true for studies in California. In the Suh 2004 and the Linn 1999 studies in LA and the Evans 2000 study in Fresno, the personal exposures were less or comparable to the outdoor concentrations; only the Suh 2003 study of COPD subjects in LA found personal exposures greater than outdoor concentrations. Similarly, the report should note that, while Boston and Detroit had *higher indoor* than outdoor concentrations of both PM10 and PM2.5, California cities generally had *lower indoor* concentrations for PM10 and PM2.5 than ambient concentrations.

> **Response**: Changes were made to the text to clarify these relationships, and additional edits may be made in the final version of the report. However, the statement that personal exposure levels are often higher than both indoor and outdoor concentrations is correct, for the general population and for certain seasons. Additionally, personal exposures are more often correlated with indoor concentrations than outdoor, particularly in population-based studies, although a few studies have found stronger correlations with outdoor PM. Study design, season, and study population are all key characteristics that affect the PM levels measured. The PTEAM study, a large population-based study of the general

population conducted in Riverside, found a much higher personal PM exposure levels relative to indoor and outdoor levels, which were similar. Since PTEAM, most studies have improved the study design (by using a longitudinal rather than cross-sectional design), but have focused on sensitive populations (many elderly or with compromised health), rather than the general population. Members of sensitive groups often have different activity patterns than the general population: they may be less active, and conduct fewer activities that generate indoor PM, and they may operate their homes differently (more or less window-opening), resulting in lower personal exposures and indoor concentrations than healthy families with active children and pets might generate. There are also distinct seasonal differences in the relationship among indoor, outdoor, and personal concentrations, and also differences between PM2.5 relationships and PM10 relationships: these are both reflected in the Fresno data, for example. Finally, it should be noted that the Suh studies (2003 and 2004) both involved very small sample sizes…about a dozen homes in each (the sample size indicated is the number of daily measurements…this will be clarified in the final report).

We have several suggestions for Table 2.5

- The values reported should be geometric means or medians, not means, which can be elevated by one or two high values.

  Response: Some studies did not report geometric means, so arithmetic means are shown. Additionally, the arithmetic mean is more reflective of the entire distribution, including the high end values that may represent the most impacted portion of the population.

- The entries should be grouped first by regions of the country
- All studies in one city (e.g., LA, Boston) should be grouped together to ease comparisons

  Response: We are not attempting to compare indoor concentrations by region of the country. Season, urban vs. rural location, subject type (COPD, etc) and activity level, and several other factors are at least as important, if not more so, in determining indoor PM concentrations. Regional groupings might be easier for identifying California studies, and may be provided in the final version of the report; however, studies of indoor PM are sufficiently new that studies in other regions of the country remain very informative.

- Some measure of the dispersion of the data should be included (e.g., SD, GSD)
- The number of samples should be reported

  Response: Done.

- Residential data should be separated from office building data
- Include the other office building data mentioned on page 50

  Response: The office building data from the BASE study are now shown at the bottom of each table. The other two studies are not included in the table because they were conducted in the early to mid-80s, in buildings where substantial

smoking occurred, and are not particularly relevant to current conditions in California.

- Where possible, report data separately for buildings where smoking did and did not occur during the sample collection

  Response: Clarification has been added in the text that most of the studies shown in the table are from homes or buildings where little or no smoking occurred, most often because of the health problems of the participants. One exception is the PTEAM study, in which a very small percentage of homes were smoking homes. A footnote will be added to the table in the future.

Other points:

? p. 49 top: Spengler et al. reported measurements of personal, indoor, and outdoor PM sampling long before the PTEAM study—we think it was the 6 City data reported in the 1980s

? p. 49 3rd paragraph, 6th line:, rather than "for each of the three groups," actually,, for 3 of the 4 groups for outdoors, and all groups for indoors

? p. 49, 3rd paragraph, 6th and 7th lines: the data on PM10 personal are missing from the Tab le—please include

? p. 50 2nd line: 19.6 here but 19.5 in table—reconcile

? p. 50, 8th and 9th lines: a percentage is missing

Response: Corrections and additions have been made, thank you.

? p. 50, 2nd paragraph: The data discussed here are quite different from other comparable data, where the personal exposures were comparable to the indoor, and less than the outdoor; the discussion is therefore misleading

Response: Comment noted.

? p. 50 3rd paragraph, line 8; the indoor concentrations reported should be compared to outdoor concentrations. Did the BASE study uniformly exclude buildings with smoking (we think it did not)? This should be considered in it evaluation here.

? Do we have data on smoking for any of the home studies reported? If so, report whether smoking occurred during sampling, and separate reports from homes with smoking from those without smoking and place on adjacent lines

? The last two points may lead to an observation along the lines that smoking is a major contributor to indoor PM. The Spengler study alluded to earlier also provides data on this point, and estimates an increase in PM of about 1 ug/m^3 per cigarette smoked per day, or 20 ug/m^3 per pack.

Response: Information from Spengler et al was added in the PM source emissions section. Where smoking occurs, it is indeed the greatest contributor to indoor PM, as shown in many earlier indoor PM studies not included in Table 2.5. However, as mentioned above, most of the studies in Table 2.5 had little or no smoking in the homes during monitoring. We will review these studies and

determine whether there is sufficient additional information to warrant separation of smoking and non-smoking locations in this table.

Carbon Monoxide (CO)

- Page 52. It seems unlikely that the epidemiological studies cited here determined CO *exposures* with a high degree of accuracy.
- Owing to indoor sources and also variable proximity to vehicular emissions, one doubts that ambient CO is a good proxy for CO inhalation exposure.

  Response: The California CO study was a study of death certificates, not an epidemiology study, where CO poisoning was the known cause of death. Ambient CO was not measured or discussed in this study. Rather, blood CO levels and the decedents' immediate history were used to assign a cause of CO poisoning in the death certificates.

- First two lines on p. 52 state natural gas, propane and kerosene fuels add to > 99%, but motor vehicles "also took a substantial toll" (31% in figure 2.2)

  Response: The summed percents relate to the preceding sentences, which discuss the percent of the indoor deaths attributable to the different types of indoor sources. Those indoor sources contributed about 50% of the total deaths due to accidental CO poisoning, as stated in the preceding sentences. Additional clarification will be added to the final version of the report.

- How many homes had indoor CO greater than the state standard of 20 ppm?

  Response: It is unclear which study this comment is referring to. The CO mortality study did not involve measured data, as indicated above. In the Wilson et al study, several homes had levels over 20 ppm; we will insert the actual number of homes in the final report.

- Page 53, §2.2.4.3. The first line ("most homes have relatively low CO levels") begs for support. CO levels in the wintertime may often be persistently elevated owing to high ambient levels (e.g. in Los Angeles) and to the use of improperly vented heating devices (e.g. in the mountains).

  Response: Our statement is correct and well-supported. In the two large California studies cited (Wilson et al 1993 and Sheldon et al 1993), both of which included winter data and sample sizes of 277 and 280 homes, respectively, neither showed more than a few homes with CO levels above health-based standards. Ambient CO levels have decreased sufficiently in Los Angeles that there are very few winter excursions above the ambient standards.

- Perhaps move figure 2.2 and the discussion of types of appliances to section 2.2.4.3, Emissions.

  Response: We believe it fits best with the discussion of the results of the mortality study, but the information from that study could be split as suggested.

- The paragraph that refers to Springston et al. (2002) should point out that these were commercial buildings (in which combustion appliances might have been absent altogether).

  Response: We will check that for the final version of the report.

- Related to vehicle emissions, probably more important than CO in ice rinks is accidental poisoning deaths associated with idling motor vehicles in attached garages. See Marr et al. (1998).

  Response: We have added a section on attached garages to the report.

- Page 98, §3.1.1. We see no rationale for excluding CO poisoning deaths from motor vehicle emissions as many of these occur in indoor spaces. See Marr et al., 1998.
- Page 98, middle. A better reference (than CPSC, 1997) to the recent state of accidental CO poisoning deaths nationwide is Mott et al., 2002.

  Response: In the California CO mortality study, vehicle-related CO deaths often occurred outdoors, under circumstances such as individuals trying to warm up the car (using the heater while idling a long time), and many were associated with alcohol use. The statistics on the number of deaths that occurred in an attached garage were not readily available, and would likely add very little to the total CO mortality estimate.

Pesticides

Pesticides are substantially discussed in the report, but notably absent from either the "high priority" or "medium priority" source lists for mitigation. The omission seems inappropriate. The underlying reason for the omission is unclear.

Response: Pesticides were added to the medium priority sources for mitigation.

Health effects section (p. 81, para 5 ff) on pesticides is weak. More CA data on pesticide concentrations should be available now from the ongoing studies by Pat Buffler and Brenda Eskenazi at UC Berkeley, however.

Response: Information was added to the pesticide health effects section. However, time did not permit consulting with the investigators suggested above.

Non-industrial Workplace Exposures

With respect to workplace exposures, the report focuses mostly on office exposures, and these are treated well. However, there are other non-industrial workplace exposures that should be addressed. Brief mention is a made of a few of these occupations (janitors, barbers, and beauticians), however, a more systematic effort is needed to address the exposures of these workers, and to evaluate other non-industrial occupations with important exposures, e.g., auto repair shops, indoor construction work. This would be, however, an enormous task, and perhaps the scope of the report should

actually be restricted to home and office exposures, although the original charge to ARB was broader. A clear statement is needed at the start of what is covered.

> **Response:** We realize the information on non-industrial workplaces was weak. Thank you for acknowledging that inclusion of this information could be "an enormous task". We have included a section, 2.4, to highlight some of the exposure information that is known regarding non-office, non-industrial workplaces. While not a robust assessment, the new section provides a reasonable handle on the scope of the many indoor air quality issues that arise in non-industrial workplaces in California. Additional information may be added to the final report.

COMMENTS BY SECTION

Executive Summary

As the summary stands now, a reader cannot get a good overview of either the health outcomes or of the sources.

The report contains a large amount of data on a very broad topic. However, it does not synthesize the data well. Thus the second bullet states 230 excess cancers occur from indoor carcinogens, yet that excludes those from ETS, radon. The authors should choose a template to summarize the data, either by health outcome or by source—currently it is mixed, and not all the data are presented. To the degree possible, numbers should be presented for all of the outcomes for all of the relevant exposures (e.g., numbers of lung cancers attributable to VOCs and radon, but not ETS, are presented; no numbers are presented for the number of coronary heart disease deaths and cases attributed to indoor air problems, yet this is likely to be a much larger number).

> **Response:** It has been a challenging task to organize the information in this report. We have attempted to organize information by pollutant with Table ES-1 serving as a summary for what is known about the pollutants, sources, and associated health effects. Methods for mitigation are organized by source categories (Table ES 3.1 and 3.2) because manufacturing changes have the potential to address multiple pollutants (if more than one is present) associated with a given product. The numerical estimates for the major health endpoints were specified throughout the summary.

Page 6, penultimate paragraph. The reference to ambient PM mortality should not refer to an association with "exposures" but rather with "concentrations." (It is very important to be precise in distinguishing among all of the parameters of concern.)

> **Response**: Statement corrected, thank you.

Page 8, first paragraph. Worth mentioning here that ozone produces PM as a byproduct as well.

> **Response:** Text now includes PM.

Chapter 4 Existing Regulations

The discussion of current regulations, guidelines and practices seems very complete and the review committee did not identify any major omissions. As a general observation however, the discussion presented in this chapter is not always careful to note that many of these regulations, etc. are explicitly focused on outdoor air quality and only secondarily – if then – on indoor air quality.

> **Response:** The text was revised.

Transference of standards based on outdoor epi to indoor air should be done with caution. Outdoor epi is done by looking at changes in health based on changes in outdoor levels, not changes in exposure. Since most people spend most of their time indoors and the indoor concentrations of outdoor pollutants is less than they are outdoors, the real impact per unit exposure is higher. Consider, for example, that a change of 50 ug/m^3 of PM causes so much ill-health in the outdoor epi. If the penetration level is 80%, however, it is actually showing the effect for only a 40 ug/m^3 change in exposure. Indoor levels are much more closely related to real exposure, however, and so, arguably the same amount of ill-health represented by 50 ug/m^3 of outdoor PM would occur with a 40 ug/m^3 of indoor pollution (from indoor sources). Standards should be modified accordingly. All this of course does not take into account the different populations that may exist in certain indoor environments (more vulnerable) or different toxicities of indoor and outdoor particles.

Put another way, having a less stringent standard for indoor than outdoor air does not meet the "laugh test" for protecting public health. Would then an appropriate approach for pollution control be to ban chimneys and other methods of taking pollutants out of buildings where people spend time? To the extent that indoor environments can be considered a public good (and there is a substantial basis for doing so to a significant degree), the only possible reason to have less stringent standards indoors would be because it has been shown that IPM is less unhealthy than outdoor PM because of its chemical/physical characteristics. Arguments about population vulnerability and less than 100% penetration from outdoors to indoors only argue for more strict standards indoors.

> **Response**: Ambient air quality standards were used as approximate benchmarks in our indoor air quality guidelines, similar to what was done for the WHO guidelines for indoor and outdoor air quality. Ambient air quality standards are comprised of two parts: a concentration and an exposure duration, or averaging time, that are relatively safe, with a margin of safety. A person exposed to a certain level of PM for one hour would experience the same health impact whether he/she is standing indoors or outdoors. As long as the averaging time is specified, there is no need to adjust an indoor guideline level….the level is set based on the duration of exposure, or averaging time, and the concentration. Also, to clarify, this section discusses indoor air quality guidelines, not standards: there are no indoor air quality standards.

Specific comments

1. Page 111. The limitations of workplace regulations are more severe than those listed. They do not apply to indoor environments that are not workplaces. Also, they have been developed from an industrial hygiene perspective in which one or a few key chemicals dominate exposure. They seem entirely ill designed to serve as a basis for evaluating the health and comfort risks posed by the complex mixtures found in, e.g., modern office buildings.

   Response: There are few buildings that are not workplaces covered by Cal/OSHA's regulations. Unlike federal OSHA, Cal/OSHA's rules apply regardless of the number of employees. Only federal buildings are not covered by state regulations. As indicated in our text, Cal/OSHA has not only PELs, which we agree (and have stated in the report) are inadequate to protect many people, but they also have other regulations such as the Minimum Building Ventilation Standard, which applies broadly, and is protective of, or provides benefits to, any person in the building.

2. Page 113. Have the radon in drinking water regulations been promulgated and finalized?

   Response: Not yet. Will clarify in final report.

3. §4.3 Emission Limits. Much of this section is far from the issue indoor air pollution (e.g. the discussion of ARB's consumer products and architectural coatings programs). Note the earlier comment on whether the coating program has a positive impact on indoor air.

   Response: Language has been added regarding the impact on indoor air (ARB has prohibited some TACs in certain products, for example).

4. p. 125. The section on "DHS non-binding guidelines" does not discriminate among VOC emissions based on toxicity. Nor does it consider the possibility of secondary emissions caused by oxidation (e.g. owing to ozone exposure) of materials.
5. Page 126, first full paragraph. This sentence doesn't make sense as written: "emissions from a single material or product cannot exceed one half the chronic REL." Emissions would be expressed in mass per time. The REL is expressed in mass per volume. They cannot be directly compared.
6. Page 127, §4.3.3.3. This statement requires substantiation: "These programs have been successful in reducing emissions from their products over the last few decades."
7. Page 128, bottom. As in comment 64, the discussion of the GEI emissions criteria mixes measures. The implication is that emissions are to be limited so that individual VOCs "must meet the criteria of less than 1/10th of the threshold limit values…." In addition to the problem of equating emissions to concentrations, this also seems like an ineffective criterion for ensuring good indoor air quality. (An indoor environment in which multiple chemicals approached 1/10th of their respective TLVs would not be healthful for general occupancy.)
8. Page 131, lower half. The citation Maeda, 2004 does not appear in the reference list. (The committee did not audit the concordance between citations and references in general.)

9. Page 132, §4.4.1.3. Weatherization and duct sealing would affect not only indoor levels produced by combustion appliances, but from other sources, too.

Response: Text was revised for most of these comments; remainder will be made in final version.

Chapter 5 Methods to Prevent and Reduce Indoor Air Pollution

In a document that is painstakingly detailed with respect to regulations and guidelines (30 pages), it is disappointing to find control and mitigation so thinly discussed (5 pages in the main report, plus 2 in the executive summary). The major themes are more or less correct: source control, ventilation, public education, air cleaning devices, and building operation and maintenance. But the level of treatment of these topics is superficial to the point of being simplistic. This section is the weakest part of the report.

Response: We agree that the section needed work, and have revised and expanded the entire section.

Detailed critique is provided below for the first two topics.

- *Source Control (p 15 and 141)*
  - ? "There are many actions that can be taken to reduce indoor air pollution and, in some cases, completely eliminate sources." The problem of source control is much more complex and challenging to implement than acknowledged in this section.
  - ?

  Response: Text was revised.

  - ? Actions taken to "completely eliminate sources" are only pertinent for indoor emissions, and only for a subset of pollutants. When sources are outdoor air or the occupants themselves, then complete elimination is not an alternative.
  - ?

  Response: Section was revised, but it was never stated that all sources could be eliminated.

  - ? "The most effective preventive measure is to use building materials, consumer products, and appliances that emit little or no air pollution." This might be a practical alternative for some indoor air quality problems, but only if such information were widely available, which is not the case. Specifying reliable and practical test methods that work across the many diverse source categories is but one of many challenges.

  Response: Several available methods are mentioned in the options for mitigation section, but we agree that others are needed.

  - ? Reformulation of manufactured products and processes to eliminate certain toxic compounds and minimize overall emissions is an appropriate strategy. The report should acknowledge that not all indoor air problems are of the sort that can be controlled by this means (e.g., SBS, for which the causative agents are not yet known). It should also acknowledge that emitted species

vary markedly in their toxicity and so it is the composition of what is emitted in addition to the total amount that matters. Third, it should acknowledge the potential significance of secondary emissions, occurring because of reactions among the constituents or because of degradation/oxidation with time following installation or use.

Response: Some revisions have been made, and additional clarification will be added in the future**.** A more detailed assessment of potential risk reduction measures would be conducted before specific product reformulation is recommended.

- *Ventilation. (p 15, p 142-143)* Ventilation and its role in influencing indoor air quality is a much richer subject than can be adequately addressed in the brief treatment it receives in this report. Overall, relative to its importance, the document undervalues ventilation as a part of the overall indoor air pollution issue, particularly in office buildings and other structures with mechanical ventilation. While source control is an important part of an overall IAQ management strategy, so is the reliable provision of an adequate supply of good quality ventilation air to indoor spaces. Emissions control is clearly most important to avoid extreme problems (e.g., CO poisonings). But emissions will never be reduced to zero. Ventilation has an important role affecting IAQ in typical indoor environments. Important elements of the ventilation dimension of the IAQ issue that are inadequately discussed in this report include the following:
  - ? Data on current ventilation rates in the California building stock and trends.
  - ? Relationship between ventilation and indoor pollutant levels.
  - ? Challenges of providing adequate ventilation in the new residential building stock, given tighter construction practices.
  - ? Tensions between reducing ventilation for energy efficiency (CEC's focus) and the need for adequate ventilation to ensure good IAQ (and the technical potential for improving ventilation efficiency or effectiveness).
  - ? Challenge of providing good quality ventilation air when ambient air is unacceptably polluted, a common condition in California.

  Response: Information and Figure 5.1 were added. Some additional information may be added in the final version.

  - ? Consideration of "ambient air" as either a "high priority" or "medium priority" source category for mitigation.

  Response: The mitigation of pollution in ambient air is already a high priority…it is the reason for ARB's existence. However, it is not the focus of this report. Improved air filtration was included in recommendations for improved building operation and maintenance and indoor clean air technology development.

  - ? (p 22-23) Ensuring adequate ventilation in the building stock is glaringly absent from the 9 recommended "elements of an indoor air pollution reduction program." (Brief mention of proper venting of combustion sources under point 6, and low-noise fans under point 9 are not commensurate with the importance of this issue.)

Response: Recommendation No. 7 was revised to specify ventilation as a high priority. However, ventilation problems are largely ones of enforcement and diligence, not lack of laws or regulations. The Energy Commission has authority to establish minimum levels of outdoor air, and Cal/OSHA has authority over HVAC operation and maintenance.

- *Litigation*. Needed here also is a section discussing the history of litigation and its threat as an intervention for IAQ, which is referred to several places in the text, but never discussed directly. One of the arguments for keeping the government out of IAQ is that the courts work well enough to keep us safe. Clearly, there have been successes, which should be acknowledged, but also there are failures. It would also be good to examine how well this can be expected to work in future and whether there might be legislation that could make it work better.

  Response: Such information was added to the text.

Chapter 6 Prioritization of Sources and Pollutants

The criteria for choosing priorities need to be made a bit more clear as well as how they were applied. Indeed, it is the committee's view that there are really two (or possibly more) tiers – Tier 1 – where the indoor exposures for which the contaminant concentration or exposure distributions are fairly certain and the exposure-health-risk relationships are reasonably well understood and Tier 2, where there are uncertainties in these characterizations that would prevent informed actions (based on current knowledge). Within these tiers, prioritization criteria could be defined and applied.

Response: We believe that the two tiers of prioritization by source is the best approach, because then multiple pollutants emitted from a single source can be addressed at once. It is unclear where the panel believes the known health effects to be less certain.

- The EPA has done some interesting graphs showing the result of multiplying the range of exposures to a substance times the range of potencies expected to derive a range of impact. Can CARB do something similar?

  Response: Such an effort would take substantial time. (EPA spent two years on such an assessment for indoor pollutants, which has not yet been released.) This approach would be a useful approach for a comprehensive program.

- Are there (hidden) criteria of cost-effectiveness, i.e. are some things left off because they are considered undoable at reasonable cost?
- Are there (hidden) criteria of political feasibility, i.e. are there some things left off (e.g., candles, incense) because they would not be acceptable?

  Response: No, nothing is hidden. This analysis was not intended to be as detailed as indicating all sources in a category that might be addressed.

- What happened to lead? It is clearly important, but perhaps A) it was determined not to be an air issue because most exposure is by other routes or B) it was felt that it is being handled already. Explanation needed.

- Mercury, although perhaps less important, raises the same questions.

  Response: DHS manages the lead health program for the state. Mercury is handled by DHS and some local health departments.

- Pesticides are conspicuous by their absence on either the high or medium list of priorities.
- What happened to attached garages? And the related issue of gasoline vapor exposures? Nasty stuff, gasoline.

  Response: Both were added as suggested in the revised report.

How would all the proposed activities be organized and coordinated within the state? This seems like an extremely important but unaddressed issue.

Response: An agency in charge of a comprehensive program would clearly need to routinely coordinate with other agencies and interested parties. For state agencies, the California Indoor Air Quality Interagency Working Group serves as a good example of how such coordination might be accomplished. That group meets quarterly and is coordinated by DHS to foster communication and collaboration among agencies in addressing indoor air issues. Additionally, ARB has convened state agency review groups for this report and for the Portable Classrooms Study report, and received excellent input and suggestions on both reports. Finally, the state Sustainable Building Task Force and other interagency groups have successfully involved all agencies needed to assess specific issues or to provide input on various projects, and serve as successful prototypes for effective interagency communication and coordination.

Chapter 7 Options to Mitigate Indoor Air Pollution

(Pages 22-25 and 153-159) This portion of the report advances recommendations about actions that can be taken to improve indoor air quality in general, and in schools in particular. These recommendations are consistent with underlying science, to the extent that it is currently understood. A few observations about this section are worth considering during final revision of the report.

- (p 153) An effective overall management system for indoor air quality requires not only knowledge of pollutant sources, behavior, and consequences, but also strong expertise in building sciences. Part of the difficulty in effectively responding to the indoor air pollution problem is not only because of a lack of clear authority for the issue within any state agency, but also because of fragmented and incomplete relevant expertise.

  Response: Comment noted.

- (p 156-157) The emphasis on source control throughout this report should be tempered by the findings reported here for schools. Take note that the key problems reported in schools could not be addressed merely by effective source control. In addition, effective (and quiet) ventilation systems and proper maintenance are reported to be of importance.

Response: Our report did not indicate that source control would handle all indoor air problems. The report indicates throughout that ventilation, public education, and other measures are all part of an effective, comprehensive program to reduce indoor pollution. The text has been edited to emphasize this. Also, regarding schools, one of the most important problems identified was the elevated levels of formaldehyde, which require source control for effective risk reduction.

- (p 22 & 153) This sentence, which appears in both the executive summary and in the main report should be reconsidered: "The approach used to reduce toxic air contaminants in ambient air, in which source emissions are reduced without setting enforceable air quality levels, seems most applicable to indoor air." his statement seems to reflect an ARB-centered perspective in which only policy tools that have been developed for ambient air pollution control can be employed. The indoor environment shares some attributes with ambient air pollution, but it is sufficiently distinct to invite fundamental rethinking of policy approaches, rather than an effort to find the best fit from the current portfolio.

  Response: This statement was intended to differentiate from an approach more like the criteria standard-setting approach, in which strict standards are established and must be met. Instead, our TACs program allows identification of exposures and risks, with mitigation based on best available technology, not meeting a pre-set standard. Thus, as technology improves, additional reductions can be implemented.

Other Points

Methodological issues: When discussing mortality impacts, it is a bit misleading to sum simple deaths across diseases and risk factors. The disturbing issue about exposures to pollution or other risk factors, of course, is actually the premature mortality they cause. Thus, at the least, the term "premature" morality (or deaths) should be the wording.

Response: Text was revised.

As everyone dies, however, the degree of prematurity is critical, which is why lost-life-year measures are becoming more widely used. It would not seem possible that ARB can change the favored metric in one report, but it would seem worthwhile to offer a lost-life-year evaluation in parallel to the simple mortality tabulation, which requires knowledge/ assumption about the age distribution of the premature mortality. Even better, of course, would be a combined measure (QALY, DALY, etc) that combines lost years due to premature mortality with those due to illness/injury. The committee recognizes that doing so is probably beyond the resources and time available for this report, however.

Response: Comment noted. However, adding such an approach is beyond the time and resources available for this report.

There seems to be no discussion of "counterfactual" (CF) values in the assessments, but rather an implication that the entire pollutant level is up for grabs. Is it a reasonable

assumption that it would be possible to achieve zero concentrations of pollutants indoors with any conceivable set of interventions? We think not as there is no indoor environment in the world that has ever achieved it, bar perhaps Level 5 laboratories or some such. Just as it is inaccurate and misleading to account 100% of outdoor concentrations to "air pollution", so it is for indoor pollution (even not accounting for the outdoor pollution going indoors). The WHO Outdoor Air Comparative Risk Assessment (CRA), for example, used 7.5 $\mu g/m^3$ PM2.5 as the counterfactual level for outdoor air pollution (Cohen et al., 2004). It also did a sensitivity analysis that showed that changing the CF up or down has a big effect on the resulting overall estimate of burden, as you might expect. Zero, however, would be clearly wrong and misleading.)

The best CF to use would be one actually demonstrated in real interventions, preferably determined as a shift in the exposure distribution as it would be in the real world (also done in the CRA for some risk factors). In lieu of that, you could take the current 10% level or some such as the CF level with the assumption that it would be possible to reach that level with feasible interventions.

> **Response**: We did not mean to imply that risk can be reduced 100%. Clearly, the level of a pollutant outdoors will limit the levels that can be achieved indoors, in many cases. We have added some information reflective of a "counterfactual", such as the estimate of the remaining risk if indoor levels of radon were decreased to the ambient level, and will add additional information in the final report.

Section 2.1 The Key Health Impacts identified are asthma and cancer, and both are in fact very important; two others are cited, irritant effects and sick building syndrome (which is not itself a health effect). However, there are some other very important health impacts that should be carefully considered: heart disease, reproductive effects and diseases of the immune and nervous systems. Asthma and heart disease deserve more attention, given the large numbers of people affected, and therefore the public health impact of these diseases. Where there is uncertainty about the effects of indoor air pollutants on these other diseases, but some evidence exists, the uncertainty should be acknowledged.

> **Response**: Additional information was other impacts was added to the text.

The committee is also worried about Section 2.1.2, which seems to conflate chemicals with quite different levels of IARC carcinogenic status (table 2.4, p. 38) without noting the uncertainty in doing so.

> **Response**: Table 2.4 is simply to inform the reader regarding the IARC status of some common indoor air pollutants.

Mercury: It has been shown that the concentration can reach nearly 20 mg/m^3 indoors where liquid mercury has been spilled, and the Threshold Limit Value is 0.025 mg/m^3 for elemental mercury. Clearly seriously high levels of mercury are possible if elemental mercury is left in place, e.g., from a broken thermometer.

> **Response**: This is an accidental situation, and is currently being addressed through a program in the state to collect old mercury thermometers.

Risk perception: You may want to explore at bit more the anomaly that we heavily control hazardous waste dumps/leaking gas tanks/etc. and yet allow the very same chemicals almost free rein inside our houses within easy access of our young children, and so on. There is a natural tendency to be more concerned with uncontrolled "waste" but we are fooling ourselves if we act as if the material in our house is really under our control or somehow safer because we are still using it. This stretches a bit past IAQ per se, but is an important aspect of the risk perceptions related to it

> **Response**: We agree with the statement and the public's (and decisionmakers') lack of information on indoor air quality or their perception that it does not present a problem. However, public concern is growing as the public becomes more aware of the problem. This is evident in the number of public phone calls we receive from those seeking information, and the large and increasing number of visits to our indoor air quality website.

A double solidus should be avoided, as it can be ambiguous. Instead of $g/m^2/h$, for example, use $g/(m^2\text{-}h)$, which has no ambiguity. (One could use $g\ m^{-2}\ h^{-1}$, of course, but this might be too technical-looking for the intended audience.)

> **Response:** The representation of units was not changed. We felt this would be more understandable by the lay audience.

The word "data" is used as plural in some places and as singular in others.

> **Response:** We try to treat "data" as a plural term, and will edit the final version carefully.

Minor comments on Executive Summary

- Page 1, 1st paragraph. Not clear what is meant by "will be considered by the California Air Resources Board."

  > **Response**: The report will be discussed by our Board at their March meeting, to receive Board member comments before it will be finalized and sent to the Legislature.

- Page 1, final paragraph. Rather than "much less diluted," it is better to say that indoor emissions are "diluted much more slowly". (Also, P 28, first full paragraph.)

  > **Response:** Change was made.

- Page 1, final paragraph. Smith's "rule of 1000" has been substantiated by other investigators. The point made has much broader support than "one investigator has calculated." Sample references: Bennett et al., 2002; Lai et al., 2000. (Also, P 28, middle.)

  > **Response:** Additional support by other investigators was noted.

- Page 2, Figure ES-1 (and elsewhere). Please provide references for figures and tables that display published data.

  Response: Some notations were added to figures, and more will be cleaned up for the final version.

- Page 3, Table ES-1. Another major source of carbon monoxide is attached garages. Another major source of "formaldehyde and other aldehydes" is environmental tobacco smoke.

  Response: Suggestions were added to table.

- Page 5, Figure ES-2. Apparently the basis for this figure is detailed in the main report. The executive summary should point to the main body of the report where the estimates are developed.

  Response: So noted.

- Page 5, middle paragraph. Better to refer to "radium-containing" rock and soil as the source of radon. (Uranium is the ultimate progenitor, but geochemistry can cause separation between uranium and radium, the immediate parent.)

  Response: Some changes were made, but text remains uranium in some places for ease of understanding by lay readers.

- Page 8, first paragraph. Worth mentioning here that ozone produces PM as a byproduct as well.

  Response: PM was added to text.

- Page 9, final paragraph. To the best of my knowledge, swamp coolers are relatively uncommon in California. Better to focus on the main ventilation processes: infiltration, natural ventilation, and flow induced by fans (central air, and local exhaust).

  Response: Swamp coolers are fairly common in some parts of the state. An ARB-sponsored study specifically identified swamp coolers as a factor in moving the outdoor air (and ozone) to the indoors. Swamp coolers have also been associated with increased levels of dust mite allergen in homes.

- Page 10, final paragraph. Specify a time period when referring to IAQ costs as "potentially in the billions of dollars" (per year?).

  Response: Yes, per year. Will clarify here and throughout the report in the final version.

- Page 12, first paragraph under V. Should point out that workplace standards also don't apply to where children spend most of their time.

Response: Workplace standards in California do apply in schools and in publicly operated day care centers. They of course do not apply in homes, where children spend most of their time.

- Page 13. Proposition 65 is not mentioned in this list, although it seems that it has had a salutary effect on air pollutant exposure associated with certain consumer products, such as the elimination of TCE from "White-Out."

  Response: Prop 65 is discussed later in the report. We agree that is can have substantial positive impacts on indoor air, although it was not intended to address indoor air.

- Page 15, "reduction at the source". Control through source reduction is not so simple as reducing total mass emission rates. Toxicity can vary by orders of magnitude among species, and so the toxicity of emissions needs to be factored into any source-control strategy.

  Response: We agree. We would add that the percent of the population affected and the typical duration of exposure are also important considerations in determining an appropriate source control strategy.

- Page 16, "air cleaning devices". The discussion in the executive summary could also acknowledge the potential for future improvements in air cleaner technology (as discussed on p. 155 (ICAT for IAQ).

  Response: We will add a statement to that section in the final version of the report. Mitigation option no. 9 (indoor ICAT) includes a statement regarding the need for effective, low noise air cleaners.

- Page 17. Groundwater is also an important source of radon. Building materials are potential sources when they contain elevated levels of radium (not "radon gas").

  Response: Both suggestions were included in the tables.

- Page 18, first paragraph. Meaning of phrase is unclear: "the gap in reducing exposure and risk from categories of indoor sources."

  Response: That phrasing was deleted.

- Page 19, first paragraph. Contrary to what is stated, disease transmission can certainly occur because infectious agents are "emitted into the indoor environment per se."

  Response: Text has been corrected.

- Page 23, point 8. Good to mention the importance of indoor chemical reactions, in particular the importance of pollutant-surface interactions as an area in need of further study (e.g. ozone-carpet).

Response: We agree that pollutant surface interactions are an aspect of indoor chemistry that needs further research. However, we believe that is a fine point not appropriate for the executive summary.

Minor comments, Chapter 1

- Page 27, middle. There are not "several journals" that are "devoted exclusively" to the field of indoor air quality. Only one is first-rate: *Indoor Air*. Other journals carry IAQ articles, but are not devoted to it.

  Response: Text was adjusted accordingly.

- Page 29, bottom. Careful: Children do not "inhale a greater quantity" in an absolute sense (although they do per unit body weight).

  Response: Text indicates their increased inhalation is relative to body weight.

- Page 30, "children's activities". Reference group is unclear for "younger children spend more time near indoor sources…."

  Response: We will recheck and clarify in the final version.

Minor comments, Chapter 2

- Page 32, second sentence. Not clear what the antecedent is of "some."

  Response: Corrected.

- Page 32, third paragraph. Not clear what the basis is for the statement "only a fraction of indoor pollutants have been identified."

  Response: Clarification made.

- p. 32, para 3, end. This is quite misleading. With our growing analytic capability we are approaching a stage where we can essentially measure just about anything just about everywhere. This is not an indication of concern, however, for it is dose and toxicity that drive risk, not occurrence.

  Response: Indeed dose and toxicity drive risk and that point is made elsewhere in Chapter 2. However, to the extent that any toxic air contaminant is introduced into an indoor environment, especially one with children present, it provides the potential for exposure, and contributes to whatever burden may already exist in that environment.

- 34, line 3: asthma increase called "tremendous" before any discussion of it.

  Response: "Tremendous" was removed.

- p. 34, para 2: Careful, increase in simple asthma prevalence does not track directly to increases in asthma burden, which is driven by severe asthma attacks and deaths.

  Response: We remain concerned about the rise in asthma prevalence, however, for all of the reasons discussed.

- Page 39. On the theme of ETS exposure, it may be worth noting the estimates that have been made of population intake to specific hazardous air pollutants from residential ETS (Nazaroff and Singer, 2004).

  Response: Such information was added to the ETS discussion in section 2.3.3.1.

- Page 40. Not sure that isoprene does not react rapidly with ozone, in comparison with air-exchange rates. (See Atkinson and Arey, 2003, for confirmation.)

  Response: The mention of isoprene relates to the work of Wilkins et al., (2001) who studied its reactive chemistry.

- 40, last line: What terpene?

  Response: The point is that at room concentrations the reactivity products are probably more irritating than the precursors. The 'terpene' refers to those generally studied, pinene, limonene, etc.

- Page 41. In addition to forming formaldehyde, and secondary PM, the ozone-terpene reaction system generates the OH radical, which is a major story line for indoor air chemistry. This was an important aspect of the Fan et al. (2003) paper. The seminal paper on this topic is Weschler and Shields (1996).

  Response: Comment noted.

- p. 42, para 2, end: need citations here.

  Response: Several citations were added.

- Page 42, bottom. Worth noting is another key difference between indoor PM and outdoor PM: age of aerosol. Particles with organic content often tend to be emitted with the carbon in a chemically reduced form. As particles age in the atmosphere, the carbon is slowly oxidized. This changes the polarity and water solubility of the organic surface of the particle and could conceivably affect the toxicity. In this way, a typical indoor combustion particle might be quite different than a typical outdoor combustion particle.

  Response: Good point, will incorporate in final revision.

- Page 45, lower third. The statement that NAAQS for PM are "often exceeded in California's indoor environments" is not well supported by evidence presented. If true, then the current empirical basis is limited, since there are not too many

indoor PM measurements in California. (The PTEAM study is a noteworthy exception to this statement; however, that is a special case since the measurements were made during the autumn in Riverside, conditions that would tend to produce higher indoor PM levels than typical for California as a whole.)

Response: The studies shown in Table 2.5 for California do support that statement. All of the Los Angeles studies of indoor PM2.5 show indoor averages that exceed the California standard for PM2.5, annual average of 12 $\mu g/m^3$. Similarly, the indoor averages of all of the studies of PM10 conducted in Los Angeles (not just PTEAM) exceed the state PM10 annual standard of 20 $\mu g/m^3$. Finally, as indicated in the statement, homes with indoor sources would be especially likely to exceed the standards, based on the very high indoor PM emissions measured in the studies discussed in section 2.2.2.

- Page 45, §2.2.2. Is this line justifiable from existing data? "Indoor PM concentrations are typically equal to or higher than concurrently measured outdoor levels." Evidence suggests that residential PM levels are comparable between outdoors and indoors (lower without smoking; higher with smoking), but that in commercial buildings w/o smoking, indoor PM levels are systematically lower than those outdoors.

  Response: See response in earlier responses on PM.

- Page 46, first full paragraph. There are a few recent papers on indoor particles of outdoor origin that provide a stronger basis for the discussion than those papers cited here. See, for example, Riley et al., 2002; and Ott et al., 2000.

  Response: We will add these citations in the final version of the report.

- Page 46, first full paragraph. The discussion of residential ventilation here is a bit skewed. Dominating are infiltration, natural ventilation (windows), and mechanical flow induced by central air systems, exhaust fans, and vented combustion devices (e.g., fireplaces). Swamp coolers and whole house fans are rather less common (although perhaps not in Riverside).

  Response: There are few fireplaces in southern California. Swamp coolers and whole house fans are listed as examples of mechanical ventilation devices in homes that would influence indoor-outdoor relationships.

- Page 46 and elsewhere. No mention is made of the degree to which pollutants penetrate from outdoors to indoors along with infiltration air. Several papers have been published on this topic since the mid 1990s, among them being Liu and Nazaroff (2001).

  Response: Information will be added.

- Page 46, 2nd full paragraph. A thorough review of the role of ETS as a source of indoor PM can be found in Nazaroff and Klepeis (2004).

  Response: Thank you. Citation will be added.

- Page 55. Missing from the discussion of nitrogen oxides is the potentially important chemistry involving the nitrate radical (NO_3), which would be formed indoors whenever NO_2 and O_3 are found together. See Weschler (2004).

  Response: Revisions will be added to the text.

- Page 56, §2.2.6. Reference to "swamp coolers" seems misplaced here, as it would seem to be a relatively uncommon configuration for the California housing stock.

  Response: The number of homes with swamp coolers is not insignificant, and the Energy Commission previously encouraged their use, because they are more energy efficient than refrigerant coolers. Also, in the USC study Children's Health Microenvironmental Study, Steve Colome found that homes with swamp coolers in use had ozone nearly as high as outdoor levels on high ozone days.

- Page 57. Suggest updating the reference of Weschler et al. 1989 to Weschler, 2000.

  Response: We will update this and others in the final version of the report.

- Page 57, last paragraph. Ozone from any source can react with indoor surfaces (not only ozone from ozone generators), including carpets (Weschler et al., 1992, as cited; and Morrison and Nazaroff, 2002).

  Response: Will clarify in final version.

- Page 60, first bullet in 2nd list. Should not equate "emissions" with "concentrations." What does it mean to have "carpet emissions were generally below the limit of detection of 1 $\mu g/m^3$?"

  Response: Correction made.

- p. 65, para 2: this seems to have about the only mention of attached garages, which we understood to actually be a major source of exposure. Is this not so?

  Response: A section has been added on attached garages.

- Page 72, first paragraph. The result cited from Gilpin et al. (2001) is reported here differently than on the next page. Here it says that 78% of households with children didn't permit indoor smoking in 2001. (Implication: 22% of all households with children permit indoor smoking.) On the next page, it says that 22% of *smoking parents* still allowed smoking inside the home.

  Response: Will be corrected in the final version.

- Page 72, bottom. Substantially more detail on ETS emission factors of toxic air contaminants is now available in the papers by Singer et al. (2002, 2003).

Response: Thank you.

- 72, para 3: Not clear why there is a discussion in this report about active smoking in pregnant women. Will lead to confusion.

  Response: The one sentence about smoking during pregnancy serves to illustrate health effects associated with ETS.

- p. 74, Table 2.8: what are the averaging times?

  Response: Nicotine and RSP concentrations in this table represent different averaging times because they are from multiple studies. The ETS document prepared by ARB to identify ETS as a TAC has a full summary of the averaging times.

- Page 75, first bullet. The statement that "colds are more often transmitted by direct contact" should be supported with a reference. My understanding is that the mode of transmission of rhinovirus is largely unknown.

  Response: Text was changed**.**

- Page 85, §2.3.6.2. Should also mention candles with metal wicks as a source of lead exposure. (Van Alphen, 1999)

  Response: This was added to the report.

- Page 88, §2.3.8. Cooking (hot oils) should also be mentioned as a potentially important source of PAHs (it is in §2.3.8.2, but not in the opening paragraph). (Reference: Siegmann and Sattler, 1996).

  Response: Cooking oils were added to opening paragraph. Citation was included in source discussion.

- Page 91, Asbestos: This section should first explain the relevance of asbestos fibers >5 microns long. Explain the standards and the restriction to biologically relevant fibers. Keep the units constant, e.g., fibers/ml or per m^3.

  Response: Text was modified to discuss the impact of fiber length and the use of fiber length for standard setting. The units are the same as those used by the authors of various papers.

- Page 93 Health effects of PBDEs: Even if NOELs are not available, give LOELs. Again, some context is needed to understand other exposures.

  Response: Additional information will be added to the final version of the report. A statement from Rudel et al. (2003) was added in the concentration section that indicates 15 compounds measured in her study exceeded guideline levels. Dr. Rudel also notes that existing guidelines do not consider endocrine effects.

- Page 94 Sources of PBDE: Should explain why PBDE is added to these materials (fire retardant). PBDE Concentrations: Concentrations should be given here, and contextualized with health data/standards/health studies.

  Response: Text has been added to the source section to indicate PBDEs are fire retardants. Concentration data were added and contextualized with a statement about guideline levels.

Minor comments, Chapter 3

- Page 98, §3.1.1. Why exclude CO poisoning deaths from motor vehicle emissions, as many of these occur in indoor spaces? See Marr et al., 1998.

  Response: See previous response.

- Page 98, middle. A better reference (than CPSC, 1997) to the recent state of accidental CO poisoning deaths nationwide is Mott et al., 2002.

  Response: Thank you, we will incorporate this.

- Page 100, middle. "In addition, cigarette consumption by California adults was found to be about half of the US average…." On what basis? Per smoker? Per adult?

  Response: We will clarify in the final version.

- Page 106-107. Where is this statement substantiated?: "with proper measures, it is probably feasible to eliminate at least 50% of the particle exposures that contribute to asthma exacerbation, and likely more."

  Response: See earlier response; however, this statement will be removed.

- Page 107. At a few points in this discussion, it is suggested that the 2% productivity reduction owing to SBS is "conservative." Is the evidence sufficiently strong to conclude whether this is a conservative estimate or not.

  Response: The estimate was based on a mid-point value, so it is a conservative estimate in that context. Subsequent studies (summarized in Wyon 2004) suggest that even greater worker performance improvements, on the order of 5-10%, are achievable. This more recent information has been included in the text.

- Page 110. The last sentence on this page seems speculative, especially regarding IQ effects. Improved IAQ in schools will lead to higher intelligence quotients among students?!

  Response: Yes, the sentence does refer to "potential benefits." There are studies that indicate students have higher test scores when in more comfortable environmental conditions. Lead exposure, which would be reduced by improved

building maintenance and dust control in schools and homes with lead present, has specifically been associated with decrements in children's IQ's.

Minor comments, Chapter 4

- Page 111. The limitations of workplace regulations are more severe than those listed. They do not apply to indoor environments that are not workplaces. Also, they have been developed from an industrial hygiene perspective in which one or a few key chemicals dominate exposure. They seem entirely ill designed to serve as a basis for evaluating the health and comfort risks posed by the complex mixtures found in, e.g., modern office buildings.

  Response: The second and third paragraphs in this section discuss the shortcomings of Cal/OSHA regulations relative to full protection of workers.

- Page 111-112. I'm pleased to see the discussion of Prop 65 and the new EPA radon in water regulations in this section. (Have the radon in water regs progressed since 2000; update?)

  Response: The regulations have not been promulgated yet.

- §4.3 Emission Limits. Much of this section is far from the issue indoor air pollution (e.g. the discussion of ARB's consumer products and architectural coatings programs).

  Response: These two ARB programs do limit emissions from products. They serve as examples of how the agency can work with industry to derive products with less adverse environmental impacts. Additionally, ARB has restricted the use of several TACs in certain types of products.

- Page 126, first full paragraph. This sentence doesn't make sense as written: "emissions from a single material or product cannot exceed one half the chronic REL." Emissions would be expressed in mass per time. The REL is expressed in mass per volume. They cannot be directly compared.

  Response: Text was changed to indicate the emissions are put through a modeling process to estimate a room concentration.

- Page 127, §4.3.3.3. This statement requires substantiation: "These programs have been successful in reducing emissions from their products over the last few decades."

  Response: Text was changed.

- Page 128, bottom. As in comment 64, the discussion of the GEI emissions criteria mixes measures. The implication is that emissions are to be limited so that individual VOCs "must meet the criteria of less than 1/10th of the threshold limit values…." In addition to the problem of equating emissions to concentrations, this also seems like an ineffective criterion for ensuring good

indoor air quality. (An indoor environment in which multiple chemicals approached 1/10th of their respective TLVs would not be healthful for general occupancy.)

Response: Text was changed.

- Page 132, §4.4.1.3. Weatherization and duct sealing would affect not only indoor levels produced by combustion appliances, but from other sources, too.
  Response: text was changed.

Minor comments, Summary

- Page 160, near bottom. The underlying reason for the "rule of 1000" is not because of the factors cited, but rather because the rate of ventilation provided to buildings per occupant is about 1000 times less than the amount of wind-supplied "ventilation" to an urban area, per inhabitant.

  Response: Text has been changed to reflect this correction.

Additional references that might be used in the report: most cited above.

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