

# Scientific Peer Review of the

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

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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.

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.

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.

The report also notes, at p. 90 (3<sup>rd</sup> 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.

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.

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.

#### *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.)
2. p20. 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.
3. p20. 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.
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.
5. Based on the discussion above, the characterization of the risk estimate as “preliminary” on p 160 is unwarranted.
6. The radon discussion in Appendix II (p. II-4) is inconsistent with the exposure and risk estimates performed elsewhere in the report.

### **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 95<sup>th</sup> 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 95<sup>th</sup> percentile peak concentration.

One issue not addressed by the report directly is how much HCHO concentrations decline with building age due to outgassing, 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.

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.

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 95<sup>th</sup> 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.

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.

#### *Other VOC/TAC*

The review of VOC and TAC is quite extensive in the report. In many respects, however, the it presents an indiscriminating 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.

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).

#### *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.
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.
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)

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.

5. Page 60, first bullet in 2<sup>nd</sup> list. Should not equate “emissions” with “concentrations.” What does it mean to have “carpet emissions were generally below the limit of detection of 1 µg/m<sup>3</sup>?”

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, 3<sup>rd</sup> bullet; once again, given its demonstrated toxicity, report levels

## **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.)
- 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.
- 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.

### *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.
- 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.
- 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.

- 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.
- 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).
- 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.
- 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.)
- The discussion of health effects of biologically 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.
  - 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).
  - 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.)

- 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.
- 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.
- 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 3<sup>rd</sup> paragraph, p 79.
- 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.
- 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.

### *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.
  - 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).
- 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.
  - 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.
  - At the top of p 106, the figure \$24 million should be \$240 million.
  - 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.
  - 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)

## **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 CalEPA 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.

### *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.

Many of the compounds in ETS have been identified by CA EPA as Toxic Air Contaminants or are on the Prop 65 list of carcinogens and reproductive hazards. A very important point to make is the fact that as ETS levels decrease, the concentration of many VOCs, toxic compounds, and particles would also be expected to decrease. These VOCs and particles have known adverse health effects, and a connection should be made to these.

### *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).

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?

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.

### *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.

The risk estimate given on p. 100 is based on estimated US lung cancer rates, rescaled 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.

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).

### **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.

### *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 PM<sub>2.5</sub> 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 PM<sub>10</sub> and PM<sub>2.5</sub>, California cities generally had *lower indoor* concentrations for PM<sub>10</sub> and PM<sub>2.5</sub> than ambient concentrations.

We have several suggestions for Table 2.5

1. The values reported should be geometric means or medians, not means, which can be elevated by one or two high values
2. The entries should be grouped first by regions of the country
3. All studies in one city (e.g., LA, Boston) should be grouped together to ease comparisons
4. Some measure of the dispersion of the data should be included (e.g., SD, GSD)
5. The number of samples should be reported
6. Residential data should be separated from office building data
7. Include the other office building data mentioned on page 50
8. Where possible, report data separately for buildings where smoking did and did not occur during the sample collection

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 3<sup>rd</sup> paragraph, 6<sup>th</sup> 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, 3<sup>rd</sup> paragraph, 6<sup>th</sup> and 7<sup>th</sup> lines: the data on PM<sub>10</sub> personal are missing from the Table—please include
- p. 50 2<sup>nd</sup> line: 19.6 here but 19.5 in table—reconcile
- p. 50, 8<sup>th</sup> and 9<sup>th</sup> lines: a percentage is missing

- p. 50, 2<sup>nd</sup> 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
- p. 50 3<sup>rd</sup> 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 its 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  $\mu\text{g}/\text{m}^3$  per cigarette smoked per day, or 20  $\mu\text{g}/\text{m}^3$  per pack.

### **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.
- 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)
- How many homes had indoor CO greater than the state standard of 20 ppm?
- 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).
- Perhaps move figure 2.2 and the discussion of types of appliances to section 2.2.4.3, Emissions.
- 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).
- 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).
- 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.

### **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.

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.

### **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 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.

### **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).

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.)

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

### **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.

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<sup>3</sup> 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<sup>3</sup> 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<sup>3</sup> of outdoor PM would occur with a 40 ug/m<sup>3</sup> 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.

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

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 1/10<sup>th</sup> 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 1/10<sup>th</sup> 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.

## **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. 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.
  - 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.
  - “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.

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

## 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.

- 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?
- 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?
- 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.
- 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.

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

## 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.
- (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.

- (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.” This 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.

## **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” mortality (or deaths) should be the wording.

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.

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  $7.5 \mu\text{g}/\text{m}^3$  PM<sub>2.5</sub> 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.

*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.

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.

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

*Risk perception:* You may want to explore a 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

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

*The word “data”* is used as plural in some places and as singular in others.

#### *Minor comments on Executive Summary*

- Page 1, 1<sup>st</sup> paragraph. Not clear what is meant by “will be considered by the California Air Resources Board.”
- 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.)
- 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.)
- Page 2, Figure ES-1 (and elsewhere). Please provide references for figures and tables that display published data.

- 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.
- 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.
- 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.)
- Page 8, first paragraph. Worth mentioning here that ozone produces PM as a byproduct as well.
- 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).
- Page 10, final paragraph. Specify a time period when referring to IAQ costs as “potentially in the billions of dollars” (per year?).
- Page 12, first paragraph under V. Should point out that workplace standards also don’t apply to 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.”
- 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.
- 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).
- 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”).
- Page 18, first paragraph. Meaning of phrase is unclear: “the gap in reducing exposure and risk from categories of indoor sources.”
- 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.”
- 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).

*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.

- Page 29, bottom. Careful: Children do not “inhale a greater quantity” in an absolute sense (although they do per unit body weight).
- Page 30, “children’s activities”. Reference group is unclear for “younger children spend more time near indoor sources....”

*Minor comments, Chapter 2*

- Page 32, second sentence. Not clear what the antecedent is of “some.”
- Page 32, third paragraph. Not clear what the basis is for the statement “only a fraction of indoor pollutants have been identified.”
- 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.
- 34, line 3: asthma increase called “tremendous” before any discussion of it.
- 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.
- 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).
- 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.)
- 40, last line: What terpene?
- 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).
- p. 42, para 2, end: need citations here.
- 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.
- 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.)
- 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.
- 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.
  - 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).
  - 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).
  - Page 46, 2<sup>nd</sup> full paragraph. A thorough review of the role of ETS as a source of indoor PM can be found in Nazaroff and Klepeis (2004).
  - Page 55. Missing from the discussion of nitrogen oxides is the potentially important chemistry involving the nitrate radical (NO<sub>3</sub>), which would be formed indoors whenever NO<sub>2</sub> and O<sub>3</sub> are found together. See Weschler (2004).
  - 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.
  - Page 57. Suggest updating the reference of Weschler et al. 1989 to Weschler, 2000.
  - 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).
  - Page 60, first bullet in 2<sup>nd</sup> list. Should not equate “emissions” with “concentrations.” What does it mean to have “carpet emissions were generally below the limit of detection of 1 µg/m<sup>3</sup>?”
  - 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?
  - 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.
  - 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).
  - 72, para 3: Not clear why there is a discussion in this report about active smoking in pregnant women. Will lead to confusion.
  - p. 74, Table 2.8: what are 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.

- Page 85, §2.3.6.2. Should also mention candles with metal wicks as a source of lead exposure. (Van Alphen, 1999)
- 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).
- 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<sup>3</sup>.
- Page 93 Health effects of PBDEs: Even if NOELs are not available, give LOELs. Again, some context is needed to understand other exposures.
- 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.

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

#### Minor comments, Chapt 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.
- 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?)

- §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).
- 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.
- 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.”
- 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 1/10<sup>th</sup> 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 1/10<sup>th</sup> of their respective TLVs would not be healthful for general occupancy.)
- 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.

#### 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.

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

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