

## **Comments on the Public Review Draft of the *Report to the California Legislature, Indoor Air Pollution in California (Indoor Air Report)***

### **General Comments:**

The Public Review Draft of the *Report to the California Legislature, Indoor Air Pollution in California* (Indoor Air Report) is a good review of the problem of indoor air pollution. OEHHA agrees that indoor air pollution is a significant problem that is largely unregulated. This report provides a strong case for the significance of the indoor air pollution problem and the need to address it. It pulls together a diverse body of information on the occurrence, concentration and toxicology of indoor air pollutants, describes the regulatory framework and includes suggestions for helping to address indoor air pollution. As such, this document, with modification as described in the comments below, should be very useful to the Legislature.

Because of the diverse audience (from Legislators to UC peer reviewers), the level of technical language and explanation varies throughout the reports rather widely. Some of the report is still quite technical and the reader would need more than an average science background. In other places, the report is written more for the lay person. It is therefore particularly important to be aware of inferences that might be drawn by a lay audience, even though a person with more expertise might not draw those same inferences. It is also important that the report carefully reflect the general scientific consensus around toxicological and public health issues because the lay reader will not come to the report with that perspective. OEHHA's specific comments mainly address the instances where the report in our opinion deviates from the general scientific consensus on specific issues, or could be worded more clearly to avoid inaccuracies and confusion. OEHHA is recognized as the lead agency for chemical risk assessment in California. Specific comments follow:

### **Specific Comments:**

#### **A. Executive Summary**

The executive summary is well written and conveys the essence of the report nicely. There are however, several changes that we suggest below to improve the accuracy.

1) Table ES-1 (and also Table 2 in Chapter 2). The description of potential health effects for organic chemicals, formaldehyde and other aldehydes, (last column in table) starts with cancer. The risk of the other listed health effects is actually considerably greater for typical indoor air concentrations and so should come before cancer in the list. The same is probably true for ETS. We realize that this table was not meant to prioritize the health endpoints, but that is what readers will do. Also, in the first row last column - a colon is needed after "at high levels".

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2) Page 5 top paragraph – We suggest deleting the word “substantially” before “lower” in the second to last sentence when speaking of the decrease in attributable risks of ETS exposure anticipated because of the decreases in smoking behavior in California. The implication is that OEHHA’s revised estimates of cases of disease attributable to ETS will drop considerably. OEHHA is still in the process of estimating those attributable risks. While smoking has decreased in the workplace, the state’s population has increased. As the state’s population increases, the number of exposed people increases and the number of attributable cases increases. Thus, there may be a small drop in total attributable cases rather than the large drop anticipated by the statement in the report.

3) Page 5 under irritant effects , third sentence, add “respiratory and eye irritation,” before “headache” as that is probably the most frequently noted symptom from formaldehyde exposure. Also, the following sentence indicates that irritation is only seen after chronic exposure which is incorrect. Irritation of the eyes, nose, and throat is seen following both acute and chronic exposures.

4) Page 7 under Toxic Air Contaminants, the first sentence indicates that chemicals have been identified as TACs largely because of carcinogenic properties. This statement is not true. While there are a number of carcinogens that are identified as TACs, most are on the list because they are Hazardous Air Pollutants. Many important TACs are not carcinogens.

Also on page 7 under ETS, we would list Sudden Infant Death Syndrome as one of the effects as this is a very important health effect attributable in part to ETS exposure.

5) Page 11, top paragraph, second to last sentence starting with “These costs for morbidity do not include...”. We suggest adding “impacts from asthma exacerbation by VOCs” to this sentence since this is likely to be significant and is not currently in the estimate (right now, only asthma exacerbation by ETS is in the calculation).

6) Page 13. OEHHA now has 79 chronic RELs, not 71 as stated in the sentence.

7) Page 18, first paragraph. The sentence starting “Reformulation of other products such as cleaning agents to remove terpenes...” needs to be revised. Removing terpenes from cleaning agents will not, as implied, decrease carcinogenic risk. Limonene, the most common terpene in cleaning agents, is carcinogenic by a mechanism which is widely agreed to be not applicable to humans. Other terpenes are carcinogens but, to our knowledge, these are not added to cleaning products. It is probably best to end the sentence with “...reduce irritant effects” and drop the suggestion that cancer risk decreases.

## **B. Introduction**

- 1) Page 23 , third paragraph, first sentence. Which causes of sudden death is the document referring to here. SIDS? Heart attack? Both? The statement should be clarified.
- 2) Page 25 top first full sentence. It is not accurate to say across the board that all chemicals are absorbed more rapidly across the lung in children than in adults due to a larger breathing rate per unit surface area. Absorption across the lung is influenced by whether the absorption is diffusion limited, flow limited , etc, which in turn depends on the physicochemical nature of the compound. Suffice it to say that the exposure is elevated in kids because of the higher breathing rate/unit surface area.
- 3) Please add “and OEHHA” to the sentence page 25 midpage “ARB has re-evaluated the AAQS for particulate matter and ozone.” The statute requires that OEHHA provide a health-based recommendation to ARB for each AAQS. Thus, we have a significant and resource-intensive role in the reviews of the AAQS. Also, the ozone AAQS re-evaluation is not yet finished, so the sentence should read as follows: “ARB and OEHHA have re-evaluated the AAQS for particulate matter and are currently evaluating the AAQS for ozone.”

## **C. Section 2 – Health impacts, sources and concentrations of indoor air pollutants**

- 1) Section starting on Page 27. This section needs a description of the concept of dose-response. We are concerned that just listing health effects without putting them into perspective creates a somewhat alarmist tone and results in inaccurate assumptions by the lay reader. A solution would be to add something like this to the opening paragraphs:

“It is important to note that health effects are determined not only by the specific toxicology of the air pollutant but also by the exposure and absorbed dose. The higher the exposure and dose, the higher the risk of adverse health effects. In addition, more severe effects generally occur with higher doses. It is not possible in this document to describe the dose-response relationship for all indoor air pollutants. Information on dose-response can be found on the OEHHA website ([www.oehha.ca.gov](http://www.oehha.ca.gov)).”

Also on page 27, at the bottom, the document should cite McConnell et al. (2002) regarding contribution of air pollution to asthma. This is the Southern California Children’s Health Study paper linking ozone to asthma induction in active children.

- 2) Page 31, second paragraph, first bullet. Our understanding of the data is that formaldehyde is not associated with non-occupational asthma. Although the literature is inconsistent, most occupational health scientists would say that high occupational exposures are needed to see formaldehyde-specific asthma.

3) Table 2.4, page 32, “Common Carcinogenic Indoor Air Pollutants”. Table 2.4 correctly notes that the xylenes and 1,1,1-trichloroethane are listed as Class D by USEPA. None of these compounds are currently classified as carcinogens by USEPA, IARC or California’s Proposition 65. Thus, these compounds should be removed from Table 2.4 Common Carcinogenic Indoor Air Pollutants. For 1,1,1-trichloroethane, the USEPA IRIS database states, “There are no reported human data and animal studies (one lifetime gavage, one intermediate-term inhalation) have not demonstrated carcinogenicity.” For xylenes the USEPA IRIS database states, “Under the Draft Revised Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1999), data are inadequate for an assessment of the carcinogenic potential of xylenes. Adequate human data on the carcinogenicity of xylenes are not available, and the available animal data are inconclusive as to the ability of xylenes to cause a carcinogenic response.” The USEPA IRIS database for xylenes simply states under Carcinogenicity Assessment for Lifetime Exposure, “Not available at this time.” It should be noted that other common carcinogenic indoor air pollutants such as PAHs and ETS, listed by multiple authoritative bodies as carcinogenic do not appear in Table 2.4. These should be added, particularly since they are discussed further in the document.

Lastly, formaldehyde has recently been upgraded by IARC to a known human carcinogen. This should be noted in Table 2.4

4) Page 34 first full paragraph. Rather than stating that “U.S.EPA methodology indicates a safe level of risk as...”, it would be more appropriate to state “EPA considers acceptable levels of cancer risk to be...”. Neither EPA nor OEHHA uses the term “safe level” for carcinogenic substances.

Also on this page under section 2.1.3., “respiratory track” should be “respiratory tract”.

5) PM section

a) On page 36, Section 2.2.1, the following paragraph appears:

“Major epidemiologic studies have shown a strong association between ambient (outdoor) PM concentrations and increased mortality and morbidity (e.g., Dockery *et al.*, 1993; Pope *et al.*, 1995) and an increase in the rate of death from cardiovascular and respiratory disease (Samet *et al.*, 2000). Because they are based on particle size and include a mix of particles from combustion sources, soil, and other sources, these effects from ambient PM and their magnitude are directly relevant to assessing the potential risk from indoor PM. These effects of ambient PM are summarized below.”

The statement that indoor PM is equivalent to outdoor PM and therefore epidemiological studies of PM exposure based on outdoor monitoring are directly relevant to indoor PM exposure needs to be qualified a bit more. Indoor PM may in some cases be similar in composition to outdoor PM. However, there is considerable variability in the chemical composition, acidity and size distribution of outdoor PM depending on geological conditions, traffic mix, meteorological conditions, proximity to

major roadways and significant stationary sources. The heterogeneity of both indoor PM and outdoor PM should be acknowledged in the report. The relative contribution of indoor PM and outdoor PM to the total PM effects on morbidity and mortality is unknown and cannot be known given the way that the current epidemiological studies are conducted. Indoor PM could be considerably more or less toxic than outdoor PM. Although considerable progress has been made in elucidating the toxicological mechanism of PM toxicity, it is currently unclear so that it is somewhat (although not entirely) difficult to draw inferences between indoor and outdoor PM. We agree that it is reasonable and prudent public health policy to assume that indoor PM is toxic and to apply outdoor standards in the face of such uncertainties, but the uncertainties should be acknowledged with some detail.

It might be useful here to cite the ARB/OEHHA 2002 PM document as a useful reference for the health effects of PM.

b) Page 37 under absenteeism, please cite the Southern California Children's Health Study publication that described increased school absenteeism associated with PM.

c) Page 38 middle paragraph. We suggest deleting the sentences "This may be due to PM emissions from indoor combustion sources being relatively "fresh" (smaller in size and possibly more reactive) when breathed by people indoors compare to the more "aged" PM from outdoor sources (by the time it reaches people), thus having potentially greater toxicity. Additionally, the multiple toxics and respiratory pollutants in the indoor PM mix may make synergistic and cumulative effects more likely although those cannot yet be quantified". These statements are speculative. The sentences seem to be trying to state that indoor PM is more toxic than outdoor PM. The sentences imply that freshly made indoor PM is smaller and therefore indoor PM gets into the deep lung better than outdoor PM. However, size is not linearly related with deposition as the sentence seems to imply. Also, the sentences seem to imply that indoor PM, because it is "freshly made", is more reactive than outdoor PM. Outdoor diesel PM, which is in the respirable size range and so gets into the deep lung, is "freshly made" if you are standing near an idling vehicle or on a busy street. Finally, multiple exposures to many chemicals occurs outdoors as well as indoors, so that second sentence seems irrelevant to us.

d) Sections 2.2.2 and 2.2.3 could be considerably shortened without much loss of information.

6) Listing health effects not known to occur at typical indoor air exposure levels without explanation confuses the lay reader. As noted in the first comment above, the concept of "the dose makes the poison" is missing in the report and the lay reader cannot read between the lines. For example, on page 50, Section 2.3.1.2, it is stated that studies have shown that formaldehyde causes allergic sensitization. This is true but it has only been unequivocally demonstrated in workers exposed to considerably higher concentrations in the workplace. There is little evidence that allergic sensitization occurs at typical indoor

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exposure. Any statement on sensitization should be qualified by indicating that sensitization has been described following relatively high occupational exposures.

7) Page 46, top of page. One needs to be careful in overstating the effects of CO in Ritz et al. (2000). The paper did find effects of air pollution on birth outcome, but it is difficult to attribute the effects solely to CO due to presence of other covarying pollutants. The document should state the following sentence: “However, attributing the effects strictly to CO is difficult due to the presence of other correlated pollutants.”

8) Page 50 Section 2.3.1.1 As noted above, formaldehyde is now considered by IARC to be a known human carcinogen. This section needs to be updated.

9) Formaldehyde REL. On page 53, it is stated: “Although not evident in Figure 2.4, the results from Phase II of the California Portable Classroom Study (PCS) indicated that formaldehyde levels in at least 4% of California classrooms exceed OEHHA’s interim 8-hour REL of 27 ppb (Whitmore et al., 2003), the level at which an 8-hour exposure might result in irritant effects *and initiation of an immune system response in a sensitive individual [italics added].*” The REL was developed to protect against irritation. OEHHA developed the interim 8-hour REL and we do not support the statement that concentrations above 27 ppb might result in initiation of an immune response in a sensitive individual. The scientific evidence for initiation of immune response at levels below workplace exposures is not strong.

10) Page 58 at top. The wording of the ending phrase is a bit awkward with respect to mutagenicity and carcinogenicity. Carcinogenicity and mutagenicity are considered to have no threshold for exposure. It may be best to say “...;however, risk of these health effects may be low given typical use of these products.”

11) Page 60, section 2.3.3, second to last sentence. This sentence should read “Many chemicals in ETS have been identified as toxic air contaminants...” rather than “many of the chemicals in ETS...” There are thousands of chemicals in ETS and most are as yet unidentified.

12) Page 61 under Health Effects of ETS. The OEHHA ETS document is not yet finalized. We are working on responses to comments at the moment. The assertion of causality for breast cancer is being challenged, and OEHHA is re-evaluating the data. Please reword the last sentence of the first paragraph in this section as follows: “More recently, there is evidence from analyses of epidemiological studies that ETS exposure may have a causal association with breast cancer.” It is stated too certainly in the report as currently written.

13) Section 2.3.5. Pesticides

a)On page 65, the following is stated:

“Two classes of widely used insecticides in the U.S. are the organophosphates and pyrethroids, both are neurotoxins. Neurological signs resulting from acute toxicity may include nausea, headaches, dizziness, and general weakness. Pesticides are often measurable in house dust and carpet dust; levels of contamination are discussed below. The effects of pesticides on children are a particular concern because their behavior can lead to greater exposure than to an adult. Children spend time on the floor where they contact dust that may contain pesticides. The hand-to-mouth behavior of young children may lead to ingesting pesticides.”

Organophosphates are neurotoxins and can cause salivation, lachrimation, urination, defecation, nausea, dizziness, respiratory arrest, general weakness and death, depending on the dose. These symptoms have been described in pesticide workers exposed to agricultural organophosphates. These symptoms should not result from typical indoor air concentrations resulting from typical applications using the less acutely toxic types of organophosphates previously, but no longer, approved for home use. As pointed out in the Report, diazinon and chlorpyrifos were banned from residential use due to neurodevelopmental toxicity, potentially occurring in children at lower concentrations closer to some measures of indoor concentrations. The environmental half-lives of these organophosphates are such that indoor exposures should significantly decline over a period of a few months following cessation of use. The report needs to help the lay reader put the threat from these pesticides in perspective.

Pyrethroids' insecticidal action is due to neurotoxicity but humans and mammals are much less susceptible. Toxicologists consider pyrethroids as a class much less toxic than organophosphates. Pyrethroids act through a different mechanism than organophosphates. Dermal parosethia (skin numbing) has been reported in applicators exposed to very high concentrations, but neurotoxicity is not reported in humans at levels of exposure typical for home use. Subtle human neurotoxicity at typical indoor pyrethroid concentrations is possible but there does not appear to be a scientific consensus that it occurs. The lay reader could easily draw the conclusion that pyrethroids have the same toxicity as organophosphates, since both are simply described as neurotoxic. The reader may also conclude that such effects could occur with typical indoor exposures, which is not true.

The Report cites Landrigan et al., 1999 that pyrethroids are linked to childhood cancer (Section 2.3.5.1). We cannot find a statement by Landrigan et al. (1999) linking pyrethroids to cancer. Landrigan et al. (1999) does state that exposure to pesticides can cause disease in childhood as well as in later life including cancer, reproductive and disorders of the immune system, and neurological and behavioral disorders. In Table 1 from Landrigan et al. (1999), DDT, lindane, dieldrin and chlordane are correctly listed as carcinogenic. These pesticides have been banned for many years but exposure to these pesticides continues through dietary sources and exposure to indoor residues. These pesticides have very long environmental half-lives. OEHHA could not find any reference to permethrins being linked to cancer other than speculation that the estrogenic activity of some permethrins might contribute to cancer (Wolf, 1998). Thus the statement that pyrethroids have been linked to childhood cancers should be struck. In addition, the last

sentence at top of page 66 implies that organophosphates are endocrine disruptors. To our knowledge this is not true. The last sentence should be changed to read “Chronic exposure to pyrethroids has been linked to possible hormonal disruption.”

b) On page 66, Section 2.3.5.3, it is stated “Chlorpyrifos, cis and trans-permethrin, ortho-phenylphenol, piperonyl butoxide (PBO), and esfenvalerate were detected in over 80% of the classrooms suggesting indoor contamination from outdoor sources.” All of these pesticides are and have been used extensively inside (except currently chlorpyrifos which is now banned except for some agricultural uses). Cis and trans permethrin are used exclusively inside because they breakdown in the presence of UV light. It is most likely that these pesticides were found indoors because they were used indoors.

14) On page 70, it is stated “Organic and elemental mercury can cause permanent damage to the brain and central nervous system. Other health effects from exposure to mercury include tremors, changes in personality, loss of sensation and muscle coordination, vision and hearing impairments, and deficits in cognitive function. Elemental mercury ( $\text{Hg}^0$ ) is most toxic when inhaled. It can cause respiratory tract irritation, severe stomach discomfort, skin rashes and elevations in blood pressure and heart rate. All three types have been linked with kidney damage. The risk from long-term, low level exposure to inorganic mercury is not well understood. Carpi and Chen (2001) estimated that 10% of the U.S. homes may have indoor mercury levels that exceed the U.S. EPA Reference Concentration of  $0.3 \mu\text{g}/\text{m}^3$ .” The next paragraph starts with the following sentence: “Indoor air is the second most common route of exposure to mercury in the general U.S. population (fish consumption is first).”

The lay reader is not informed that the health effects described have only been described with workplace exposures at 25 to  $60 \mu\text{g}/\text{m}^3$  (OEHHA, 2000). The lay reader could quite reasonably conclude that typical indoor mercury exposures and/or fish consumption could cause these serious health impacts. While mercury is most definitely a developmental toxicant, it is not likely that typical mercury exposures in indoor environments are close to sufficient to produce developmental neurotoxicity. On page 71, Section 2.3.7.3, it is stated, “As little as one drop 0.05 ml of liquid  $\text{Hg}^0$  in a sealed bedroom sized room (assuming a room volume of about  $33 \text{ m}^3$  and no air exchange) can result in an air concentration equal to the U.S EPA Reference Concentration. This statement is true but somewhat deceptive to the lay reader. The U.S. EPA reference concentration is a safe level for long term 24 hour per day 7 days a week exposure for years. Exceeding the Reference Concentration does not necessarily mean that health impacts even with such long term exposure would occur but only that the likelihood of health impacts is increased. The paragraph needs to be revised to avoid confusion.

Mercury in paints was banned in interior use latex paints in 1990 and in exterior paint in 1991 (USEPA) (<http://www.epa.gov/ttn/atw/hlthef/mercury.html>). The same USEPA document states that almost all medicinal uses of mercury have been discontinued. However, it is stated in the Report on page 70, Section 2.3.7, “Inorganic mercury is intentionally added to latex paint, medicines and disinfectants to impart anti

bacterial properties.” Later on in Section 2.2.7.2, it stated that mercury was banned in interior latex paints in 1990. This is likely to be confusing to the lay reader. The removal of mercury from paint and the ban on the sale of mercury thermometers in California as of July 2002 without a doctor’s prescription (SB633) ([http://www.dtsc.ca.gov/Schools/EA\\_FS\\_SB633.pdf](http://www.dtsc.ca.gov/Schools/EA_FS_SB633.pdf)) should over time eliminate much of the indoor air exposure in California. OEHHA suggests that this be pointed out so that the lay reader knows that indoor exposure to mercury has been at least partially addressed. This allows the lay reader to put mercury exposure in perspective relative to other indoor air problems.

15) On page 69, Section 2.3.6.1, it is stated that much of the lead present in indoor air appears to result from the infiltration of lead particles in outdoor air. This could be true if the house does not have lead based paint. However, lead based paints were used both indoors and outdoors and resuspended lead dust from crumbling indoor paint is the primary source in many cases.

16) Section 2.3.11 discusses indoor exposure to PBDEs and other endocrine disruptors. The use of diethylstilbestrol and dioxins as examples of hormone disruptors is likely to mislead a lay audience because of a lack of perspective relative to indoor air exposures and no explanation of the concept of dose response. DES is not an indoor air contaminant and was specifically designed as a drug to have powerful hormonal properties. Dioxin is more of an outdoor air emissions problem than an indoor air problem. In addition, the vast majority of dioxin exposure comes from the food chain, not inhalation of indoor (or outdoor) air.

There is nothing in Section 2.3.11 that allows the lay audience to distinguish between the hormonal effects reported from diethylstilbestrol and dioxin exposure from the effects that could occur with indoor exposure to hormone disruptors. The general public has at best a tenuous understanding of the concept of dose-response. It would be more useful to use examples of endocrine disruptors found in indoor air (like PBDEs and diethylhexylphthalate).

### **References:**

Garey J, Wolff, Estrogenic and antiprogestagenic activities of pyrethroid insecticides. *Biochem Biophys Res Commun.* (1998) Oct 29;251(3):855-9.

(OEHHA, 2000) Air Toxics Hot Spots Program Risk Assessment Guidelines Part III Technical Support document for the Determination of noncancer Chronic Reference exposure Levels. Office of Environmental Health Hazard Assessment , California Environmental Protection Agency, April 2000.

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McConnell R, Berhane K, Gilliland F et al. (2002) Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359:386-91.