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August 25, 2004

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Dear Ms. Shimmer:

The American Chemistry Council Phthalate Esters Panel (Panel) submits these comments on the draft *Report on Indoor Air Pollution in California* (AB 1173, Keeley). The Panel consists of the major domestic manufacturers and some users of phthalate esters. These comments pertain to statements about phthalates in the draft Indoor Air Quality Report (draft IAQ Report).

The draft IAQ Report includes phthalates among substances it states pose substantial health risks in indoor air. However, the data strongly indicate that phthalates in indoor air do *not* pose substantial health risks. The Panel urges the California Air Resources Board (CARB) to remove phthalates from the Indoor Air Quality Report altogether, lest resources be diverted to control substances that evidence indicates pose no substantial health risk. If CARB continues to include phthalates in the IAQ Report, it should revise its statements about phthalates in accordance with these comments, and should provide readers with perspective on the very low risk posed by phthalates in indoor air.

If you have any questions, please call Marian K. Stanley, Senior Director and Manager of the Phthalate Esters Panel, at (703) 741-5623, or email her at marian_stanley@americanchemistry.com.

Sincerely yours,

A handwritten signature in black ink that reads "Courtney M. Price". The signature is written in a cursive style with a large initial 'C'.



**Before the
California Air Resources Board**

**COMMENTS OF THE
PHTHALATE ESTERS PANEL OF THE AMERICAN CHEMISTRY COUNCIL
ON A DRAFT REPORT ON INDOOR AIR POLLUTION
MANDATED BY AB 1173 (KEELEY, 2002)**

AB 1173 Indoor Air Quality Report)
<http://www.arb.ca.gov/research/indoor/ab1173/ab1173.htm>)

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EXECUTIVE SUMMARY

The American Chemistry Council Phthalate Esters Panel (Panel) submits these comments on the draft *Report on Indoor Air Pollution in California* (AB 1173, Keeley). The Panel consists of the major domestic manufacturers and some users of phthalate esters. These comments pertain to statements about phthalates in the draft Indoor Air Quality Report (draft IAQ Report).

The draft IAQ Report includes phthalates among substances it states pose substantial health risks in indoor air. However, the data strongly indicate that phthalates in indoor air do *not* pose substantial health risks. These comments make the following points:

- Although phthalates are frequently detected in indoor by the highly-sensitive techniques of modern chemistry, their concentrations are extremely low (they are reported in nanograms per cubic meter). The exposures that could potentially result from these very low concentrations air are well below benchmarks that have been established for the protection of human health.
- The weight of evidence shows that phthalates do not mimic or block estrogen or androgen hormones. Some (but not all) phthalates cause decreased levels of testosterone when given to rodents in very high doses, but human exposures from reported indoor air concentrations would be far below such levels. Some phthalates influence male reproductive development in rodents, but do not do so in primates even at very high doses, indicating the rodent studies may not be relevant to humans. And, for these effects also, human exposures from reported indoor air concentrations would be far below the effect levels in rodents.
- There is not reliable evidence that phthalates cause or worsen asthma. Studies that report an association between phthalates and asthma have not controlled for potential confounders; most importantly, they cannot distinguish between phthalates causing or worsening asthma, versus persons with asthma selecting phthalate-containing products (e.g., vinyl flooring) to reduce dust concentrations in their homes. In studies in mice, phthalates did not stimulate the production of cellular products in the mice that are associated with the types of allergic reactions in the lung that typically lead to an asthma attack.
- Contrary to the statement in the draft IAQ report, di(2-ethylhexyl) phthalate (DEHP) is currently classified by the International Agency for Research on Cancer (IARC) as Group 3, “not classifiable as to human carcinogenicity,” on the basis that the mechanism by which DEHP increases the incidence of tumors in rodents is not relevant to humans. Other recent reviews and the California courts have likewise found that DEHP does not pose a risk of cancer to humans. However, even assuming that DEHP could be a human carcinogen, exposures from reported indoor air concentrations would be well below California’s No Significant Risk Level for DEHP.

- The scientific evidence does not support the draft IAQ Report statements that other phthalates are known indoor air carcinogens. To the contrary, the evidence suggests that, like DEHP, other phthalates are not likely to pose a risk of cancer to humans. The statements indicating other phthalates are known carcinogens should therefore be removed from the report.

For these reasons, the Panel urges the California Air Resources Board (CARB) to remove phthalates from the Indoor Air Quality Report altogether, lest resources be diverted to control substances that evidence indicates pose no substantial health risk. If CARB continues to include phthalates in the IAQ Report, it should revise its statements about phthalates in accordance with these comments, and should provide readers with perspective on the very low health risk posed by phthalates in indoor air.

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INTRODUCTION

The American Chemistry Council Phthalate Esters Panel (Panel) submits these comments on the draft *Report on Indoor Air Pollution in California* (AB 1173, Keeley). The Panel consists of the major domestic manufacturers and some users of phthalate esters.¹ These comments pertain to statements about phthalates in the draft Indoor Air Quality Report (draft IAQ Report).

The draft IAQ Report includes phthalates among substances it states pose substantial health risks in indoor air. However, the data strongly indicate that phthalates in indoor air do *not* pose substantial health risks. Exposures from reported indoor air concentrations of phthalates are well below health benchmarks established to be protective of human health. The Panel therefore believes it would be appropriate for the California Air Resources Board (CARB) to remove all discussion of phthalates from the IAQ Report. Their inclusion in the Report may cause unwarranted concern and may lead to resources being misdirected toward control of substances that evidence indicates do not pose substantial health concerns.

If CARB nevertheless continues to include phthalates in the IAQ Report, then it should provide readers perspective on the very low risks posed by these substances in indoor air, as discussed below. It also should correct inaccurate statements about phthalates in accordance with these comments.

I. THE EVIDENCE STRONGLY INDICATES THAT PHTHALATES IN INDOOR AIR DO NOT POSE SUBSTANTIAL HEALTH RISKS

The draft IAQ Report states: “Available scientific information indicates that indoor air pollution poses substantial health risks in many indoor environments” (p. 1). It then includes phthalates in a table on “Sources and Potential Health Effects of Major Indoor Air Pollutants” (Table ES-1, p. 3 and Table 2.1, p. 28).² The implication is that phthalates are major indoor air pollutants that pose substantial health risks. However, the scientific evidence clearly establishes that this is not the case.

A. Reported Indoor Air Concentrations of Phthalates Are Extremely Low – Well Below Health Benchmarks

Phthalates are detected in indoor air samples, but at extremely low levels – generally well less than 1 microgram per cubic meter (ug/m³). Clark et al. (2003) have summarized indoor air concentrations for phthalates from a comprehensive review of the

¹ The Panel members are BASF Corporation, Eastman Chemical Company, ExxonMobil Chemical Company, Ferro Corporation, and Teknor Apex Company.

² Phthalates are included in the category of organic chemicals, for which potential health effects are listed as “Cancer; eye, nose, throat irritation; possible worsening of asthma; headaches; at high levels; loss of coordination; damage to liver, kidney and brain.” They are also included in the category of endocrine disruptors, with potential health effects listed as “Mimic or block natural effects of hormones (estrogen and others); developmental abnormalities.”

literature. Their data is provided in Table 1, along with health benchmarks for comparison. Table 1 demonstrates that the levels of phthalates detected in indoor air are far below levels established for the protection of health.

Table 1. Indoor Air Concentrations of Phthalate Esters

Phthalate and Region ^a	Indoor Air Concentrations in nanograms per cubic meter (ng/m ³) ^b				Chronic REL ^c ng/m ³	Exposure as ug/kg/day ^d	EPA RfD ug/kg/day ^e
	Median	Mean	Min	Max			
Dimethyl							
Europe	10	20.2	<1	129	--	0.037	--
Diethyl							
USA	340	NA	NA	NA	--	0.097	800
Europe	171	621	25	3234		0.92	
Dibutyl							
USA	NA	0.2	0.2	420	--	0.12	100
Canada	NA	2.9	NA	NA		0.00083	
Europe	551	1032	<3	9445		2.7	
Butylbenzyl							
USA	35	NA	NA	140	--	0.040	200
Europe	13	35	<3	465		0.13	
Di(2-ethylhexyl)							
USA	55	109	20	240	70,000	0.069	20
Canada	NA	NA	<500	3100		0.89	
Europe	111	245	18	1046		0.30	

NA = not available

- If a region is not included for a given phthalate, there were no data available for that region.
- From Clark, C., Cousins, I., Mackay, D., and Yamada, K. (2003). Observed concentrations in the environment. In: Phthalate Esters, The Handbook of Environmental Chemistry. 3Q. C. Staples, ed., Springer, New York, pp. 125-177.
- The noncancer chronic reference exposure level established by the California Air Resources Board and the California Office of Environmental Health Hazard Assessment.
- The exposure of a 70 kg person who breathes 20 cubic meters of air a day, containing phthalate at the maximum reported concentration, and assuming that all measured phthalate is bioavailable and absorbed by the blood stream. Based on the maximum value reported by Clark et al. (2003).
- The reference dose established by the U.S. Environmental Protection Agency, from the Integrated Risk Information System (IRIS) database Agency (www.epa.gov/ngispgm3/iris).

For di(2-ethylhexyl) phthalate (DEHP), CARB and the Office of Environmental Health Hazard Assessment (OEHHA) have established a chronic reference exposure level (REL) of 70 micrograms per cubic meter (ug/m³), or 70,000 nanograms per cubic meter (ng/m³).³ The

³ The REL is for noncancer endpoints. Cancer is discussed in Part II, below.

highest indoor level reported for DEHP is over 20-fold below that and the mean value for the United States is over 600 times lower.⁴

CARB and OEHHA have not established RELs for other phthalates. However, the U.S. Environmental Protection Agency (EPA) has developed oral reference doses (RfDs) for several phthalates. “The RfD is a numerical estimate of a daily oral exposure to the human population, including sensitive subgroups such as children, that is not likely to cause harmful effects during a lifetime.”⁵ If one conservatively assumes that all phthalate measured in the air is bioavailable and is absorbed into the bloodstream, then the air concentration can be converted to an equivalent oral concentration and compared to the RfD. This is a conservative approach, because absorption of inhaled chemicals is usually less than 100%, because some of the phthalate may be bound in a PVC matrix and not bioavailable, and because phthalates appear to be less toxic by parenteral routes (such as inhalation) than by the oral route (FDA, 2001). Nevertheless, as shown in Table 1, exposure even from the maximum reported air concentrations of phthalates would be well below EPA’s RfDs.

The RfDs are themselves set at values well below doses required to cause effects in rodents. The RfDs for phthalates are three or more orders of magnitude below even the most sensitive, reliable LOAELs (lowest observed adverse effect levels) reported for rodent studies. Yet primate studies indicate that humans are likely far less sensitive to phthalates than are rodents. For example, slight histopathological testicular effects have been reported in rodents dosed with 38 mg DEHP/kg/day for 90 days (Poon et al.), but no such effects were seen in a study of monkeys receiving up to 2500 mg DEHP/kg/day for about 455 days (Tominari et al., 2003). Thus, it is likely humans can be exposed to levels well in excess of the RfDs without experiencing adverse health effects. Since these reported indoor air levels of phthalates represent exposures far below the RfDs, they should not pose a substantial health risk.

The draft IAQ Report mentions that in a study by Rudel et al. (2003), “[t]he most abundant compounds in [indoor] air included bis(2-ethylhexyl) phthalates (DEHP) . . . [and other compounds]” (p. 78). CARB should not confuse frequency of detection with “abundance.” Nor do concentrations above some other measured chemicals necessarily indicate a risk. Phthalates are used in a wide variety of products, and, when looked for with modern, highly-sensitive analytical techniques, they are frequently detected. But, again, the levels detected are extremely low. The concentrations reported by Rudel et al. (2003) are similar to those summarized in Table 1, and represent exposures several orders of magnitude below levels that have caused health effects in animal studies. In this sense, the studies reflect that phthalates are not at all abundant in indoor air, but rather sparse. Certainly the science does not support making phthalates a focal point of concern for indoor air quality.

⁴ In 1999, OEHHA proposed a chronic REL of 10 ug/m³ (10,000 ng/m³) for DEHP. The Panel submitted comments explaining its belief that the science did not support that low an REL. Even if that were the REL, reported levels of DEHP are well below that level.

⁵ Definition of “Reference Dose (RfD)” at <http://www.epa.gov/glossary>.

B. The Weight of Evidence Is that Phthalates Do Not Mimic or Block Hormones

The draft IAQ Report includes phthalates in the category of endocrine disruptors, with potential health effects listed as “Mimic or block natural effects of hormones (estrogen and others); developmental abnormalities” (Table ES-1, p. 3 and Table 2.1, p. 28). However, the weight of the evidence is that phthalates do *not* mimic or block hormones.

The weight of evidence indicates that phthalates do not react with the estrogen receptor in live animals. Harris et al. (1997) reported that several phthalates weakly interacted with the estrogen receptor in screening tests under *in vitro* conditions, but that many – including di(2-ethylhexyl) phthalate (DEHP) – did not. Harris et al. also reported that monoesters, the phthalate metabolites that are present *in vivo*, were estrogenically inactive. A subsequent *in vivo* study by Zacharewski et al. (1998) showed that phthalates were not estrogenically active when tested in rats. More recent studies in rodents provide additional evidence that phthalates do not affect processes under estrogenic control (Gray et al., 1999; Moore et al., 2001). The current view is that, although some phthalates may interact with estrogen receptors under *in vitro* conditions, they are not estrogenic *in vivo*, at least in part because they are metabolized to inactive forms before absorption (Foster et al. 2000; Moore, 2000; Parks et al., 2000).

With respect to testosterone-mediated effects, some phthalates (*but not all*) have produced effects on male reproductive development in rats (Gray et al., 1999; 2000; Mylchreest et al., 1998; 1999; 2000). Researchers have determined that this process does not involve androgen receptor-mediated interactions – that is, phthalates neither mimic nor block androgen – although there is evidence of an effect on testosterone synthesis, due to some other as yet unknown mechanism (Gray et al., 1999; 2000; Parks et al., 2000). The effects on testosterone levels are observed at very high doses – doses far above exposures that would occur from reported indoor air concentrations of phthalates.

As just indicated, phthalates do cause developmental abnormalities, *in rodents* and at high doses. The studies in primates discussed in Section I.A. indicate that the effects in rodents may not be relevant to humans. Even assuming human relevance, however, the levels of potential exposure from reported indoor air concentrations are far below levels that produce developmental effects in rodents, as discussed in Section I.A., above.

C. There Is Not Reliable Evidence that Phthalates Cause or Worsen Asthma

Among the potential health effects listed for organic chemicals, in which category the draft IAQ Report includes phthalates, is “possible worsening of asthma” (Table ES-1, p. 3 and Table 2.1, p. 28). There have been some studies which have reported an association between phthalates and asthma prevalence; however, those studies are subject to a number of flaws and in no manner can be considered reliable evidence that phthalates cause or promote asthma.

Most importantly, an association is not proof of causation. In the case of asthma, patients are commonly advised to remove sources of dust from their homes, such as carpets. Thus, such homes are more likely to have phthalate-plasticized vinyl flooring. The studies published to date cannot distinguish whether the association of phthalates and asthma is because

the phthalates contributed to asthma, or because the occurrence of asthma led to greater use of phthalate-containing products.

The draft IAQ Report discusses a report by the National Academy Institute of Medicine (IOM, 2000), which “examined the scientific literature relating indoor air pollutants and other factors to asthma” (p. 29). The draft IAQ Report lists “plasticizers” as substances identified by the IOM as possibly associated with exacerbation or development of asthma (Tables 2.2 and 2.3, pp. 29-30). What the IOM report actually concluded about plasticizers (such as phthalates) was: “While the reports described above have attracted some interest in the research and building trades communities, there is inadequate or insufficient evidence to determine whether or not an association exists between nonoccupational exposure to plasticizers and the development or exacerbation of asthma.” (IOM, 2000).

Subsequent to that report, studies have been undertaken to investigate the potential for phthalates to cause respiratory sensitization. Butala et al. (2004) tested four common PVC phthalate plasticizers – di(2-ethylhexyl) phthalate (DEHP), diisononyl phthalate (DINP), di-isoheptyl phthalate (DIHP) and butyl benzyl phthalate (BBP) – in a mouse model. The phthalate applications did not stimulate the production of cellular products in the mice (IgE, IL-4, and IL-13) that are associated with the types of allergic reactions in the lung that typically lead to an asthma attack. These results indicate that DEHP, DINP, DIHP, and BBP are not likely to produce asthma.

Questions have also been raised as to whether some phthalates could act as adjuvants, i.e., whether they might exacerbate the effects of other allergens (Larsen et al., 2001a; 2001b; 2002; 2003). These novel studies exhibited some variability, and did not show clear dose-response relationships. Larsen et al. (2002) concluded that some phthalates were adjuvants based on elevated levels of IgG1 and IgE. The authors considered that IgG1 and IgE were good markers for Type 1 allergy in human, and that they were co-regulated in mice via the Th2/IL-4 pathway. However, as summarized above, Butala et al. (2004) found phthalates to have no effect on IgE or IL-4 levels. To investigate this further, a research program has been undertaken with two aims: to determine if the results of Larsen and associates could be replicated in an independent laboratory, and to define the underlying mechanism(s). Participants in the program include the developers of the murine respiratory sensitizer model used by Butala et al. and the initial investigators of the Larsen et al. studies. Initial work from this program has not repeated the original findings of Larsen et al. Work continues to explore many possible variables to explain this difference. At the present time, however, the weight of evidence is insufficient to support a link between phthalates and asthma.

II. THE WEIGHT OF EVIDENCE IS THAT PHTHALATES IN INDOOR AIR DO NOT POSE A CANCER RISK

To quantify potential health risks from indoor air pollutants, the draft IAQ Report relies primarily on risk estimates from the 1994 California Comparative Risks Project. DEHP was one of the chemicals included in that project. The draft IAQ inaccurately indicates that DEHP is classified by the International Agency for Research on Cancer (IARC) as a possible human carcinogen, when IARC in fact classifies DEHP as “not classifiable as to human carcinogenicity” because IARC found the tumors seen in rodents treated with DEHP to not be

relevant to humans. Other recent reviews and the California courts have likewise found that DEHP does not pose a risk of cancer to humans. However, even assuming that DEHP could be a human carcinogen, potential exposures from reported indoor air concentrations are well below California's No Significant Risk Level for DEHP. The scientific evidence does not support the draft IAQ Report statements that other phthalates are known indoor air carcinogens. Therefore, the Panel believes that CARB should eliminate phthalates from any discussion of carcinogenic risk of indoor air pollutants.

A. IARC No Longer Classifies DEHP as a "Possible Human Carcinogen"

On page 32, the draft IAQ Report includes DEHP in a table of "Common Carcinogenic Indoor Air Pollutants" (Table 2.4). The table shows the U.S. EPA classification of DEHP to be Group B2, probable human carcinogen, and then indicates in parenthesis "IARC classification 2B, possible human carcinogen." This is inaccurate.

In 2000, IARC reviewed the extensive data that had been generated on DEHP carcinogenicity since IARC had classified it in the early 1980's. IARC determined that DEHP should be reclassified to Group 3, "not classifiable as to human carcinogenicity," on the basis that "the mechanism by which di(2-ethylhexyl) phthalate increases the incidence of hepatocellular tumours in rats and mice is not relevant to humans" (IARC, 2000). CARB should correct the IAQ Report to correctly reflect the current IARC classification of DEHP.

B. There is a Strong Consensus Among Reviewing Scientists that DEHP Does Not Pose a Risk of Cancer to Humans

Other recent reviews agree with the conclusion of IARC.

- *ILSI Workshop*. The International Life Sciences Institute (ILSI) Risk Science Institute formed a workgroup in 2001 to review information on the mechanisms by which peroxisome proliferating chemicals produce carcinogenic responses in rats and mice. The report of the workgroup was published in late 2003 (Klaunig et al., 2003). For peroxisome proliferators in general, the workgroup concluded: "In summary, the weight of evidence overall currently suggests that the rodent [mode of action] for liver tumors is not likely to occur in humans, taking kinetic and dynamic factors into account" (Klaunig et al., 2003, p. 693).⁶ DEHP was included as a case study by the group, with the following outcome: "The data lead to a conclusion that a carcinogenic response induced via the [modes of action] for liver tumorigenesis in the rodent is not likely to occur in humans following exposure to DEHP" (Klaunig et al., 2003, p. 704).

⁶ On the basis of the ILSI workgroup conclusions, the U.S. Environmental Protection Agency (EPA) has proposed a science policy: "When liver tumors are observed in long term studies in rats and mice, and 1) the data are sufficient to establish that the liver tumors are a result of a PPAR α agonist MOA and 2) other potential MOAs have been evaluated and found not operative, the evidence of liver tumor formation in rodents should not be used to characterize potential human hazard" (EPA, 2003, p. 15).

- *Health Canada Assessment.* As part of an evaluation of the use of DEHP in vinyl medical devices, Health Canada reviewed the cancer data and accepted the conclusions of IARC (2000) that DEHP is not classifiable as to its carcinogenicity to humans (Health Canada, 2002).
- *Doull et al. Assessment.* In 1998, a panel of scientific experts, chaired by Dr. John Doull, reviewed the data for DEHP in light of EPA's draft cancer risk assessment guidelines. The panel concluded: "DEHP should be classified as unlikely to be a human carcinogen under any known conditions of human exposure" (Doull et al., 1999, p. 352).

Thus, the consensus of a large number of scientific experts is that DEHP is not reasonably anticipated to be a human carcinogen.

Further, the California courts have found this to be the case. In *Baxter Healthcare Corporation v. Denton*, No. 99CS00868, (Sacramento Co. Super. Ct. 2002), the Superior Court of Sacramento found that DEHP poses no significant risk of cancer to humans. The California Court of Appeal recently upheld this finding. *Baxter Healthcare Corporation v. Denton*, 120 Cal. App. 4th 333; 15 Cal. Rptr. 3d 430; 2004 Cal. App. LEXIS 1054; 2004 Daily Journal DAR 8099; 34 ELR 20042 (Cal. App. 3d Dist. 2004)).

In light of the strong scientific consensus of these reviewers and the findings by the California courts, the Panel believes it would be appropriate for the IAQ Report to remove any reference to DEHP as a possible or probable human carcinogen.

C. Exposures to Indoor Air Concentrations of DEHP Are Far Below the California No Significant Risk Level

Even assuming that DEHP could be a human carcinogen, potential exposures from reported indoor air concentrations would not pose a significant risk of cancer.

OEHHA has recently reviewed the carcinogenicity data for DEHP and revised the No Significant Risk Level (NSRL) to 310 ug/day.⁷ Table 1 shows a maximum reported indoor air concentration for DEHP of 3100 ng/m³, or 3.1 ug/m³. For a person breathing 20 m³ a day, the exposure would be 62 ug/day, well under California's NSRL. Therefore, under California standards, DEHP in indoor air cannot be considered to pose a significant cancer risk.

D. Other Phthalates Are Not "Known Indoor Air Carcinogenic Pollutants"

The draft IAQ Report notes that the 1994 California Comparative Risks Project estimates "did not include all known indoor carcinogenic pollutants (. . . other phthalates were not included, for example)" (p. 33, *see also* pp. 82 and II-3). There is not justification for indicating that other phthalates are known indoor carcinogenic pollutants. The Panel strongly

⁷ See Notice of Modifications to Text of Regulations Title 22, California Code of Regulations Sections 12705 and 12805 (08/24/02), at http://www.oehha.org/prop65/CRNR_notices/FSR12705_82302.html.

believes that the references to “other phthalates” should be removed from the statements about other carcinogenic pollutants in the final IAQ Report.

No phthalate other than DEHP has been classified as a known or probable human carcinogen. EPA classified BBP in 1987 as a possible human carcinogen based on effects seen in one sex of one species, but in 1999, IARC determined that BBP should be classified as Group 3, “not classifiable as to human carcinogenicity” (IARC, 1999). High doses of DINP have produced tumors in rats and mice, but a panel of experts convened by the Consumer Product Safety Commission (CPSC) concluded that human doses of DINP are not plausibly associated with a significant increase in cancer risk (CHAP, 2001), and the CPSC staff have concluded that “DINP is not likely to present a cancer risk to humans” (CPSC, 2003). A two-year dermal toxicity study of diethyl phthalate by the National Toxicology Program found no evidence of carcinogenic activity in rats and only equivocal evidence of carcinogenic activity in mice (NTP, 1995).

Thus, there is not an adequate basis for stating that other phthalates are known to be carcinogenic indoor air pollutants. To the contrary, the evidence suggests that, like DEHP, other phthalates are not likely to pose a risk of cancer to humans.

CONCLUSION

For the reasons discussed herein, the science does not support an assertion that phthalates in indoor air pose a substantial risk to human health. To the contrary, reported concentrations of phthalates in indoor air would result in exposures far below health benchmarks designed to be protective of human health. The Panel therefore urges CARB to remove phthalates from the Indoor Air Quality Report altogether, lest resources be diverted to control substances that evidence indicates pose no substantial health risk. If CARB continues to include phthalates in the IAQ Report, it should revise its statements about phthalates in accordance with these comments, and should provide readers with perspective on the very low risk posed by phthalates in indoor air.

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