

**USGBC TSAC PVC Draft Report dated December 17, 2004 (released 12/22/04)
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Submit to tsac@committees.usgbc.org, any time before midnight on February 15, 2005.

Comments submitted by:

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General comments on the committee approach:

The committee used a novel, non-validated method for analysis. Their reasons for choosing this method are unclear. Other options exist but why they were rejected is unknown. The committee's commitment to this method required certain sacrifices that affect that ultimate outcome. For example, whenever the methods do not accommodate the data or require data that are not available, the authors are forced to 1) ignore some data 2) make assumptions that may not be warranted. No sensitivity analysis is provided that gives a sense of what the impacts of those maneuvers might be. For example, what happens to the analysis if you DO consider mercury? What happens if you DO consider all solvents in the resin industry? What happens if you DO consider phthalates and asthma? And so forth.

The committee was apparently so intent on integrating occupational with general population impacts that they adapted the tools for their purposes, and in doing so, used them inconsistently. How (or where) they actually incorporated general population concerns in the integration remains a mystery from the data given.

General comments on TRACI:

TRACI acknowledges large uncertainties in input parameters. It estimates a dose per unit release rate and assumes that that dose is equivalent throughout a large population. It makes no allowance for disproportionately exposed individuals who exceed that estimate because of where they are. (Landscape effects will be more or less important depending on the chemical of interest and manufacturing circumstances)

TRACI is meant to provide ranking and relative comparisons of chemicals rather than absolute risk. In doing so, TRACI acknowledges that estimating absolute risk is virtually impossible to do with validity because of multiple sources of uncertainty. Yet, the committee removed human cancer and non-cancer health effects from the TRACI analysis and attempted an absolute risk assessment. (No details are provided in the document to show any of the numerical values used or assumptions or simplifications). The authors justify that approach by saying that it provides a method for integrating the population wide health effects with the occupational health effects. However, the uncertainties in the population-wide analysis are so large that this integration becomes virtually meaningless.

I am including some comments below on specific sections of the draft report. Some are included as examples of the weaknesses and limitations in the committee’s general approach. These weaknesses strike at the core utility of the chosen method. As an exercise this project serves the purpose of showing that the method is not suitable to the task at hand.

Comments:

Page #	Line #	Comment	Supportive citations
10	4	: “Studies show...” Comment: The statement in the executive summary suggests that there is more than one study in people. Only one study has been done and this is cited in the report. (Rais-Bahrami) A group of children who had undergone ECMO while infants were assessed after attaining puberty for characteristics of sexual development. The study reported no differences in sexual development or hormone levels. The limits of this single study are not discussed to any appreciable degree in the draft text. In this study, no DEHP exposure data were available for any of the study participants. Having undergone ECMO was simply used as a surrogate for exposure. Hence, children who would have had relatively little exposure to DEHP were considered “exposed” along with those who may have had higher exposures. This approach will certainly lead to exposure misclassification. All data on hormone levels were reported as averages, and as within normal limits. At the ages of these adolescents, hormone levels vary considerably and minor variations in hormone	

		<p>levels that might be attributable to early life exposures would not be discernable using this approach. Moreover, as mentioned in the text, sperm counts were not evaluated in this group. In summary, there are no studies of humans that are of sufficient quality to determine whether or not infant exposure to DEHP from medical devices causes adverse impacts on the developing reproductive tract.</p> <p>[Recognizing this data gap, the National Toxicology Program’s Committee on Environmental Risks to Human Reproduction (NTP-CERHR) concluded that the available animal data are relevant to humans, as did the FDA in their safety assessment of DEHP]</p> <p>It is noteworthy that the committee highlights this [single, negative] study as “studies” in the executive summary, while dismissing positive studies addressing other health effects—e.g. asthma and phthalates (see below)</p>	
16	10	<p>“The most important type of morbidity is cancer, as it more likely results in the individual’s death.” This is a value judgment that ignores several important factors. Many kinds of cancer do not lead to death. In fact, concentration on cancer mortality data rather than incidence data frequently gives a skewed impression of population wide cancer burdens. Moreover, non-cancer health effects often effect far more people in a population than cancer and in the end, represent a greater public health threat.</p> <p>Unfortunately, we have no reliable tracking data for the incidence or prevalence of non-cancer health effects such as asthma, infertility, neurological problems, and the like. Even birth defect monitoring is strikingly deficient in most states. As a result, it is virtually impossible to draw any conclusions about the true incidence or prevalence of most non-cancer health effects in specific locales.</p>	
Sect 2.3		<p>TRACI. LCA: Ecotoxicity uses 2,4 D toxicity equivalence for comparison of building materials. Whereas using a common metric for comparison purposes is an advantage for computational purposes, it satisfies the needs of modelers and life cycle analysts far better than it reflects underlying understanding of</p>	

		toxicology. From a toxicologic perspective, it makes no sense to use “2,4-D equivalency” as a way to compare the toxicity of such diverse compounds. The toxicity of the chemicals of concern in these building materials is chemical-specific and cannot be reduced to a single metric. The health impacts of these compounds on aquatic and land based organisms are far too diverse.	
23	1-40 ff	Switched to intake approach for “absolute risk” as opposed to “relative risk” as would be obtained through TRACI. The committee appears to have done this in order to integrate RA with LCA. But it implies far more certainty than is justified. E.g, 24-16 suggests that this “absolute” risk is a valid estimate. The intake fraction is estimated for an entire population and is almost certain to lead to an underestimate of risk for the most highly exposed. Exposures are not equally distributed through large populations.	
23	32	Intake fraction: It distributes the intake over the entire population, which dilutes it into insignificance. This is simply not the case for many of the chemicals associated with these building materials. Biomonitoring studies show that some people are more heavily exposed than others, depending on their proximity to manufacturing or disposal facilities.	
24	6-18	TRACI does not provide estimates of absolute risk in any meaningful way. It is used for comparing materials but is characterized by assumptions and simplifications that make absolute risk determinations invalid.	Bare, et al. TRACI, The tool for the reduction and assessment of chemical and other environmental impacts. J of Industrial Ecology. Vol 6 no. 3-4)
24	19	Continues to treat mortality as the endpoint for cancer. It is completely unclear how the analysis treats non-fatal cancers.	

24	25	Switching from TRACI approach to non-cancer impacts to an intake fraction based approach spreads exposures over a large population and completely dilutes any effect that might result in disproportionately exposed populations.	
24	36	Using intake fractions (Bennett) for an average person in the US population is irrelevant to this analysis	
25	19	Treatment of chemical groups: The chemicals within each group vary considerably with respect to their toxicity. (in some cases, by orders of magnitude). The assumption that the constituents in each chemical group are in the same proportions is completely unrealistic, and averaging TRACI factors for specific chemicals within each group is unjustified. It may be that the information available does not permit another approach, but the necessity of such an unrealistic assumption demonstrates the extremely limited utility of the tool. The assumption is invalid, in which case, the conclusions drawn are also invalid.	
29	32	“Because occupational exposures were the focus of this exercise....” But, for non-cancer and cancer effects in the life cycle analysis, the authors of the report used this method (RA) for the general population (not only workers) as well. (removed from TRACI and attempted an absolute risk assessment)	
29	33	The committee did not review recent toxicity studies. They do not explain why. Such a review is essential to the committee’s approach. Toxicity studies are readily available. The review that they did undertake appears in some instances to be inconsistent and somewhat arbitrary. For example, although the committee says that DEHP is “believed to be carcinogenic” in 29-30, they then cite recent evidence that DEHP is “non-carcinogenic” in 30-18—27. The extent to which the analysis throughout includes or does not include current toxicity data is unclear. Moreover, it is unclear when a study, or collection of studies, reaches a “threshold” for inclusion. (see discussion of phthalates and asthma, for example)	

29	42	Discussion of general population exposure limits for individual compounds: Why is this discussion limited to DEHP? Where are the other compounds? Since the end of life concerns were treated with a risk assessment instead of the TRACI method, where are they in this discussion?	
30	16	If a one order of magnitude difference in risk estimates is not an unacceptable degree of error, it would help if the committee were to describe what degree of error is unacceptable. Thresholds for unacceptable degrees of error and criteria for including or dismissing considerations or studies in this report are notably absent.	
31	10	The discussion of PELs notes that they may be frequently exceeded (31-12,13). This is another obvious reason why the risk assessment should not be considered a valid estimate of absolute risk. The data gaps are substantial and obvious. (see comment on 37-33)	
31	29	Discussion of the “protective” or “conservative” nature of the PEL assumptions: The notion that workers have a chance to “recover from central nervous system effects that are typically caused by many organic solvents (e.g. headache, dizziness, and tingling)” trivializes important occupational health hazards. Moreover, many workers exposed to organic solvents at these levels do not “recover” but rather develop permanent, meaningful cognitive, behavioral, and other neurological disorders.	White, R, Proctor S. Solvents and neurotoxicity, Lancet. 1997 26;349(9060): 1239-43.
31	41	It seems that the primary goal is to compare occupational risks to those of the general population. That seems to drive the entire approach. It is unclear how or why that became the primary goal of this effort. It is unclear how this goal is equivalent to the charge of evaluating whether or not a non-PVC credit is justified. Moreover, occupational risks are given primacy over general population or end-user risks (see 28-32) in some sub-analyses. These variable emphases appear to be completely arbitrary or forced by limited availability of data, yet the committee ignores these limits when discussing their “successful” integration of occupational with general population risks.	

33	34	<p>The decision to ignore phthalate emissions from vinyl products in residences, schools, etc. is unjustified. Any consideration of the causal relationship between phthalate inhalation via air or contaminated dust and bronchospasm/asthma is completely lost from the analysis.</p> <p>Butala et al show no IgE response, but Larsen shows adjuvant activity that may provide insight into mechanisms.</p> <p>It is striking that the committee dismisses the accumulated data addressing this issue by critically examining the limitations of the various reports while failing, for example, to bring the same critical analysis to the limits of the single report of sexual development in children exposed to ECMO during infancy.</p> <p>The committee’s criteria for including or excluding a study are unclear. An overview leaves one with the distinct impression that negative studies are more likely to be included in the analysis than positive studies.</p>	<p>DEHP dust levels and asthma (Bornehag, Environ Health Perspect 2004)</p> <p>PVC wall coverings and wheezing (Jaakkola, AJPH, 2000)</p> <p>DnOP, DINP—adjuvant for IgE response to ovalbumin in mice; SC injection (Larsen, Pharm Toxicol 2002)</p> <p>DEHP alone; dermal; no IgE response, mice (Butala, Toxicol 2004)</p> <p>Inflammatory response via prostaglandin pathway (Oie, Environ Health Perspect 1997)</p>
33	26	<p>“End users” do not include inhabitants of residences in this report. This is an obvious and significant omission, justified by lack of data about the relative contribution of various sources to environmental levels. This omission again serves to illustrate the limits and weakness of this approach. It’s difficult to imagine support for an analysis that does not consider the impacts of some of the building materials on the people who live in the buildings.</p>	

34	5	<p>The report cites Clausen et al with respect to DEHP emissions from PVC flooring. Clausen et al studied DEHP emissions in experimental chambers and noted that the dust layer increased emission rate by increasing the concentration gradient above the surface of the PVC. Clausen et al concluded “it was difficult to obtain reliable emission data for DEHP from the PVC because of the sensitivity to the test conditions.” “ The relevance of gas-phase emission data of DEHP in relation to indoor climate must be reevaluated since particles and dust on the PVC surface increase emission. Thus, inhalation of resuspended dust may be the most important exposure route.”</p> <p>Although it is true that the total exposure to DEHP via this route may be less than the reference dose for DEHP, this exposure pathway is likely to be extremely important when considering the potential for DEHP-inhalation to cause exacerbations of asthma, bronchospasm, or other respiratory symptoms. (see comment on 93-40) Moreover, inhalation exposures add to aggregate phthalate exposures which are of course what’s important to an organism.</p>	
Sect 2.5.5		<p>The discussion of uncertainty analysis is virtually silent about the assumptions that have been previously made with respect to the justifications for omitting certain data. (see, for example, the rationale for ignoring “end users.”) The impacts of these omissions on the final conclusions are not explored.</p>	
37	33	<p>Here PELs are claimed to be “protective”. The committee, however, previously acknowledges that PELs are frequently exceeded (see comment 31-10)</p>	
38	34	<p>“The committee did not undertake a comprehensive comparison of the various options for all applications.” This important limitation should be made explicitly clear in the final report, executive summary, and conclusions.</p>	
39	1-6	<p>The assumption here is that the approach that the committee did use will serve as a valid screen for any clear trends. A valid “screening analysis” must err on the side of “false positives”. If a screening analysis is biased toward “false</p>	

		negatives” it serves little purpose except to impede more definitive investigation. The approach used by the committee is characterized by many limiting assumptions, data gaps, and restricted analyses. Though the committee believes some assumptions to be “conservative”, numerous comments above indicate examples of assumptions that are invalid and that are likely to bias the analysis toward finding “no trend”. (e.g averaging toxicity values in classes of chemicals, omitting end users from the analysis, ignoring reports of biological effects from inhaled phthalates, etc.)	
47	1-12	Note that no studies have been done on fetal or infant primates, and therefore no data to support any conclusion about saturation of metabolic pathways or GI uptake of phthalates in this age group.	
47	1-29	General population exposures to DEHP from all sources are probably higher than previously estimated. Newly discovered metabolites of DEHP, which are present at higher concentrations than metabolites previously identified, imply significantly higher exposures to the parent compound than previous estimates. Any additional source of phthalate exposure must be considered in the context of cumulative, aggregate exposures.	Preuss R, et al. Biological monitoring of the five major metabolites of DEHP in human urine using column-switching liquid chromatography-tandem mass spectrometry. J Chromatogr B Analyt Technol Biomed Life Sci. 2005; 25;816(1-2):269-80.
47	15	See comment on 10-4 in executive summary.	
49	33	Reducing total emissions may also be achieved by materials use policies that avoid the problem completely rather than controlling emissions through “command and control” technologies. Progressive materials policies that move the industries to fundamentally cleaner materials should be a primary goal of LEED.	

51	35	<p>The discussion implies that the dioxin on the foliage of trees “disappeared”. That dioxin will predictably be found and remain in local soils and represents an ongoing source of exposure.</p> <p>Notably absent from the above is any discussion of landfill fires as a dioxin source. Debris from building demolition or construction is likely to be disposed of in landfills where it may catch fire and burn in conditions that foster dioxin/furan formation.</p>	
53	11-30	<p>Failure to account for exposures to some solvents in the resin manufacturing industry because of lack of exposure information is likely to lead to significant under-recognition of effects unique to the resin industries. For example, methyl ethyl ketone is an irritant and potentiates the neurotoxic impacts of other solvents.</p>	<p>Methyl ethyl ketone ACGIH. Documentation of the threshold limit values and biological exposure indices. Vol:7th ed (2001).</p>
90	13	<p>Potential neurodevelopmental toxic effects of mercury are omitted from the analysis “because the impact of metals on the general public is not well addressed using current TRACI methodology.” Inasmuch as the 65 tons of mercury “lost track of” is a problem “unique to the manufacture” of PVC, and the neurodevelopmental toxicity of mercury is a matter of significant public health importance, the committee’s deference to TRACI’s shortcomings suggests that the method is driving the analysis. If the method is unable to accommodate the realities, an alternative method should be found. Omission of mercury from the analysis is one more example of the analytic limits of this report.</p>	
92	4	<p>See comments above. This Rais-Bahrami study is extremely limited because of its lack of exposure assessment in individuals and lack of measurement of sensitive endpoints. No conclusions can be drawn from this study for the purposes of this report.</p>	
92	23	<p>Although additional studies are necessary to fully understand species differences in tissue sensitivity and toxicokinetics of DEHP, the NTP panel, Health Canada, and the FDA have independently concluded that animal studies of the reproductive and developmental effects of DEHP are relevant to humans. Absorption of DEHP from the gastrointestinal tract of primates and humans may</p>	<p>NTP-CERHR FDA—Safety assessment of DEHP Health Canada—Safety assessment of DEHP</p>

		be limited by saturation at higher doses but not at lower, environmentally relevant doses. This possibility is discussed in the NTP and FDA documents, but is not mentioned in the present document.	
93	40	It appears that the risk assessment and LCA failed to include any consideration of the potential link between phthalates and asthma. The report discusses the various studies of humans showing a link between PVC building products in the indoor environment and asthma in children. The reports notes that an animal study found no IgE production after phthalate exposure and that therefore, it is unlikely that phthalates would contribute to allergic asthma (IgE mediated). However, the report fails to comment on the possibility that the children described in the various studies may have had non-IgE mediated (non-allergic or non-atopic) asthma, a condition that is well-known. Non-IgE mediated asthma or bronchospasm resulting from phthalate exposure is a condition that is supported by the cited documents. It appears that this potential impact was ignored in the risk assessment or in the uncertainty analysis.	See references above on the same topic