

The Environmental Health Impacts of Polyvinyl Chloride (PVC) Building Materials



a briefing paper for the U.S Green Building Council
Final Rebuttal

December 1, 2000

OVERVIEW

This document is the final rebuttal, filed by the Center for Maximum Potential Building Systems and the Healthy Building Network, in response to the U.S. Green Building Council's invitations for comments on proposed Materials Credit 9 for LEED™ Commercial Interiors. The document, consisting of five independent papers, identified as Section I-V and 4 supporting documents identified as Attachment 1-4, specifically responds to initial position papers and rebuttals submitted by the Vinyl Institute and other parties on November 3, 2000 and November 20, 2000, respectively.

The USGBC's deliberations over PVC are timely. They occur as the Danish government has acted to prohibit lead stabilizers in all PVC (November, 2000), and within weeks after the release of a report by the Worldwatch Institute, *Why Poison Ourselves: A Precautionary Approach to Synthetic Chemicals* examines the environmental impacts of PVC. Worldwatch Senior Researcher Anne Platt McGinn notes that "Every stage of that [PVC] lifecycle --from manufacture, to use, to disposal -- creates dangerous chemicals, including some of the most notorious POPs" (McGinn 49). She concludes: "Of course, the more effective strategy over the long term will be to identify whole material substitutes for PVC. . . . Fortunately, not all plastics are as bad as PVC." (McGinn p.59). We have attached a copy of this report as Attachment 2. A summary of the sections and attachments follows.

Section I addresses the question of whether the proposed language of Materials Credit 9 falls within the scope of the USGBC mission and the goals of the LEED™ program.

Section II addresses the question of whether Life Cycle Analysis is the appropriate tool for establishing materials standards within the LEED™ program.

Section III is a comprehensive response to the rebuttal arguments submitted by the Vinyl Institute on November 20, 2000.

Section IV is a specific rebuttal to the submission from the American Chemistry Council's Phthalate Esters Panel that was referenced in the Vinyl Institute's submission of November 20, 2000.

Section V addresses the extent to which vinyl is being recycled in the United States based upon a survey of the firms listed in the Vinyl Institute's Directory of U.S. and Canadian Companies Involved in the Recycling Vinyl Plastics.

In addition to Attachment 1, which offers brief biographies of the principle authors of the major sections of this rebuttal, this submission also includes 3 other attachments that offer further independent corroboration of our arguments.

Attachment 2 contains a copy of the brand new Worldwatch report, *Why Poison Ourselves? A Precautionary Approach to Synthetic Chemicals*.

Attachment 3 contains copies of letters from firefighters in support of safer alternatives to PVC building materials.

Attachment 4 is a review of Dr. Thornton's book, Pandora's Poison: Chlorine, Health and a New Environmental Strategy from the scientific journal, *Nature*.

A final note to the reader. The information presented in this document is authoritative and accurate. We have taken great care to ensure that arguments do not overlap between or among papers, and that to the extent possible, the papers' formats are consistent, well edited and easy to read. However, we were constrained in these efforts by the failure of the Vinyl Institute to submit their first rebuttal in either a timely manner or in the prescribed electronic format. We received one hard copy of their voluminous submission two days late, after office hours, on November 22nd, the day before the Thanksgiving holiday. This made widespread distribution of their papers to the authors of this document impossible until the Monday preceding the established December 1st deadline for this final submission. It took considerable effort for the authors of this to meet both this deadline and their own professional standards under these highly unprofessional circumstances. For this we owe them a debt of gratitude and deep appreciation for their dedication to the cause of protecting environmental health

Section I . Materials Credit 9 is consistent with the emerging concepts and standards used in LEED™ for similar materials, and should be retained: A rebuttal to arguments contained in the Vinyl Insitute’s submission of November 3, 2000, including a proposal for amending the credit language.

**Barbra Batshalom
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November 30, 2000**

The Mission and Vision of the USGBC and LEED™

The Material Credit 9 proposed for adoption in the U.S. Green Building Council’s Commercial Interiors LEED™, has emerged as a pivotal litmus test of USGBC’s leadership role. Throughout its public pronouncements, leadership and advocacy are cornerstones of USGBC’s central mission.

•In its promotional materials, the U.S. Green Building Council defines itself as

“...the building industry’s only balanced, non-profit, consensus coalition promoting the understanding, development, and accelerated implementation of Green Building policies, programs, technologies, standards and design practices.”

• USGBC’s Chairman, Steven Winter, elaborates further in identifying two key roles provided by the organization: providing services to its members and advocating green building practices. Specifically, on the second point, he remarks that

"The second role is to pursue the USGBC's advocacy interests: we advocate green building practices that will result in a healthy planet. Many of our members, like the Natural Resources Defense Council and the Rocky Mountain Institute, participate in the Council because it mirrors their own advocacy policies, and provides an opportunity to pursue them in collaboration with private sector organizations. If you or your organizations also want to improve the health of the planet and its inhabitants by helping buildings become friendlier to the environment, the USGBC is the place to learn about it and to do it, through such vehicles as publications, conferences, and setting standards."

• USGBC’s signature initiative, LEED™ (Leadership in Energy and Environmental Design), could not be more explicit in defining the USGBC as a vehicle for leadership. In particular, LEED™ is presented as a tool for market transformation.

• USGBC CEO Christine Ervin writes that the USGBC is committed to “setting a high bar for expectations” and “accelerating progress”. She cautions against “lowest common denominator results” in a context of “taking responsibility for the next industrial revolution.”

Adoption of Material Credit 9 to eliminate the use of PVC is entirely consistent with

USGBC's mission and vision, and appropriately reinforces USGBC's leadership role among green building practitioners, proponents, and policymakers. Taking leadership positions in addressing controversial issues has enhanced USGBC's credibility and promoted the adoption of LEED™. Indeed, healthy dissent and active dialogue have been distinguishing features of the USGBC process; these have galvanized, not jeopardized, the organization's integrity since its inception.

According to the USGBC Bylaws, "any drafts or works representative of the Council must be balloted by its members, and every written comment that the Council receives on its submitted work must be resolved." The proposed credit is therefore subject to member approval, and will only be adopted through that process. Many ways of disseminating information on the LEED™ process exist, such as a list-serv for members, that allows for open and ongoing discussion of any issues involved in LEED™.

PVC is not the first material to be singled out in a LEED™ standard. Asbestos, tobacco smoke, CFCs, HCFCs and Halons are all addressed in LEED™ 2.0, while urea formaldehyde is specifically referenced in the draft LEED™ CI. And, while LEED™ references third-party standards whenever available, no third-party standards are referenced for many of these materials, including for tobacco smoke, CFCs, HCFCs and Halons. In referring to tobacco, CFCs, HCFCs, and Halons, the LEED™ 2.0 Reference Guide indicates "there are no referenced standards for these credits". Instead, the inclusion of these materials in LEED™ reflects emerging concepts and international concerns. The proposed Material Credit 9 is similarly positioned and would be similarly referenced, identifying a material, like the aforementioned, that singularly represents unacceptable risks to ecological sustainability and human health and that, fundamentally, is inconsistent with green building practices.

The question therefore hinges not on the degree to which PVC is harmful, but on whether the use of toxic, carcinogenic, energy-intensive materials such as PVC should be discouraged in Green Building guidelines. We submit that the proposed Material Credit 9 to eliminate the use of PVC resonates with the USGBC's mission and vision, contributes to its credibility as a resource for the building industry, and enhances its leadership position. Furthermore, the proposed credit conforms to the consensus-based and voluntary principles of LEED™ and is consistent with the emerging concepts standards used in LEED™ for similar materials. We therefore urge the Council to include this credit in the LEED™ Commercial Interiors document.

The Language of Materials Credit 9

Materials Credit 9 is logical, understandable, and reflects the practical experience of its authors. We are confident it would prove to be practical and achievable if included in the pilot version of LEED™ Commercial Interiors. The pilot phase provides ample opportunity to adjust for any problems or confusion that might emerge before finalizing 1.0 CI.

We are confident because we know that, in practice, many practitioners already single out vinyl as a material to exclude in order to elevate a building's environmental performance. We have also documented the successful actions taken by local, state, national, and regional governmental bodies to restrict PVC consumption for environmental reasons, as well as numerous commitments to reduce and/or eliminate the use of PVC building products in the private sector (Thornton, 16-19). To the extent that substitution strategies may prove impractical or unachievable in specific applications, pilot testing provides the appropriate mechanism for refinement. Therefore, we see no rational basis for eliminating this credit.

In its November 30, 2000 submission to the Committee, the Vinyl Institute does make several cogent points with regard to ambiguities in the language of Materials Credit 9. We disagree with their conclusion however, that the goals of the credit are impractical and that it must be withdrawn. To the contrary, we believe that these ambiguities could easily be addressed in technical guidance documents, and that the practicality of the credit is best tested through the pilot process.

Alternatively, minor modifications to the credit language would clarify and improve upon the credit, without losing any of the original intent, and provide an equally sound basis upon which to test the practicality of the credit in the pilot process. If the Committee chooses to amend the language of the credit, we suggest the following.

First and foremost, we recommend that the PVC materials credit be separated from the requirement referencing IARC-listed chemicals. The two requirements involve fundamentally different and possibly inconsistent analyses, especially in consideration of some of the legitimate points raised by the Vinyl Institute. Eliminating a finish material such as PVC involves a simple and straightforward process of seeking alternative materials that are not otherwise proscribed by LEED. The listed chemical requirement necessarily involves a more sophisticated and widespread analytic process which sweeps in not only finish materials, but also solvents, adhesives, resins, etc. Bifurcating these requirements into distinct credits places an appropriate emphasis on two different but important considerations in specifying commercial interiors.

Second, we agree with the Vinyl Institute's assertion that the current language is ambiguous relative to virgin vs. recycled content PVC. If the language is to be amended, this credit should drop the distinction of "virgin" and instead be written to prohibit all PVC products. Recycled PVC harbors most of the impacts and potential impacts of virgin PVC (e.g., uniquely severe environmental hazards at the point of initial production and during accidental or deliberate combustion). Current market trends confirm that many companies are developing and manufacturing PVC-free products including for carpet tile backing, wallcoverings, resilient flooring, to name a few. To offer equal credit to recycled PVC products penalizes the industry leaders and subverts the USGBC goal of driving markets and accelerating progress.

Third, with regard to the elimination of "all chlorinated plastic compounds," we believe this language to be essentially redundant. PVC plastic represents the

overwhelming majority of chlorinated plastics under any definition. To the extent that some chlorinated compound might be present in another plastic, this could be difficult for LEED™ users to discern. In any case, it would represent a negligible chlorine contribution.

Fourth, the Vinyl Institute identifies legitimate concerns with regard to those who opt to navigate through the listed chemicals requirement. Common sense dictates that the drafters of the credit did not intend to require the elimination "any product whose production may produce a listed chemical as an intentional or unintentional by product" (VI. p.3). A straightforward interpretation of the requirement to mean the elimination of the direct use of the listed chemicals can be achieved by inserting the word "direct" before the word "use" in the current language.

To conclude, should the Committee choose to modify the language of Materials Credit 9 we suggest the first that the credit be retitled "Materials Credits" (dropping the word "alternative" since the credit attaches to the elimination of bad things, rather to the prescribed selection of "alternative" things). Second, the following language should distinguish between two distinct credit opportunities:

- 1) "Eliminate the use of PVC and PVC composite materials." (1 point)
- 2) "Eliminate the direct use of any chemical listed in the following: . . ." (1 point)

With these amendments, the would be more clear, without losing any of the original intent., and provide an equally sound basis upon which to test the practicality of the credit in the pilot process

Section II: Life Cycle Assessment is An Inappropriate Tool for Identifying Materials for Deselection: A rebuttal to arguments in the Vinyl Institute's submission of November 3, 2000.

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We vehemently challenge the appropriateness of life cycle assessment (LCA) to be used as the arbiter for material decisions for USGBC's LEED™ as suggested by the Vinyl Institute's Timothy Burns. While LCA provides a valuable framework to understand the complex of impacts associated with a given material or product through its "life", to bring attention to specific stressors, and to reveal relative advantages and disadvantages between specific materials, current methodological deficiencies render it seriously flawed as a scientific basis for decision making. Chief among these are inadequate data, lack of consensus on methods for impact assessment, and an overall lack of standardization across the entire LCA process. Further, LCA is an inadequate tool for LEED™ as it is a step removed from the research that documents the hazards associated with chemical use and the actual impacts of those chemicals on human and environmental health (Owens, 1997; and Todd, 1999). Indeed, LCA has never been used as the sole tool for deciding how to manage a material in public policy, even in the Netherlands, the country with the most experience using LCA (Ehrenfeld, 1997, and Stevels, et al., 1999)

Reflecting these concerns, Stevels, et al., conclude that "Due to a lack of standardisation LCA is not yet appropriate for external comparisons or absolute calculations" (1999, p. 21). We are, in fact, years away from having an "adequate comparative life cycle assessment" as desired by Mr. Burns, a fact that even he acknowledges: "While, admittedly, it is difficult to obtain complete life cycle assessments of competing products or materials, great progress is being made in this area."

Methodological Flaws

A quantitative LCA includes three stages: inventory analysis, impact assessment, and improvement assessment (Consoli, et al., 1993). While there is general consensus on the LCA inventory methodology, there are no U.S. databases acknowledged to be of sufficient quality to serve as a basis for public policy. And, although good proprietary databases exist, these are inappropriate as a basis for public policy or LEED™ unless users have access to the databases at low or no cost. (Todd, 2000) In Europe, for example, where a handful of proprietary databases are on the market (Vezzoli, 1999), the data remain in private hands and closed to public scrutiny (Stevels, et al., 1999).

Further, both the impact and improvement assessment stages are still quite crude. An impact assessment is required to make a choice among materials by aggregating impacts into a single number requiring data normalization and impact category weighting. A

quantitative assessment that distills comparative impacts into a single number, while simplistically elegant, is filled with pitfalls.

Indeed, among LCA experts there is agreement that impact assessment is filled with so many choices on the part of the analyst that it is very subjective. For example, Graedel, et al. (1995), conclude that "impact analyses are inevitably contentious, in part because they involve value judgments in comparing and balancing different impacts" (p. 134A). Given that value judgments are a clear part of the impact assessment process, a challenge for using LCA in the public sphere is addressing the multiplicity of values that participants bring to the table. And the values that prevail in the weighting scheme will affect which materials are considered environmentally preferable.

Due to these and other problems, Owens concludes that "LCA will not provide definite, overall answers that many users may desire or clear unequivocal answers for some impact categories. This significantly limits LCA's ability to conduct an overall or comprehensive assessment and to compare alternatives" (Owens, 1997, p. 48). The clearly value-laden impact assessment process means that we will not achieve Mr. Burns' goal of a "science-based decision making" process if we rely on comparative LCAs for material decisions.

In addition to the value-laden impact assessment stage, LCA in general, as a decision making tool, is still in its infancy. A result is that data, methods, and assumptions are not standardized across LCAs. This has led Stevels, et al., to conclude that "Due to a lack of standardisation LCA is not yet appropriate for external comparisons or absolute calculations" (1999, p. 21).

Since LCA impact assessment methodology has not reached consensus, its use in LEED™ would mean the USGBC would have to decide which impact categories to include and which weighting system to impose. These already challenging tasks would be complicated further by the fact that many argue that an appropriate weighting system must take regional conditions into account for many impacts (Todd, 1999).

BEES

The methodological flaws identified above also extend to BEES™ (Building for Environmental and Economic Sustainability), developed by the National Institute of Standards and Technology (NIST) and the U.S. Environmental Protection Agency (USEPA), another decision-making tool suggested by Mr. Burns. Mr. Burns states that a BEES™ study powerfully affirmed "the importance of systematic, multidimensional, life cycle studies in determining the true environmental attributes of competing products", and implies that BEES™ be a basis for material decisions in LEED™. The study referred to by Mr. Burns found vinyl tile environmentally preferable to natural linoleum. The BEES™ 1.0 software used in the referenced study has been acknowledged to have serious flaws, some of which extend in to the current 2.0 version.

† **Data Scope and Quality.** In version 1.0 data were missing on the impacts of mining and forestry operations and on highly toxic emissions such as endocrine disruptors (EBN, 1998). In version 2.0, EBN noted that the "data quality problems range from seemingly simple mistakes to larger concerns about whole sets of data collected from different sources without peer review" (EBN, 2000, p. 18).

† **Questionable Assumptions.** Relevant to the linoleum versus vinyl tile debate is both materials were assumed to have the same useful life of 18 years. However, Spiegel and Meadows (1999), for example, estimate that vinyl tile has a useful life of 8-15 years compared to 40 plus years for linoleum. Also linoleum was assumed to have twice the maintenancerequirements as vinyl tile. Wilson (1998) estimated that the opposite is true. Because linoleum does not require periodic stripping and waxing, annual maintenance costs are projected to be almost \$1.00 less per square foot for linoleum (\$0.50 per square foot for linoleum and \$1.45 per square foot for vinyl tile) (p. 13).

† **Lack of Transparency.** "Hard numbers are difficult if not impossible to come by for most of these processes, so the data are inevitably derived by various approximations. Yet the program provides scores to the second decimal point, without any guidance as to which, if any, numbers might justify that level of accuracy" (EBN, 1998, pp. 14-15).

Transparency problems extend to Version 2.0. EBN found that formulas that should explain how data are used are not available, much of the upstream data is proprietary, and verifying even simple questions about data sources was difficult (EBN, 2000). The use of proprietary data should stand as a red flag for using the data in LEED or public forums because the accuracy of the data cannot be independently verified.

The Version 1.0 problems highlight a danger of LCAs in policy-making decisions. A program, embedded with many assumptions and data gaps and lacking in transparency, cranks out a result that indicts or exonerates a material; that result is broadcast to the public despite widely acknowledged limitations of the assessment. While the result is suspect and everyone knows it, it is now the de facto standard for defining that material's environmental performance. This is a reason to use ecological, epidemiological, and toxicological data, as described below, rather than LCA, for deselecting a material.

Another flaw in Version 1.0 that carries over into Version 2.0 is in the impact assessment, which includes data normalization and weighting. As always with an impact assessment, the weighting scheme employed significantly affects the final numbers. In an Environmental Building News interview describing his review of the BEES™ impact assessment methodology for the U.S. EPA, Greg Norris explained that "if the weighting system determines that global warming impact is twice as important as human toxicity, greenhouse gases will have twice the affect on the final score, even if ... they are released in miniscule quantities, while toxic emissions are huge." This is because the scoring of a material is done in comparison to another material rather than with reference to an outside benchmark or absolute measure of impact (1999, p. 4). Norris' (1999) findings are consistent with other critiques of LCA. For example, Owens (1997) found

that impact assessments can "yield contradictory, conflicting, and incompatible results" (p. 46).

BEES is illustrative of the flaws that exist in LCAs in general. Given these flaws quantitative, comparative LCAs should not be touted as effective tools for comparing materials until there is a standardized impact assessment methodology. To reiterate a point noted above by Stevels, et al., "Due to a lack of standardisation LCA is not yet appropriate for external [public] comparisons or absolute calculations [emphasis added]."

Use a Weight-of-Evidence Approach

Materials identified in LEED™, either for selection or deselection, should be based on a weight-of-evidence approach rather than a comparative LCA approach. The weight-of-evidence approach is more in keeping with the process by which legislative and governmental bodies identify toxic chemicals for serious reduction or phase-out and is based on toxicological, epidemiological, and ecological studies. This is an approach scientific, legislative, and regulatory bodies have used to make toxic chemical policy recommendations and decisions.

For example, the International Joint Commission (IJC) took this approach when it recommended that the U.S. and Canadian governments "adopt and apply a weight-of-evidence approach to the identification and virtual elimination of persistent toxic substances." As the IJC reasoned:

At some point, the emerging mass of data and information must be accepted as sufficient to prompt or, in the case of the [Great Lakes Water Quality] Agreement, ratify action against environmental contaminants. Therefore, the Commission has adopted a "weight-of-evidence" approach. Taking the many studies that indicate injury or the likelihood of injury together, we conclude that the evidence is sufficient that many persistent toxic substances are indeed causally involved, and there can be no defensible alternative: their input to the Great Lakes must be stopped (IJC, 1992, p. 22).

Rare, likely non-existent, is a case in which toxic chemical policy was set based upon a comparative LCA. Rather when legislatures or governmental bodies identify a chemical for phase-out, they rely upon toxicological, epidemiological, and ecological research. Examples abound.

† When the Governing Council of the United Nations Environment Program (UNEP) issued a mandate in 1997 to protect human health and the environment from

persistent organic pollutants (POPs) by phasing-out their use, the decision was based on the fact that POPs are toxic, persistent, bioaccumulative, and biomagnify up the food chain. As UNEP (1999) states, "POPs pose an international risk to public health and the environment which no country acting alone can solve" (p.2). Its decision was not based on a LCA.

† When the Swedish Parliament enacted a new environmental policy bill (Environment Bill, 28 April 1999) that included provisions defining environmentally preferable products (Annex 1, Further Guidelines for a Chemicals Policy), it was based on criteria critical to sustainability, not on a LCA. It includes the following provisions:

"New products introduced onto the market are [shall] largely [be] -- Free from man-made organic substances that are persistent and bioaccumulative, and from substances that give rise to such substances and -- free from man-made substances that are carcinogenic, teratogenic and endocrine disruptive - including those which have adverse effects on the reproductive system."

...

"All use of phthalates and other softeners with hazardous or suspected hazardous effects on health or environment should be phased out by voluntary measure."

The Swedish Parliament defines environmentally problematic materials as persistent, bioaccumulative, carcinogenic, teratogenic, endocrine disruptive, or toxic to the reproductive system. It does not state that environmental problematic materials should be identified based on a LCA.

† When the US Congress passed the Clean Air Act Amendments of 1990, which included provisions to phase-out ozone depleting substances, it did so because of the potential for irreversible damage to the ozone layer, not because of a LCA.

† The USGBC followed the same logic as the US Congress when it developed a LEED credit for not using products manufactured with ozone depleting substances.

Conclusion

While LCA provides a valuable framework to understand the complex of impacts associated with a given material or product through its "life", current methodological deficiencies render it seriously flawed as a scientific basis for decision making. Chief among these are inadequate data, lack of consensus on methods for impact assessment, and an overall lack of standardization across the entire LCA process. These concerns extend to BEES™ which also suffers from methodological flaws. For these reasons, they are inadequate to establish standards for LEED™.

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Section III: Response to Vinyl Institute comments on “Environmental Impacts of Polyvinyl Chloride (PVC) Building Materials”

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Introduction

I welcome the opportunity to respond to the comments of Timothy F. Burns of the Vinyl Industry (VI) on my submission to the USGBC on behalf of the Center for Maximum Potential Building Systems and the Healthy Building Network. I will respond here to the arguments that Mr. Burns raises.¹ I conclude that there remains sound reason for concern and action on the health and environmental impacts of polyvinyl chloride.

The aggressive and hostile tone of the VI’s comments is unfortunate, especially because the rebuttal itself contains surprisingly little relevant information or argumentation to support the harsh characterization of our submission. For example, the VI asserts that I have selectively drawn on data that supports my case, but virtually all of the points that Mr. Burns raises in his rebuttal as excluded from my comments were in fact discussed in my original submission. Further, although Mr. Burns alleges

¹ The document we submitted had many citations of the independent scientific literature. For the sake of brevity, I will avoid restating arguments and repeating reference information that was included in that document. Where I refer to or expand upon points made in our first submission, I will cite the relevant pages of our submission where the original discussion with its citations can be found.

“misrepresentations and mischaracterizations of underlying data” and “misstatements and inaccuracies,” I have not found in Mr. Burns’ comments a single specific, supported allegation of data that I have presented inaccurately. Moreover, although the VI claims objectivity, most of the information he cites was generated by the chemical industry itself and is therefore considerably less trustworthy than the independent academic and government analyses on which our submission was based.

1. The nature and purpose of our original submission

In its rebuttal, the VI suggests that “Proposed Materials Credit 9 appears to have been based solely on an undated document entitled ‘The Environmental Health Impacts of Polyvinyl Chloride (PVC) Building Materials,’ prepared by Joe Thornton, Ph.D.” (VI, p. 3). The document that the VI refers to is our submission to the USGBC, contributed to the same process at the same time as the VI made its own submission.

As the committee members know, USGBC’s proposed standard was drafted months ago, based on concerns current in the green building and environmental communities about the health and environmental impacts of vinyl. The committee asked proponents and opponents of the proposed standard to prepare written arguments in defense of their position. In October of 2000, the Center for Maximum Potential Building Systems and the Healthy Building Network, who were asked to coordinate the “pro” submission, requested that I prepare for them a presentation in support of proposed Materials Credit 9 to eliminate the use of PVC. The document was completed on a very short timeline and submitted to the USGBC on November 1, 2000, the same date that the VI submitted its own statement in opposition to the proposed standard.

The VI has argued that our submission is not “objective” or trustworthy because it states as its purpose the presentation of a position in favor of the proposed standard (VI 3-4). I strongly disagree. In contrast to the Vinyl Institute and the American Chemistry Council’s Phthalate Esters Panel, the comments of which were enclosed with those of Mr. Burns, I have no material interest in the USGBC’s decision on PVC. As it was presented to me, the purpose of the deliberative process that the USGBC established was for the committee to receive the arguments on both sides of the matter and, in juxtaposing and analyzing them, to come to an informed judgment on the merits of its proposed standard. In my original submission, I have presented the data that are available on subjects relevant to the environmental and health hazards of vinyl fairly and accurately, and I have pointed out the strength and weaknesses of evidence that ranges from the suggestive to the conclusive. I have not presented arguments in favor of continued vinyl use because they are not, in my view, persuasive; moreover, to do so was not my charge. Conversely, neither the Vinyl Institute’s submission nor its rebuttal presented the arguments that vinyl is hazardous to health and the environment.

We have endeavored to base our concerns on independent studies performed by academic researchers and government agencies. In contrast, the three major publications on which Mr. Burns bases the bulk of his rebuttal— a study of dioxin formation in the

production of vinyl, a study of the link between the feed of organochlorines and dioxin formation in incinerators, and a critique of our comments on the issue of phthalate plasticizers – were all funded and/or conducted by the vinyl or chemical industries.

2. Vinyl versus alternative materials

The VI points out that “all manufacturing processes that extract raw materials from the earth to produce products have environmental burdens and generate toxic or hazardous by-products or wastes. All of these processes consume energy, a manufacturing process that has its own environmental burdens” (VI 4).

It is correct that production of all materials can or does cause environmental impacts. But it is surely not true that all materials have equal environmental costs. The environmental impacts of using sustainably harvested wood products, for instance, are considerably less severe than using wood products harvested from old-growth forests or pesticide-intensive timber farms. In our original submission, we provided a detailed and referenced argument that PVC is “worst-in-class” for two major reasons. First, vinyl is the only major building material that contains organically-bound chlorine; it is therefore the only one that is associated with the formation and release of large quantities of hazardous organochlorine feedstocks and by-products, including dioxins and PCBs, throughout its lifecycle. As discussed in our submission, organochlorines tend to be bioaccumulative, toxic, and persistent – consistently more so than their chlorine-free analogues -- and therefore pose uniquely severe hazards (Thornton 20-23). Second, vinyl is the only major plastic that requires hazardous additives such as heavy metals and phthalates for stability and flexibility. These unique aspects of vinyl have not been refuted in the VI submission or rebuttal.

It is for these same reasons that dozens of national, regional, and local governments, as well as private firms and organizations, have taken action to restrict the use of PVC in building and other applications, as outlined in our original submission (Thornton 16-19). This trend away from vinyl by concerned actors is not refuted by the VI.

No alternative building material is absolutely perfect, and some alternative plastics may also pose unacceptable occupational hazards during their manufacture. The fact remains that no other building material poses environmental hazards of the severity and extent that vinyl does. If the USGBC’s purpose is to set standards for what constitute environmentally sound building practices, it is surely reasonable for the Council to conclude on the basis of the available information that vinyl is not a green building material.

3. Hazards and releases of vinyl feedstocks

Health impacts and occupational exposure. The VI does not dispute that the feedstocks for polyvinyl chloride production – VCM and EDC – are known and

suspected human carcinogens, respectively (Thornton 29). Nor does the VI dispute that these substances are released into the workplace and into the environment as a consequence of PVC production (Thornton 29, VI 4). The VI also does not refute the other health impacts, including neurological toxicity, immune suppression, and testicular damage, that EDC and VCM are known to cause in laboratory animals and highly exposed persons (Thornton 29).

The VI grants that VCM is known to cause liver tumors in exposed workers but argues that occupational exposures to VCM in the United States have been substantially reduced since the 1970s (VI 4-5). We made the same point in our submission (Thornton 30). However, legitimate concerns about worker health due to exposure to VCM and EDC remain, for two reasons discussed in our submission, neither of which were disputed by the VI:

- There is no threshold for some health impacts of EDC and VCM. Both substances are genotoxic; a single molecule can cause irreversible damage to DNA. It is generally accepted by biologists that for genotoxic substances, there is no threshold below which health impacts – including initiation of cancer, birth defects, and genetic diseases – do not occur at some finite probability. Thus, reduced levels of VCM and EDC in U.S. PVC production facilities reduce but do not eliminate the health risks from these potent carcinogens (Thornton 30).
- Occupational exposures to VCM remain very high in other nations, including some in Europe and Asia, posing very high health risks to workers. Because many vinyl products in the U.S. are imported, practices in these nations are relevant to U.S. building decisions (Thornton 30).

The VI argues that there is no “conclusive” epidemiological evidence proving that brain tumors are linked to VCM exposure and cites one study that did not find a relationship between VCM and brain cancer risk (VI 5-6). Our submission did not say the data are conclusive, but the weight of evidence does suggest a link between VCM and brain tumors (Thornton 29). A recent review of all the available well-conducted studies on VCM exposure shows that four of the five studies reviewed found elevated risks of brain cancer among VCM-exposed workers.¹ Because the statistical power of individual studies is limited by the use of relatively small exposed populations, it is common practice to pool the data from individual studies together for combined analysis. When this approach, called meta-analysis, was used, a statistically significant 40 percent increase in brain cancer risk was found among workers exposed to VCM. This evidence is enough to warrant serious concern that vinyl chloride has caused an increase in the incidence of brain cancer among workers exposed in the vinyl production industry.

Environmental releases. The VI admits that EDC and VCM are released from EDC/VCM production facilities, but it asserts that the estimates in our submission are “outdated and grossly overstate EDC and VCM emissions” (VI 5). The estimates in our submission that world PVC production results in the release of hundreds of thousands of tons of EDC and VCM into the air and water each year were extrapolated from 1993 estimates by the Norwegian government, based on a detailed mass-balance and

monitoring program at a modern EDC/VCM facility in that nation. These data were collected from a nation with a well-regulated chemical industry, after the major reductions in occupational VCM exposure were achieved.

In its enclosures, the VI cites industry's self-reported emissions estimates from the U.S. Toxics Release Inventory, which suggest that 887,000 and 798,000 pounds of VCM and EDC, respectively, were released directly into the environment by U.S. manufacturers in 1998, plus an additional 2 million and 27 million pounds that were sent to sewage treatment plants and other off-site facilities.² These figures are substantially less (by a factor of 10 to 50) than the corresponding figures from Norway would predict based on the volume of U.S. vinyl production. But there are widely recognized problems with the TRI database: reported emissions are not based on actual monitoring of releases, and estimation methods may vary greatly among facilities. Most importantly, TRI release estimates are self-reported by the industry and are subject to no independent verification.

For these reasons, the carefully prepared Norwegian estimates, based on actual measurements of material flows and emissions at the plant, are far more reliable than the self-reported figures without a verifiable empirical basis that were submitted by the U.S. vinyl industry itself. It is possible, however, that the Norwegian figures may not reflect the most recent reductions in emissions. The VI's enclosure suggests that total releases of VCM and EDC by U.S. manufacturers declined by about 50 percent between 1993 and 1998; if a similar adjustment is applied to the Norwegian figures, then world vinyl production will result in releases on the order of 50,000 tons (110 million pounds) each of VCM and EDC to the air and water each year

Whether the true numbers are closer to those predicted by the Vinyl Institute or the Norwegian government, these are very large quantities – millions of pounds per year worldwide of extremely toxic substances that have no known threshold -- that may compromise health and environmental integrity in the region of their release.

Health impacts in communities near EDC/VCM facilities. The VI does not dispute specific findings of severe EDC, VCM, hexachlorobutadiene, and dioxin contamination and exposure in communities and environments near facilities that manufacture vinyl feedstocks (Thornton 30-31, 33-35) .

The VI does argue that no epidemiological studies have established a causal link between living near an EDC/VCM production facilities and cancer and other impacts (p. 5). This is a familiar situation, because conclusive epidemiological data are frequently lacking on health risks in communities near industrial facilities. As the National Research Council has pointed out³, epidemiological studies of communities exposed to pollutant releases from industrial facilities are extremely limited in their ability to detect subtle health impacts and link them to exposure, for several reasons:

- Study populations are usually small, making it difficult or impossible to establish statistically significant links between exposure and disease.
- Characterizing exposure is very difficult, because people are exposed to mixtures of chemicals that have a long latency period between exposure and effect. Exposures that occurred early in life are particularly difficult to reconstruct.
- Populations may manifest a variety of different symptoms due to chemical exposure, many of which vary naturally in the human population, making the establishment of specific causal links very difficult.

These considerations justify caution when interpreting the results of negative epidemiological studies: a failure to find a causal link does not provide strong evidence that a link does not exist. Because of the limits of epidemiological methods in this setting, the National Research Council recommended that a provisional link can be inferred between pollutant exposure and health risks when three criteria are met: 1) communities are exposed to identified substances; 2) those substances are known to cause health impacts in laboratory animals; and 3) those same health impacts occur within the exposed community. By these standards, VCM and EDC are clearly hazards to community health. If VCM and EDC are as safe as the VI asserts, one must wonder why several communities in Louisiana have been permanently relocated due to vinyl chloride contamination and exposure, a point the VI does not contest (Thornton 33-35).

4. *Formation of hazardous by-products in VCM/EDC production*

The VI does not dispute the body of evidence that extremely large quantities of highly hazardous wastes are produced in the synthesis of EDC and VCM (Thornton 31-32). Nor does the VI refute the evidence that these wastes contain considerable quantities of persistent, bioaccumulative, and toxic substances, including PCBs, hexachlorobenzene, hexachlorobutadiene, hexachloroethane, and octachlorostyrene (Thornton 31-35). The VI also does not refute the evidence that severe contamination by these by-products has been documented in the vicinity of several EDC/VCM production facilities in the U.S. and Europe.

5. **Dioxin and the vinyl lifecycle: industry estimates of dioxin releases**

As pointed out in our original submission, numerous analyses of wastes, emissions and residues from a variety of processes indicate that dioxin is formed in significant quantities at numerous points throughout the vinyl lifecycle (Thornton 25-26, 32-34, 36-38, 40, 45-46, 49-61). The VI does not dispute this evidence directly. It does, however, make two arguments in defense of the assertion that vinyl is not a major source of dioxin. In the first, the VI cites the vinyl industry's "dioxin self-characterization" study⁴ to argue that the U.S. PVC industry releases to the environment about 13 grams of dioxin (TEQ) per year (VI 5). This estimate is also the basis for the contention that vinyl production is responsible for only a small fraction of identified dioxin releases in the United States (VI 5).

There are three reasons to be very skeptical of the industry's estimate of its dioxin emissions:

- If the industry's estimate of 13 grams of dioxin per year were accurate, it would still justify action to reduce vinyl consumption immediately. U.S. EPA's current "acceptable intake" for dioxin for an average adult (weighing 70 kg) is 0.153 *billionths* of a gram per year.⁵ Based on its own estimates, then, the vinyl industry's annual releases of dioxin into the environment are equal to the acceptable annual dose for about 85 billion people.² The considerable toxicological significance of dioxin releases that sound inconsequential on a mass basis underscores the inadequacy of current methods for quantitative life cycle assessment, for which there are no weightings or consensus standards to address the relative hazards of specific substances.
- VI's estimates omit the majority of the dioxin produced during the vinyl lifecycle. The industry's "self characterization" analyzed several potential pathways for dioxin release, finding low to moderate quantities of dioxins and furans in samples of EDC, PVC products, air emissions, and wastewater and the sludge from its treatment. But numerous pathways that contain the largest amounts of dioxin, along with many PVC-related processes that are major dioxin sources, were completely ignored. No data, for example, were gathered on dioxin contamination of chemical streams that recirculate in the manufacturing process, of light ends and other wastes used in other synthesis processes, and -- most importantly, because these are known to be so severely contaminated -- heavy ends, tars, and other hazardous wastes that are sent to disposal facilities. Nor did the program address what is apparently the largest PVC-related dioxin source -- the burning of vinyl in incinerators, smelters, and accidental fires. Thus, the industry's estimates are grossly incomplete and do not effectively refute the argument that the lifecycle of PVC is a major dioxin source.
- VI's estimates have not been independently verified. In the "self-characterization," the PVC industry decided where and when samples were taken from its plants, collected them, analyzed their dioxin content, chose which data to present, interpreted them, and then submitted the results to EPA. The submission was reviewed by an independent panel, but the industry chose which data the panel saw. Information about the samples -- including the facility they come from -- was completely confidential, so neither reviewers nor the public had the opportunity to determine whether sampling times and locations accurately represented typical dioxin releases. Most importantly, no one was able to independently evaluate, confirm, or act on the information. It would be naïve to take at face value the industry's own estimates of the magnitude of its releases of dioxin, a substance that is the subject of major public concern and regulatory activity, particularly when those estimates conflict with a

² This is not to say that all of the dioxins released by the industry will result in direct human exposures. The point of this calculation is to demonstrate that, because dioxin is so exquisitely toxic, a quantity of dioxin that appears small on a mass basis is in fact extremely significant from a toxicological perspective.

large body of information gathered by the independent sources, as cited in our submission.

The VI does not refute the specific body of evidence indicating that dioxin is formed during the production of chlorine, the synthesis of EDC, the incineration of vinyl products, or the accidental combustion of vinyl building materials. Nor does the VI refute the findings from the Netherlands, Italy, Finland, and the United States of significant dioxin contamination in sediments in the vicinity of EDC/VCM manufacturing facilities (Thornton 33-34). The VI does not dispute the 1999 findings of the Centers for Disease Control that residents near an EDC/VCM plant in Louisiana have significantly elevated levels of dioxins in their blood (Thornton 34). These results indicate significant formation and release of dioxin from these facilities.

The VI does not dispute the conclusion of EPA and other scientists that levels of dioxin now in the environment, despite reductions in recent decades, are high enough to pose potential health risks to the general public, particularly children (Thornton 66-68).⁶ With that fact in mind, any product that makes a substantial contribution to dioxin pollution cannot be considered a green building material. As the only plastic that is an organochlorine, vinyl is unique in the class of building materials in its contribution to dioxin pollution.

6. *Dioxin trends*

The VI devotes almost one-third of its comments to an argument that dioxin levels have fallen considerably in some environmental media since the 1970s (VI 5-10). Despite the VI's contention that our original submission selectively excludes this evidence, we made the same point in our submission (Thornton 68-70).

The question is, what is the importance of this trend? The VI argues that production of PVC has increased since the 1970s, and that declining dioxin levels are incompatible with the conclusion that the lifecycle of PVC is a major source of dioxin. This is a simplistic argument, which fails to take account of the fact that PVC is not the only source of dioxin. There are now and have always been many categories of dioxin sources, and the contribution of each one can change over time. Trends in total dioxin levels in the environment cannot be expected to solely track trends in releases by any individual source. If one or more major sources are restricted, then total dioxin emissions may fall even if contributions from another important source are increasing.

As explained in our submission, the scientific community generally agrees that reduced deposition rates of dioxin from the air into sediment, vegetation, and foods since the 1970s are due to restrictions that were imposed around that time on other dioxin-producing materials, including dioxin-contaminated pesticides (i.e., chlorophenoxy herbicides and pentachlorophenol), the phase-out of leaded gasoline with ethylene dichloride additives, and new requirements for air pollution control devices on incinerators and steel mills (Thornton 68-70). In the 1990s, further reductions have been

achieved, as many outdated apartment and municipal trash incinerators and medical waste incinerators have also been shut down or subject to stricter controls.⁷ Drastic reductions in dioxin emissions from these sources would be expected to mask any increased contribution associated with the gradual growth in PVC production. Falling dioxin levels since the 1970s therefore do not refute the view, which is supported by specific analytical studies of dioxin generation at various points in the vinyl lifecycle, that producing and disposing of vinyl is now a major source of dioxin.

The VI also asserts that our submission dismisses evidence of dioxin formation before the advent of the chlorine industry (VI 5-6). But in fact we cited numerous analytical studies that found trace levels of dioxin in the environment before the 20th Century, along with others that found none (Thornton 58-61). What the VI does not acknowledge is that in every one of the studies in which dioxin was found in sediment layers before 1900, the levels were a tiny fraction – no more than a few percent – of current levels. The same pattern is found when dioxin levels in the tissues of ancient humans are compared to those from people from the current time (Thornton 58). The data clearly support the view that dioxin contamination is a thoroughly modern problem, associated with some change unique to the 20th century, which accelerated rapidly after World War II. As shown in our original submission, this temporal profile matches well that of the production and disposal of chlorinated organic substances.

7. Dioxins and open burning

The VI complains that our submission does not “even entertain the possibility that the rise is not due to the chlorine industry at all but is due to the extensive use of open burning for waste disposal” (VI 6-7). This is a novel suggestion, and the VI offers no data or references in support of it. The question is, what are the materials that cause (and have caused) open burning to generate dioxins? Open burning of domestic waste, including vinyl, may now be an important source of dioxins, but there is absolutely no information to suggest that trends in open burning are responsible for the rise in dioxin emissions in the 20th Century. The VI presents no data on trends in the amount of waste burned openly or in dioxin loadings from this practice. There is certainly no evidence to suggest that dioxin trends are due to changes in open waste burning, irrespective of the content of the wastes burned.

Several considerations suggest that the VI’s unsupported assertion is unlikely to be true. First, as we pointed out in our submission, studies of open barrel burning have found that burning trash from which PVC has been removed produces considerably less dioxin than burning wastes in which vinyl is present (Thornton 51). Second, dioxin levels in developing countries, where open burning is widespread but vinyl and other organochlorines have only been introduced recently, are considerably lower than in industrialized nations.⁸ This finding is inconsistent with the VI’s contention that open burning of wastes that do not contain vinyl or other organochlorines are a major source of dioxin, responsible for broad trends in dioxin loadings.

8. Dioxin and vinyl incineration: the “ASME” report

In our submission, we cited 21 studies from laboratory, pilot, and commercial-scale incinerators that show overwhelmingly that burning PVC and other organochlorines produces dioxin, burning more PVC and other organochlorines increases dioxin generation, and burning less reduces dioxin formation (Thornton 52-58). In its rebuttal, the VI cites a single study, purportedly by the “American Society of Mechanical Engineers (ASME), a professional society representing 125,000 mechanical engineers worldwide, [which] found little or no correlation between chlorine input and dioxin emissions from incinerators” (VI 11). This study is deeply flawed for several reasons, and does not provide an adequate basis to dismiss the many studies that do establish a link between dioxin generation and the combustion of PVC and other organochlorines.

The ASME study is biased. Several vinyl industry documents shed light on the purpose and origins of the ASME report. Just before U.S. EPA released its draft Dioxin Reassessment in 1994, the Vinyl Institute commissioned the public relations firm Nichols-Desenhall Communications to prepare a "Crisis Management Plan for the Dioxin Reassessment." According to the plan, "EPA will likely conclude that the incineration of chlorinated compounds is the single largest known contributor of dioxin.... We believe that PVC will be specifically mentioned and potentially slated for further regulation." In order to "prevent punitive regulation of PVC by EPA, Congress, or the state legislatures," the plan advised the Vinyl Institute how to present itself in the media and "strongly urge[d] VI to aggressively defend the industry's credibility through the use of third party sources to debunk ... EPA's misleading claims."⁹

The industry took its PR firm's advice. In 1994, the Vinyl Institute's Incineration Task Force hired the consulting firm Rigo and Rigo, Inc., to prepare an "independent" analysis, which found that the amount of dioxin released by incinerators has no relation to the amount of chlorinated organic materials fed to them.¹⁰ The VI arranged to have the report published as a product of the prestigious American Society of Mechanical Engineers (ASME), an independent professional organization. According to Vinyl Institute documents, one of the leaders of the Vinyl Institute's Incineration Task Force, Dick Magee, was also an active ASME member; Magee brokered an arrangement in which the Vinyl Institute would hire and fund Rigo to write a report that would be released under the ASME banner. According to an internal VI memo from 1994, the purpose of ASME's involvement was to create the illusion of "third-party" authority, and that the Rigo report was conceived, carried out, and rewarded in a spirit of public relations, not unbiased analysis. The memo reads:

“The Vinyl Institute has created an Incineration Task Force in anticipation of adverse EPA actions regarding dioxins and furans.... Dick Magee brought forward a proposal from the American Society of Mechanical Engineers to hire Rigo & Rigo Associates, Inc., of Cleveland, OH. The purpose of ASME as the contractor is to provide unassailable objectivity to the study....

The Incineration Task Group interviewed Dr. H. Gregory (Greg) Rigo, principal of Rigo & Rigo Associates, Inc. by phone and found him to be

extremely knowledgeable about incineration and to have several proprietary databases VI had not discovered. He is also user friendly, i.e., willing to set his priorities to our needs, and appears to be sympathetic to Plastics, Vinyl, PVC and Cl2....

The ASME proposal calls for \$130,000 for the Rigo & Rigo/ASME study. Since there are many unanswered questions regarding dioxins and since VI may want to use Greg Rigo as an expert witness or advocate to talk about the report, I am proposing an additional \$20,000 as a contingency fund, for a total of \$150,000 to be fully funded by VI.”¹¹

Methodological flaws. The Rigo report’s methods are no less flawed than its origins, undermining the reliability of its claim that burning organochlorines is not related to dioxin formation. The study was not experimental, so it could not directly refute the existence of a chlorine-dioxin link. Instead of generating new data, the authors compiled existing trial burn measurements from a large number of incinerators, statistically analyzed the relationship between indicators of chlorine feed and dioxin releases, and concluded that there is no statistically significant relationship between the two. A statistical analysis of this type -- an epidemiological study on machines, in a sense -- is particularly sensitive to design problems: if the putative cause and effect are not measured very accurately, or if confounding factors are not taken into account, then a meaningful relationship is likely to go undetected. The study’s methods were deeply flawed on several counts, suggesting that its failure to find a link between burning organochlorines and the generation of dioxin is an artifact of bad study design, not a meaningful finding that no relationship exists in the real world.

- The first problem is the study’s failure to take account of differences among facilities. Chlorine input is not the only factor that determines the magnitude of dioxin emissions from incinerators: combustion conditions, the types and quantities of substances in the waste, and other variables also affect the amount of dioxin that will be released. Because of this complexity and constant fluctuations in many of these factors, statistical relationships between stack emissions and indicators of waste input or combustion conditions can seldom be established, even at individual incinerators.¹² Massively compounding this problem, Rigo used data from a large number of incinerators operating under widely variable conditions, but he did not control or adjust for differences in facility type, waste type, operating parameters, or any other factors. There is no reason to expect that a statistical summary of data from many different facilities, with no attempt to control or adjust for confounding factors, would have detected a “signal” within all this “noise.” Even a very strong relationship between organochlorine input and dioxin output is likely to go undetected in a study designed this way.
- Rigo's study was also undermined by its use of data from unreliable sources. The emissions data in Rigo’s analysis came almost exclusively from trial burns designed for permitting purposes, without the proper kinds of controls and measurements necessary to evaluate the relationship between chlorine input and output. Moreover,

trial burn data are notoriously problematic, because they do not accurately represent the way incinerators operate in the real world, and they do not measure the much larger quantity of chemicals that are released after the feed of waste to the incinerator has stopped.¹³ In fact, many trial burns have conducted their evaluations of low- or no-chlorine wastes *after* chlorinated wastes have been burned, so the later stack samples become contaminated by continuing emissions from earlier runs. The use of results from trial burns of this sort will thoroughly scramble any relationship that might otherwise have been recognizable between chlorine input and dioxin input.

- The final flaw of Rigo’s study is its reliance on the wrong kinds of measurements. To investigate a link between the amount of organochlorines burned and the amount of dioxin produced by incinerators, Rigo should have examined the statistical relationship between the mass of organochlorines fed to an incinerator and the mass of dioxins released. Instead, the study tracked “surrogate“ measures for both of these parameters, tracking the concentration of hydrogen chloride (HCl, the primary by-product of organochlorine combustion) as a rough indicator for the amount of organochlorines in the feed; as a surrogate for the amount of dioxin released, it examined the concentrations of dioxin in the stack gas.¹⁴ The problem is that concentrations do not accurately represent quantities, for several reasons:
 - If the total flow of stack gas increases (as it generally will if more waste, and thus more chlorine, is fed to the incinerator), the concentrations of dioxin in the gas may decrease even if a larger amount of dioxin is being emitted.
 - Stack gas measurements omit pollutants directed into fly ash, bottom ash, and scrubber water, so changes in the efficiency of pollution control devices can reduce the concentration of dioxin in stack emissions while total dioxin formation increases, or they can reduce the concentration of HCl while total organochlorine input rises. (Because pollution control devices have different capture efficiencies for dioxin and hydrochloric acid, the concentrations of these materials in the stack gas after it passes through this equipment will not reflect the ratios of the amounts that were actually produced by the incinerator).
 - Hydrogen chloride can be formed not only by the combustion of organochlorines but also by the burning of chloride salts, further undermining the reliability of HCl as an indicator of organochlorine feed.

The variables that Rigo analyzed are thus grossly inappropriate substitutes for the quantities that are truly of interest; Rigo’s failure to find a relationship between the surrogates he used says nothing about whether a link really exists between organochlorine input and dioxin generation.

All the flaws discussed above cripple the “ASME” study’s ability to establish a link between chlorine and dioxin. A finding of “no relationship” is only as good as a study’s power to detect a relationship, and in this case that power can only be described as pathetically weak. On the basis of Rigo’s analysis, no reliable inferences can be drawn about whether a relationship exists between the amount of organochlorines burned and the amount of dioxin formed in an incinerator. With more than twenty well-designed

studies coming to the opposite conclusion – that burning PVC and other organochlorines does produce dioxins, and burning less of these materials will reduce dioxins – Rigo’s findings are far from persuasive. The weight of evidence from laboratory, pilot, and full-scale tests clearly establishes that vinyl is an important source of dioxin in incinerators, fires, and combustion-based recycling facilities, which together are responsible for the majority of identified dioxin releases in the world.

9. Vinyl and recycling

The VI asserts that vinyl “can be and is being recycled” (VI 10). As pointed out in our submission, this statement is true to only a very limited extent. The presence of various types and quantities of additives in each vinyl product makes post-consumer recycling extremely difficult, and only very small quantities are now recycled; even best-case estimates suggest that very limited amounts will be recycled in the future (Thornton 47-48).

The VI asserts that the low postconsumer vinyl recycling rates presented in our submission are low because most vinyl enters long-life uses and is not available for recycling (VI 10). This argument is incorrect. The figures we cited from the European Union – current postconsumer vinyl recycling rates of less than 1 percent and future recycling of just 9 to 18 percent by 2020 – expressed the amount of vinyl recycled as a percentage of the total amount of vinyl recycled or disposed, not as a percentage of total vinyl demand or production.¹⁵ The VI does not dispute the finding of the American Association of Postconsumer Plastics Recyclers that vinyl is more difficult to recycle and enjoys lower recycling rates than any other major plastic (Thornton 47).

The VI cites anecdotal information about the number of companies handling scrap vinyl (VI 10). This information in no way changes the fact that the vast majority of postconsumer vinyl is not recycled and cannot be expected to be in the near future. Virtually all vinyl in the waste stream – including the huge amount that will soon begin to present a disposal burden as long-life vinyl uses in construction begin to turn over – will be disposed of by landfill or incineration, with considerable environmental costs. No material that enjoys such low recycling rates can be considered sustainable or green.

10. Metal stabilizers

The VI attempts to minimize concern about the use of heavy metal stabilizers in vinyl by arguing that vinyl is responsible for only a small fraction of total consumption and release of these toxic metals (VI 12). The VI cites the European Union’s “Green Paper” on PVC to support its contentions. The Green Paper notes that vinyl contributes from 1 to 28 percent of the lead and 10 percent of the cadmium entering municipal waste incinerators. Because these are highly toxic, persistent, and global pollutants, and municipal waste incinerators are among the major sources of these compounds, these contributions cannot be considered negligible.

More important than the fraction of a metal that is used in vinyl is the *amount* of these substances used in PVC. After all, it is quantities, not percentages, that are responsible for the environmental hazards that persistent toxic metals pose. Consider lead, which the VI claims is not a major vinyl stabilizer (13). In Europe, lead compounds account for 70 percent of all stabilizers used in vinyl; about 51,000 metric tons of lead (over 112 million pounds) are used in vinyl stabilizers each year.¹⁶ Because lead is an infinitely persistent substance and is exquisitely toxic to the developing brain, this is a highly significant amount. The VI does not dispute that lead in vinyl is released into the environment when vinyl products burn or are disposed of, and they can be released into the indoor environment, as well (Thornton 44, 46, 51). In November 2000, the Danish government took action to ban the use of virtually all lead compounds, including those in PVC windows, cables, gutters, roofing, and pipes, by no later than 2003.¹⁷

The Green Paper also notes that 50 tons (110,000 pounds) of cadmium – also a highly neurotoxic and infinitely persistent metal -- are used in vinyl each year in Europe. This is also a significant quantity of a highly persistent and neurotoxic substance.

Consumption of organotin compounds in vinyl is estimated at 15,000 tons. The VI points out, correctly, that the organotins used as vinyl stabilizers are not known to have the endocrine disrupting activity of tributyltin, a well-studied member of the class that is not used in vinyl (VI 13). But organotins used in vinyl are potent immunotoxicants, cause birth defects, damage the liver, bile duct and pancreas, and may pose hazards to the aquatic organisms when released into the environment.¹⁸

11. Vinyl, chlorine production, and mercury

The VI asserts that only 20 percent of the chlorine produced in the U.S. by the mercury process is used to produce vinyl, an estimate for which no citation or data are provided (VI 12). If true, this estimate does not eliminate concern about mercury pollution. In its enclosures to the USGBC, the VI includes industry estimates that the U.S. chlor-alkali industry “consumed” 176,769 pounds of mercury.¹⁹ Mercury is not “consumed” in the chlor-alkali process; as we referenced in our submission, “consumption” consists of losses of mercury to the environment through products, effluents, emissions, and wastes (Thornton 27). Based on the Chlorine Institute’s estimate of mercury consumption and the VI’s 20 percent figure, vinyl manufacture is associated with the release of over 35,000 pounds of mercury per year. Because mercury is an extremely persistent, bioaccumulative, and toxic global pollutant, the industry’s own estimates suggest that mercury pollution associated with vinyl manufacture is very significant from an environmental health standpoint. The VI does not dispute the evidence of reports of continuing and severe mercury contamination near chlor-alkali plants in locations around the world (Thornton 27-28).

12. Vinyl and mold growth

The VI grants the fact that vinyl wallcoverings can trap moisture inside wall cavities, leading to the growth of fungi and bacteria, which can produce severe symptoms in exposed persons (Thornton 43-44, VI 14). The VI does argue, however, that some vinyl manufacturers have taken steps to produce mildew-resistant and vented wallcoverings. No data are presented on the effectiveness of these measures, and it is likely that most vinyl products on the market today do not have these features. As we pointed out in our submission, building industry sources recommend that the best way to prevent mold growth is to avoid PVC wallcoverings.

13. Phthalate plasticizers

The VI's comments on the hazards of phthalate plasticizers (VI 13-14) refer solely to comments on a portion of our submission from an industry organization called the "Phthalate Esters Panel." Although this body sounds as if it were an assembled panel of experts, the members of the panel are not scientists but are exclusively chemical companies that produce or process phthalates. The panel is a part of the American Chemistry Council (ACC), formerly the Chemical Manufacturers Association. Its comments were written by Chemstar, the ACC's in-house division for "chemical industry self-funded technical advocacy and research." With its membership and affiliation in mind, Chemstar's pretense to provide a "fair and objective" reading of the evidence is ironic (Chemstar 1).

Some of Chemstar's comments call attention to uncertainty and gaps in the toxicological data on phthalates. Others are misleading in a number of ways and do not undermine the concern established by independent scientific research, particularly in the recent set of reviews of phthalate exposure and toxicity conducted by an expert panel convened by the U.S. National Toxicology Program (NTP). Although some of the data concerning the impacts of phthalates on public health have not yet reached the standard of strict scientific proof, waiting for conclusive proof is not an appropriate standard for environmental policy or decisions about building materials. With limited but high-quality scientific evidence at hand, we have a sound basis for concern about the toxicity of phthalates in humans and animals. This existing body of evidence provides a fully adequate justification for choosing to avoid PVC building materials in order to safeguard health and the environment.

Environmental releases. Chemstar asserts that releases of phthalates to the environment are low, citing industry's self-reported data on releases in New York State from the Toxics Release Inventory (TRI) (Chemstar 3). It is not clear why Chemstar restricted the releases reported to a single state; the national figures are 42 to 632 times greater than those reported by Chemstar. According to the TRI database, U.S. industries released approximately 660,000 pounds of just three phthalates (DEHP, DBP, and dimethyl phthalate) directly into the environment in 1998, plus an additional 3.4 million pounds that were transferred to off-site facilities for disposal.²⁰ These are very substantial releases, and they do not include phthalates released from vinyl products

during use or disposal or the numerous other high-volume phthalates that are not subject to TRI reporting requirements.

Persistence and bioaccumulation. Chemstar argues that phthalates are not predicted to be persistent or bioaccumulative (Chemstar 3-6). The enclosed comments of Dr. Ted Schettler address these points specifically, pointing out that under some conditions phthalates are very persistent; further, some phthalates have been observed to bioaccumulate to a considerable extent. These results are not surprising, given the fat solubility of most phthalates.

The most convincing data on phthalate persistence and bioaccumulation is their actual occurrence in the environment. Despite Chemstar's predictions, it is clear that phthalates in the real environment are both persistent and bioaccumulative, as demonstrated by the fact that they have become ubiquitous contaminants of sediments, food, and human tissues, even in areas remote from sources of these compounds. These data were presented in our original submission and were not disputed by the VI or Chemstar (Thornton 71). Further, as the NTP has pointed out, the food supply is the primary route of exposure for most phthalates, indicating the tendency of these compounds to bioaccumulate (Thornton 71). *Why* these substances have accumulated ubiquitously, despite predictions based on their physicochemical properties that they will not do so, remains unclear. The fact that that they have done so, however, is beyond argument.

Phthalates in indoor air. Chemstar argues, based on the physical and chemical characteristics of phthalates, that plasticizers are not expected to enter indoor air from vinyl products in significant quantities (Chemstar 9-10). But these predictions are refuted by actual measurements of elevated levels of phthalates in indoor air where flexible PVC is present. As we pointed out in our submission, indoor levels of phthalates in buildings with abundant PVC are typically 5 to 300 times greater than outdoor levels, a finding not disputed by Chemstar (Thornton 41).

In asserting that shower curtains and other new flexible vinyl products are not predicted to release phthalates at levels that can be detected by smell, Chemstar provides no empirical data (Chemstar 10). Again, Chemstar's prediction is refuted by the common experience of the detectable odor of plasticizers from such products.

Chemstar asserts that the high levels of DEHP and other plasticizers found in indoor dust have probably not been released from vinyl products but are part of abraded PVC particles (12-13). No data are presented to support this assertion. In fact, the authors of the study referenced in our submission²¹ specifically discussed and excluded this possibility, noting that substantial quantities of phthalates were present not only in sediment dusts (where PVC particles are expected to reside) but also in suspended particulate matter. Further, they were present in both the inorganic and organic fractions of the dusts, whereas PVC particles are organic matter. Chemstar also argued that phthalates in dust are likely to be attached to particles that are not respirable. The authors of the study addressed this issue by separately measuring phthalates in suspended

particles (which are predominantly small and respirable) and those that had sedimented onto surfaces. Of the sedimented dust, the organic fraction was separated from the inorganic fraction (where large particles of sand and soil are found), and phthalate levels were found to be approximately equally high in the two fractions, indicating that phthalates were present on particles of small size, as well larger ones.

Assurances of safety based on reference doses. Most of Chemstar's arguments about the safety of phthalates rely upon comparisons of human exposure levels to the EPA's reference dose (RfD) for oral exposure to specific phthalates. An RfD is the "acceptable" exposure level which EPA predicts, based on toxicological studies, will not produce significant risk of health effects. Chemstar argues that there is no reason for concern about exposure to DEHP and other phthalates in food (Chemstar 8), in indoor air (10-11), on dust (12-13), and in blood samples from the general population (14), because the levels are below EPA's RfDs.

But EPA's RfDs are out of date and inappropriate for this purpose. EPA's RfD for DEHP, for instance, on which Chemstar relies most heavily, was established by EPA in 1986 based on a 1953 study that examined changes in liver weight in rodents exposed to DEHP, supplemented by other studies from 1982 and 1984. This RfD was calculated before any of the relevant studies of the effects of chronic low-level exposure to DEHP and metabolites on reproduction and development were published. Similarly, the RfD for BBP was based on a single 1985 study; the RfD for diethyl phthalate was based on a 1978 study; and the RfD for DBP was based on the same 1953 study as that for DEHP.²²

EPA's RfDs are therefore outdated and do not provide a useful standard on which to judge the safety or hazard of exposure to phthalates. As detailed in the attached comments of Dr. Ted Schettler, current information would support an RfD for DEHP that is very close to the daily intake of an average child in the United States. It was precisely this comparison, in fact, that led the National Toxicology Program to express "concern" about impacts on child development due to "ambient oral exposures to DEHP."

It is also not appropriate to use EPA's RfD for oral exposure to judge the hazards of inhalation exposure, as Chemstar does (10-13). A basic toxicological principle is that the potency and effects of any substance may vary when exposure occurs through different routes. Recent studies on inhalation exposure to phthalates cited in our submission establish hazards that are unique to inhalation exposures, including the observed accumulation of phthalate metabolites in the airway, the activation of biochemical cascades associated with asthma, and the increased prevalence of asthma and various signs of bronchial irritation associated with exposure to vinyl in the indoor environment (Thornton 42-43). An oral RfD is therefore irrelevant and inappropriate for predicting safe or hazardous levels of inhaled phthalates.

Assurances of safety based on the "environmental scorecard." Chemstar also dismisses concern about environmental releases of phthalates by referring to Environmental Defense's environmental scorecard, which found that ambient levels of phthalates in the air did not exceed EPA's hazard index based on inhalation exposures

(Chemstar 2). But the major concern about phthalate releases into the outdoor environment is their ultimate migration into food, which is the primary exposure route for the human population for most phthalates. The scorecard is not intended to reflect this process or route of exposure and therefore provides no relevant reassurance that environmental releases of phthalates are safe.

Reproductive effects at low doses. Chemstar states that effects on reproduction and development have been observed only at phthalate doses that far exceed those anticipated for human beings (Chemstar 26). This statement is incorrect. In cultured testicular cells, the DEHP metabolite MEHP suppress proliferation of developing sperm-producing cells at a concentration of just 10^{-7} molar, or 30 parts per billion. The internal dose of MEHP in the human population is not known precisely; however, the 95th percentile concentration of MEHP in the urine of the U.S. population is 21.5 parts per billion – about 30 percent lower than the level at which effects have been observed in testis cells.²³ Further, as Dr. Schettler details in his comments, current exposures of the average child in the United States are already in the same range as a reference dose calculated from recent findings of reproductive impacts at low DEHP doses. There is clearly a sound basis for concern that the public is already exposed to higher levels of some phthalates than we can feel confident are safe, and efforts to reduce phthalate use and exposure are well justified.

Blood levels of phthalates in the general population. Chemstar asserts that the 2000 CDC study of phthalates in the blood of the general population are not surprising and are well within the levels known to be safe (Chemstar 14). But this assertion is based again on outdated and irrelevant RfDs. Indeed, the authors of the CDC study concluded, “Some individual exposures are substantially higher than previously estimated for the general population,” and these “data indicate a substantial internal human dose of DBP, DEP, and BBP, [the metabolites of which] are of particular concern because of their developmental and reproductive toxicity in animals.”²⁴ The estimated daily DEHP exposure based on the 95th percentile and maximal levels found in the general population’s urine was estimated to be 3.6 and 46 ug/kg/day – in the same range as the updated RfD calculated by Dr. Schettler.²⁵

Evidence of health effects in humans associated with phthalate exposure. Chemstar dismisses several recent epidemiological studies linking phthalate exposure to respiratory health effects, along with a study linking premature breast development in girls to elevated phthalate levels in blood, as “fall[ing] far short of demonstrating a causal link” (Chemstar 27-30). This statement reveals a misunderstanding of the appropriate role of epidemiological data in policy decisions about environmental health and material use. The data are preliminary but sound: they do not close the case scientifically but establish adequate justification for concern that phthalates may contribute to health problems in the general population and action to reduce exposure to these substances.

Demonstrating a causal link epidemiologically is an extremely demanding exercise, which, as the history of science on cigarette smoking and cancer illustrates, typically requires decades of work by hundreds of scientists. A strict causal link is

proven only when all alternative explanations are ruled out and a specific relationship established between a single substance or source and a single effect. Epidemiological studies of toxic chemicals, however, are limited by the fact that people are exposed to complex and changing mixtures of pollutants, most of which have not been identified; by the difficulty of measuring exposures precisely for most chemicals; by the lack of unexposed comparison population; by the complex suites of effects that pollutants may produce; by the long latency periods between exposure and many health effects; and by the variability in susceptibility among individuals.

Epidemiology is by nature a form of body-counting; causal linkages can be proven only after large numbers of people have suffered health damage. Waiting for conclusive proof is not an appropriate standard for environmental policy or decisions about building materials. With limited but high-quality scientific evidence at hand, we have a sound basis for concern about the toxicity of phthalates in humans and animals. This existing body of evidence provides a fully adequate justification for choosing to avoid PVC building materials in order to safeguard health and the environment.

Neither Chemstar nor the VI disputes the suggestive evidence from Swedish studies of plastics workers, which found significantly elevated risks of testicular cancer that were related specifically to occupational exposure to PVC (Thornton 39).

Toxicological mechanisms. Chemstar provides a lengthy discussion of mechanisms of toxicity (Chemstar 15-19, 22-26) that is not particularly relevant to the debate over vinyl building materials. Continuing scientific uncertainty over precisely *how* phthalates impair reproduction and development does not undermine the consistent finding that these substances do produce testicular damage, particularly when exposure occurs during development.

Numerous studies have sought to identify the mechanisms by which phthalates and their metabolites cause the reproductive and developmental defects that have been established in the laboratory (see, for example, the discussions in the reports of the NTP expert panel on phthalates). Others have identified novel interactions with receptor proteins that raise the possibility of additional forms of biological disruption, most of which have not yet been investigated at the organismal level (Thornton 71-72). A full mechanistic explanation has yet to emerge of the etiology of the organismal effects that have been observed in laboratory animals.

Although knowledge of mechanisms is not required for action to reduce phthalate exposure and release, it is worth considering Chemstar's objection to the discussion in our submission in light of the strengths and weaknesses of the kinds of toxicological tests being discussed. Endocrine testing of chemicals takes place at several levels, including in vitro tests (test tube experiments with purified biological molecules to clarify mechanisms) and in vivo investigations, in which the whole animal is exposed to the substance and specific effects that are predicted to occur after exposure to a hormone-disrupting chemicals are measured. Chemstar points out that phthalates that have been found to bind to estrogen and androgen receptors in vitro have not been shown to cause

the hormone-like effects anticipated in vivo. This statement is correct, but the assertion that in vitro tests are invalid without in vivo confirmation is overly conservative. In vitro tests have established that certain phthalates do interact with numerous classes of hormone receptors and therefore have the potential to cause a variety of reproductive, developmental, and other effects. Just as in vitro tests do not always predict the effects that will be observed in the whole animal, in vivo tests are of limited scope and sensitivity and are therefore prone to false negative results.

- Sensitivity to endocrine effects varies wildly among species, at different life stages, in various tissues, and at different doses.²⁶ In vivo tests typically use a single species, do not involve the most sensitive periods of development, and investigate a small number of possible effects.
- Very low doses of a hormone-like substance can produce health effects that are not observed at the higher doses at which most in vivo tests are carried out; a negative in vivo result may occur because the doses were higher than the levels to which people and wildlife are exposed.²⁷
- Some synthetic chemicals – most notably the selective estrogen response modulators – induce changes in receptor structure that differ from those produced by the natural hormone. The result can be a pattern of physiological effects that differs from the anticipated “classic” effects observed with the natural substance. Because novel effects like these are not typically sought in an in vivo assay, they are unlikely to be observed, while the lack of classic impacts may be interpreted as evidence that no endocrine disruption has occurred.²⁸

For these reasons, the negative in vivo tests that Chemstar cites should not automatically dismiss the concern that is established in in vitro tests. It is clear that some phthalates interact with hormone receptors and therefore disrupt the endocrine system at the molecular level in a variety of ways; we also know that these substances are potent toxicants to the testis and disrupters of male reproductive tract development. There is no reason to dismiss these findings, simply because classic in vivo assays for estrogenic and androgenic impacts have not produced the effects anticipated.

Finally, Chemstar asserts that effects observed in rodents are not relevant to humans, because the peroxisome-proliferating effect of phthalates in rodents does not seem to occur in primates (Chemstar 15-19, 26). Chemstar’s argument is incorrect. Although this one effect is not similar across species, this finding does not imply that none of the impacts observed in rodents will occur in humans. The receptor that triggers peroxisome proliferation in the rat upon DEHP exposure, PPAR α is present in humans and regulates gene expression in both animals. As the National Toxicology Program expert panel has pointed out, the differences between species in peroxisome proliferation is apparently caused by the fact that PPAR α activates a different set of genes in humans from those it regulates in rats. The ability of some phthalates to interact with PPAR α is therefore biologically relevant to humans, although not all PPAR α -mediated effects that occur in rodents will also occur in humans. Most importantly, not all the effects of phthalates are mediated via PPAR α ; a mouse strain that lacks a functional PPAR α remains susceptible to phthalate-induced testicular toxicity, as is the guinea pig, a

species which, like humans, does not exhibit peroxisome proliferation after exposure to DEHP. The NTP therefore concluded that reproductive impacts of DEHP observed in rodents are therefore likely to be relevant across species.²⁹

14. Conclusion: Assessment of the evidence

There is much still to be learned about the effects that dioxin, phthalates, and other substances associated with the vinyl lifecycle have on human health and the environment. On some issues relevant to the environmental hazards of vinyl, there is little debate and the evidence is unequivocal. On other questions, the evidence is suggestive, but clearly adequate to justify preventative action to minimize exposures while additional research continues.

Issues that are beyond debate, which have not been rebutted by the vinyl industry in their comments, include the following:

- The feedstocks for vinyl – EDC and VCM -- are highly toxic and carcinogenic and are released into the environment and the workplace in large quantities, although there is disagreement about the precise amounts. There is no known safe dose for these substances.
- Very large quantities of persistent, bioaccumulative, toxic substances are produced as by-products when the feedstocks for vinyl are manufactured. These by-products include significant quantities of dioxins, PCBs, hexachlorobenzene, and other very hazardous organochlorines.
- Severe contamination with persistent organochlorines has been documented in the vicinity of facilities that produce the feedstocks for PVC in several nations, including the US.
- Large quantities of phthalates are released into the environment as a result of their production and use as vinyl plasticizers. Phthalates are known to cause reproductive toxicity in rodents and there is no reason to think that humans are not susceptible to these effects, particularly during development.
- Dioxin is extraordinarily toxic and is now found in the environment at levels that may pose health hazards to the general human population and wildlife.
- When vinyl burns in an accidental fire, the primary combustion product is hydrochloric acid, which can pose serious health hazards to building occupants and firefighters.
- Very large quantities of electrical energy are required to produce the chlorine that is used in vinyl. As the only major chlorine-containing building material, vinyl is unique in this way.
- Postconsumer vinyl wastes are difficult to recycle. Although some vinyl can be recycled and small amounts are recycled, even best-case scenarios estimate that only a very small fraction of the vinyl waste stream will be recycled in the coming decades.

- A significant number of governments, firms, and organizations around the world have already committed to reducing or eliminating the use of vinyl and substituting safer alternative materials, including in the building industry.

There are several other points with which the VI has taken issue, but the evidence in support of them is overwhelming:

- Phthalates are released from vinyl products into indoor air.
- The general population is already exposed to some phthalates at levels at or near a reference dose designed to prevent reproductive toxicity.
- Significant amounts of dioxins and other persistent, bioaccumulative, toxic substances are produced when vinyl is burned in incinerators, accidental fires, or other forms of combustion, such as open burning of wastes.

In addition to these unambiguous points, there are several additional issues for which there is suggestive evidence or a circumstantial basis for concern, but the available data does not allow firm conclusions:

- Phthalate exposure may be contributing to a number of health problems in humans, with such effects as premature sexual development in girls, testicular cancer in exposed workers, and asthma and other respiratory conditions as a consequence of indoor air exposure.
- Based on preliminary evidence of disruption at the molecular level, phthalates have the potential to cause a variety of effects on reproduction, development, and endocrine and metabolic homeostasis; many of these effects have not been investigated at the level of the organism.
- The lifecycle of vinyl may be associated with the production and release of more dioxin than any other single product. The exact quantity of dioxin produced worldwide in the vinyl lifecycle remains unknown.
- Exposure to EDC, VCM and the by-products of their manufacture may cause increased health risks in workers and community residents.

Vinyl is the only organochlorine used as a building material and the only plastic that requires phthalate and metal stabilizers. It therefore poses uniquely severe environmental hazards. Although some other building materials can cause real environmental impacts, vinyl stands out as egregiously hazardous. The judgment that vinyl is not a green building material has sound justification in both science and policy.

15. Notes and references

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SECTION IV

Comments on "Response of the Phthalate Esters Panel of the American Chemistry Council to a Briefing Paper Submitted by Joe Thornton PhD, Acting Under the Direction of the Center for Maximum Potential Building Systems and the Healthy Building Network" (Nov 20, 2000)

Ted Schettler MD, MPH

Science and Environmental Health Network

Nov 29, 2000

On November 20, 2000, the Phthalate Esters Panel (PEP) of the American Chemistry Council submitted to the US Green Building Council a response to a briefing paper that had been prepared by Joe Thornton, PhD. I was provided with a copy of the PEP response, and have the following comments on that document.

Environmental releases of phthalates:

The PEP says that Environmental Defense (ED) finds that not one person in the US is exposed to DEHP in ambient air at levels of concern. It is important to note that ED's hazard index, referred to by the PEP, is calculated for ambient outdoor air exposure only and does not consider indoor air. In calculating the hazard index, ED did not take into account more recent concerns about the relationship between indoor inhalation exposures to phthalates and the frequency of wheezing episodes (see below). In fact, most human exposure to DEHP is dietary, not by inhalation from ambient air. Food is contaminated with DEHP from multiple sources, including widespread environmental distribution of this high production volume chemical.

Phthalates and bioaccumulation:

DEHP tends to bioaccumulate in aquatic invertebrates much more readily than in terrestrial or aquatic vertebrates. Bioconcentration factors (BCFs) for DEHP range from 100-900 in various species of fish, according to a series of studies evaluated in a draft risk assessment of DEHP from KEMI (Sept., 1998). The European Council for Plasticizers disputes the higher figures and believes that a more realistic BCF would be about 100. BCFs for DEHP in aquatic invertebrates may be as high as 15,000 to 20,000 and are commonly several thousand.(same reference)

Phthalates and environmental persistence:

DEHP biodegrades in aerobic conditions in days to weeks, with the half-life estimates depending on the method of analysis used. However, in anaerobic conditions, biodegradation is markedly delayed. Some studies quoted in the KEMI draft risk assessment show no degradation of DEHP after months in anaerobic conditions.

Phthalates and EPA's reference dose (RfD):

The PEP notes that dietary exposures to phthalates are below levels of concern, using the EPA's oral reference doses (RfDs) as a conservative estimate of a safe daily dose. (pg. 8) EPA's RfD for DEHP is 20 microgm/kg/day. (EPA Integrated Risk Information System) A daily oral intake of 19 microgm DEHP/kg/day in childhood is essentially at this reference dose. That is, there is no margin of safety between the RfD and actual human exposures. Any individual consuming more than 19 microgm DEHP/kg/day will be exposed in excess of the RfD.

Moreover, it should also be noted that this oral RfD was established in 1986, based on chronic and sub-chronic oral exposure studies in adult rats and guinea pigs and fertility and reproductive effects using a continuous breeding protocol. No developmental studies were included in the data used to establish that RfD. Since then, however, studies in rodents and other species show that the developing organism is susceptible to some adverse impacts of DEHP at levels of exposure far lower than adults. In particular, testicular toxicity of DEHP in the developing organism appears to be a particularly critical health effect.

The PEP describes the comments of the National Toxicology Program's Center for Evaluation of Risks to Human Reproduction (NTP Panel), with respect to human dietary exposures to phthalates (pg. 8). However, the PEP fails to acknowledge that the NTP's expert panel noted that dietary DEHP exposures may exceed 20 microgms/kg/day and concluded that they did have concern about current population-wide dietary exposures to DEHP, precisely because of the unique susceptibility of developing organisms.(see below)

Phthalates in indoor air:

The PEP again uses the EPA's oral RfD's as a generally accepted safe exposure level (pg. 9-13). However, the reports by Jaakola, et al. and Oie, et al. raise entirely new questions about the role of inhaled phthalates in the pathogenesis of asthma or wheezing episodes. The potential toxicity of DEHP by inhalation exposure was not considered in the derivation of EPA's oral RfD.

The Jaakkola studies examined the relationship between PVC wall coverings (or flooring) and respiratory symptoms, including wheezing episodes. Oie, et al. measured phthalates adherent to indoor dust. In a cross-sectional study, Jaakkola, et al. found a statistically significant relationship between persistent wheezing episodes, cough, and

phlegm and the presence of PVC wall coverings. Oie, et al. quantified the phthalate plasticizers present in household dust.

The fact that DEHP inhalation exposures from indoor air contamination are well below the EPA's oral RfD is of no relevance here. The derivation of that oral RfD is discussed above. Whether one believes the indoor inhalation exposures to phthalates to be "high" or "low", it is well known that low-level inhalation exposures to respiratory tract sensitizers may easily contribute to increases in incidence and severity of asthma attacks, particularly in sensitive individuals. The epidemiological data of Jaakkola are provocative and deserve attention. It is entirely plausible that airborne phthalates or phthalate-containing dust particles may trigger respiratory symptoms at exposure levels far lower than those required to produce toxicity through oral exposure. Though the Jaakkola studies have limits and are not by themselves definitive, they are entirely supportive of a link between phthalate inhalation exposures and asthma or other respiratory symptoms.

Phthalates and endocrine disruption:

The potential for phthalate esters to disrupt hormone function is incompletely understood, and data are incomplete. As noted in the PEP comments, DBP, BBP, and DHP show some capacity to bind to the estrogen receptor and initiate gene transcription in in vitro assays. When examined in vivo (Zacharewski, et al.), these same phthalate esters did not show reproducible estrogenic effects on uterine wet weight or vaginal cornification. Uterine wet weight and vaginal cornification are commonly used as endpoints to examine for estrogenic effects. The authors concluded that these phthalates were not estrogenic in vivo at the doses tested. The potential for these chemicals to alter other estrogen-related endpoints has yet to be examined.

DBP and DEHP have been shown to disrupt development of the male reproductive system, and since the effects appeared to be "anti-androgenic", investigators hypothesized that the effects might be mediated through androgen receptor binding and blockade. However, follow up studies suggest that these phthalate esters do not exert their toxic effects through a direct blockade of the androgen receptor. Nevertheless, these chemicals do disrupt male reproductive tract development, and that observation should not be lost in a debate about the mechanism by which this disruption occurs.

It is important to understand that endocrine disruption is an evolving field of research. A chemical need not necessarily act through a receptor-mediated mechanism to behave as an endocrine disruptor. For example, a chemical might interfere with the production or metabolism of hormones in a developing organism, thereby interfering with normal hormone function in a manner totally apart from interacting with the hormone receptor. Or, a chemical might interfere with the function of a hormone other than estrogen or androgen (a non-steroidal hormone), such as follicle stimulating hormone (FSH).

The mechanism by which MEHP (the metabolite of DEHP) interferes with Sertoli cell proliferation and function is not well understood, and the NTP Panel was "not able to reach agreement that interfering with FSH signaling function was the accepted mode or mechanism of action." However, according to the NTP panel report, "Lloyd and Foster noted that initiation of spermatogenesis was dependent on FSH interaction with the Sertoli cell in young rats, but was not necessary for maintenance of spermatogenesis in adults. Their experiment in Sertoli cell cultures demonstrated that MEHP interferes with FSH interaction at the receptor level and provided a hypothesis for increased sensitivity to testicular toxicity in young animals." (NTP report, pg 73).

Lack of complete understanding of the mechanism of toxicity in no way obviates the consistently reproduced observation that MEHP alters Sertoli cell structure and function, and that young, immature testes are significantly more sensitive than adult tissues to this effect. The DEHP exposure level necessary to elicit testicular toxicity in developing laboratory animals leads to an oral reference dose as low as 37 microgms DEHP/kg/day, which is similar to the existing EPA oral reference dose of 20 microgm/kg/day. Some oral DEHP exposures in the general population approach or exceed these values.

Phthalates and reproductive/developmental toxicity:

The PEP dismisses any concern about adverse effects on human reproduction from phthalates used in building materials. However, the PEP quotes selectively from the NTP analysis when they note that "The [NTP Panel] has a minimal concern that ambient human exposures adversely affect adult human reproduction."

What the PEP fails to say is that the NTP panel went on to comment on exposures to DEHP during pregnancy and lactation. The NTP panel concluded that "In utero development is a lifestage of particular vulnerability; exposures [to DEHP] may be on the order of 3-30 microgm/kg/day; the most relevant rodent data suggest a NOAEL [no observed adverse effect level] for testis/developmental effects of 3.7-14 mg/kg/day; DEHP produces malformations in rodents, with a NOAEL of ~40 mg/kg/day; even time limited exposures are effective at producing irreversible effects; the active toxicant MEHP passes into breast milk and crosses the placenta. On the other hand, absorption from the primate gut appears to be less effective than from the rodent gut, which reduces the level of concern for oral exposure. Given that oral exposure is <30 microgm/kg/day for humans and toxic effects are seen in rodents at >3 mg/kg/day, even in the face of significant species differences in absorption, the [NTP] panel has concern that ambient oral DEHP exposures to pregnant or lactating women may adversely affect the development of their offspring." (NTP report, pg 101-102)

DEHP contaminates the general food supply in amounts sufficient to cause the entire population to be exposed at levels close to or above the oral reference dose. From a public health perspective, efforts should be undertaken to reduce this level of contamination substantially. In order to do that, one must look for the various sources of

DEHP as it makes its way into the food supply, and take the steps necessary to curb its use and environmental releases.

Attachment 1

BARBRA BATSHALOM is the founder and Executive Director of The Green Roundtable, Inc. (GRT), an independent non-profit organization whose mission is to promote and support healthy, efficient and sustaining development and building projects. The Green Roundtable, Inc., with over 500 members, also represents the United States Green Building Council in the Boston Region. Ms. Batshalom is also the founder of Integration Design, a consulting and design firm. Both organizations have among other things provided vision and strategy consulting for major development projects in the Boston area, policy and design consulting for public school systems, and public health advocacy to encourage the City of Boston, HUD and private developers to use healthy building materials and practices. She facilitates implementation of the LEED rating system for design projects that choose to use a quantified approach to high performance design. Barbra's more than ten years of architecture and design experience include positions at Wetzel Associates in Boston and at Cambridge Seven Associates in Cambridge, MA as an architectural and exhibit designer. Her background includes a BARCH from the Boston Architectural Center and undergraduate education in social psychology from Ben Gurion University in Israel.

MARK ROSSI is a Senior Research Associate with Health Care Without Harm (HCWH) in Boston. His research focuses on the use of polyvinyl chloride (PVC) products in healthcare facilities and the availability of safer alternatives. He recently wrote *Neonatal Exposure to DEHP and Opportunities for Prevention* (July 2000) and co-authored an update of the report for Europe in October. His research on alternatives to PVC has been cited in *Chemical and Engineering News*. He has also been a Research Associate at the Massachusetts Toxics Use Reduction Institute, where he promoted the development and diffusion of environmentally safer manufacturing processes and products. Prior to that, he was a Research Associate at Tellus Institute in Boston, where he completed lifecycle assessments on seven plastic packaging materials: high-, linear low-, and low-density polyethylene, polyethylene terephthalate, polypropylene, polystyrene, and polyvinyl chloride. He has published articles in *Business Strategy and the Environment*, *New Solutions*, *Pollution Prevention Review*, and *Technology Analysis and Strategic Management*. Mark has an M.A. from Tufts University and is currently a Ph.D. student at the Massachusetts Institute of Technology.

TED SCHETTLER has a medical degree from Case-Western Reserve University and a masters degree in public health from the Harvard School of Public Health. Mr. Schettler is science director for the Science and Environmental Health Network and co-chair of the Human Health and Environment Project of Greater Boston Physicians for Social Responsibility. Schettler has worked extensively with community groups and non-governmental organizations throughout the US and the Philippines, addressing the health effects of environmental contamination and toxic exposures. Dr. Schettler is co-author of *Generations at Risk: Reproductive Health and the Environment*, which examines reproductive and developmental health effects of exposure to a variety of environmental

toxicants. Ted is also co-author of *In Harm's Way: Toxic Threats to Child Development*, which discusses the impact of environmental exposures on neurological development in children. Dr. Schettler is a member of the Environment Committee of Physicians for Social Responsibility, the committee on Occupational and Environmental Health of the Massachusetts Medical Society, and participated on the U.S. EPA's Endocrine Disruptor Screening and Testing Advisory Committee and Federal Facility Environmental Restoration Dialogue Committee. Ted Schettler is on the medical staff of Boston Medical Center and has a clinical practice at the E. Boston Neighborhood Health Center.

JOE THORNTON, Ph.D., is a research scientist in Columbia University's Earth Institute and Department of Biological Sciences. His research focuses on the health and policy implications of global chemical pollution and on the molecular evolution of animal endocrine systems. He holds Ph.D., M.A., and M.Phil. degrees in Biological Sciences from Columbia University and a B.A. from Yale University. Dr. Thornton is the author of *Pandora's Poison: Chlorine, Health, and a new Environmental Strategy* (MIT Press, 2000), which the British scientific journal *Nature* has called "a landmark book which should be read by anyone wanting to understand the environmental and health dangers of chlorine chemistry." From the late 1980s to the mid-1990s, Thornton was research analyst and then research coordinator for Greenpeace's toxics campaigns in the United States and internationally. There, he authored seminal reports and articles on organochlorines, dioxin, breast cancer, waste incineration, risk assessment, and the precautionary principle. After moving to Columbia in 1995, Dr. Thornton was co-author of the article and American Public Health Association resolution that launched the campaign to eliminate polyvinyl chloride (PVC) products from medical devices, due to their central role in dioxin formation in medical waste incinerators. He has spoken before the U.S. Congress, the EPA Science Advisory Board, the American Association for the Advancement of Science, the American Public Health Association, the International Joint Commission, and a variety of other organizations and audiences. His work has been published in numerous scientific journals, including the *Proceedings of the National Academy of Sciences*, *Annual Review of Genomics and Human Genetics*, *Public Health Reports*, *Bioessays*, *Systematic Biology*, and *International Journal of Occupational and Environmental Health*.

