RUBBER manufacturers association

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September 12, 2011

Fran Kammerer
Staff Counsel
Office of Environmental Health Hazard Assessment
1001 I Street
Sacramento, California 95812

RE: Modified Text of Proposed Regulations, Division 4.5, Title 22, California Code of Regulations Chapter 54. Green Chemistry Hazard Traits.

Dear Ms. Kammerer:

The Rubber Manufacturers Association ("RMA") is the national trade association representing every major domestic tire manufacturer including: Bridgestone Americas, Inc.; Continental Tire the Americas, LLC; Cooper Tire & Rubber Company; The Goodyear Tire & Rubber Company; Michelin North America, Inc.; Pirelli North America; Toyo Tire (U.S.A.) Corporation and Yokohama Tire Corporation. RMA appreciates this opportunity to provide comments on California's revised Green Chemistry Hazard Traits proposed rule.

Earlier this year RMA filed comments on the prior draft of this proposed rule titled "Green Chemistry Toxics Information Clearinghouse Identification of Hazard Traits, Endpoints and Other Relevant Data for Inclusion in the Toxics Information Clearinghouse." Cal. Code Regs. Tit. 22§ 54 (2010). In these comments we discussed reasons why the proposed hazard traits approach was inappropriate, not supported by sounds science, inconsistent with general principles of administrative law, and arbitrary and capricious. The revised Hazard Traits proposed regulation does not address the concerns we raised in our comments on the prior version of this regulation. As a result, RMA resubmits our comments on the prior draft of this proposed rule in their entirety and ask that OEHHA carefully consider these comments in revising the proposed Hazard Traits rule.

Specifically, RMA again makes the following recommendations which are discussed in more detail in our prior comments:

- RMA has concern that the proposed rule vastly broadens the definition of hazard and substantially lowers the level and strength of evidence that can be used to classify a substance as having a hazard trait. This proposed rule systematically changes the current risk-based product regulatory framework into a hazard-based framework. Individually, at each critical juncture, assumptions that will result in a broader definition of hazard are

adopted. Cumulatively, such changes make the framework outlined in this proposed rule extreme and unworkable.

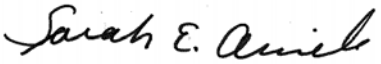
- The actual effect (albeit the unintended effect) of the proposed rule is likely to result in placing a higher priority on the substitution of chemicals (including chemicals in products) with the weakest scientific evidence because the risk assessment process increases the uncertainty and safety factors for the substances with the least scientific evidence.

- The hazard trait selection process has few or no checks and balances on the discretion of DTSC in determining whether a substance or product possesses a hazard trait. In particular, the criteria used to determine the respiratory toxicity hazard trait (for particles and fibers) are scientifically unsupportable

RMA also has concern as to how the information in the proposed Hazard Traits regulation will be applied. The proposed Hazard Traits rule will define the content for the Toxics Information Clearinghouse (TIC) and identify considerations for listing "Chemicals of Concern." Because the California Department of Toxics Substances Control's (DTSC) is still in the process of drafting a new green chemistry regulation as required by California AB 1879, it is unclear how the proposed hazard traits rule will fit into the new green chemistry regulation. As a result, RMA urges OEHHA to withdraw the proposed Hazard Traits regulation until DTSC has proposed their new green chemistry regulation, and then revise the proposed hazard traits regulation so that it has practicable application in coordination with DTSC.

RMA again thanks the OEHHA for the opportunity to comment on the proposed Hazard Traits regulation. Please contact me at (202) 682-4836 if you have questions or require additional information.

Respectfully Submitted,



Sarah E. Amick
Environmental Counsel
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Enclosure

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Subject: **Comments on the Proposed Rulemaking: Green Chemistry Toxics Information Clearinghouse Identification of Hazard Traits, Endpoints and Other Relevant Data for Inclusion in the Toxics Information Clearinghouse**

I. Introduction

The Rubber Manufacturers Association ("RMA") is the national trade association representing every major domestic tire manufacturer including: Bridgestone Americas, Inc.; Continental Tire the Americas, LLC; Cooper Tire & Rubber Company; The Goodyear Tire & Rubber Company; Michelin North America, Inc.; Pirelli North America; Toyo Tire (U.S.A.) Corporation and Yokohama Tire Corporation.

On November 1, 2010 and December 3, 2010, respectively, RMA filed comments on the prior draft proposed rule California Safer Consumer Product Alternatives regulation. Cal. Code Regs. Tit. 22, § 53 (2010) ("SCPA"). RMA appreciates the continued opportunity to offer comments on the proposed definition of hazard traits endpoints, and other relevant data ("Proposed Regulation"). Cal. Code Regs. Tit. 22, § 54 (2010). Since the impact of Definition of Hazard Traits Rule are affected by the prior portions of this proposed rule, RMA resubmits and incorporates by reference those prior comments on SCPA in this rulemaking. In summary, RMA's comments on the SPCA urged the California Department of Toxic Substances Control ("DTSC") to either exempt vehicle tires from the SCPA final rule or take the time necessary to revise this regulation to make it feasible.

II. Overview

RMA's comments on the proposed rule summarize key provisions, identify the likely impact, and discuss reasons why the proposed approach is inappropriate, not supported by sound

science, inconsistent with general principles of administrative law, and/or arbitrary and capricious. Specifically, RMA makes the following observations and recommendations which are discussed in more detail in these comments:

- RMA has concern that the proposed rule vastly broadens the definition of hazard and substantially lowers the level and strength of evidence that can be used to classify a substance as having a hazard trait. These proposed rules systematically change the current risk-based product regulatory framework into a hazard-based framework. Individually, at each critical juncture, assumptions that will result in a broader definition of hazard are adopted. Cumulatively, such changes make this framework extreme and unworkable.
- The actual effect (albeit the unintended effect) of the proposed rule is likely to result in placing a higher priority on the substitution of chemicals (including chemicals in products) with the weakest scientific evidence because the risk assessment process increases the uncertainty and safety factors for the substances with the least scientific evidence.
- The hazard trait selection process has few or no checks and balances on the discretion of DTSC in determining whether a substance or product possesses a hazard trait. In particular, the criteria used to determine the respiratory toxicity hazard trait (for particles and fibers) are scientifically unsupportable

III COMMENTS

A. Introduction

The California Health and Safety Code section 25252 requires the DTSC to evaluate and prioritize chemicals by "developing criteria that include, but are not limited to, traits, characteristics, and endpoints, developed by the California Environmental Protection Agency Office of Environmental Health Hazard Assessment ("OEHHA"), for the Toxics Information Clearinghouse."[1] The California Safer Consumer Products Alternatives proposed regulation focused mainly on the definition of "hazard trait." The proposed Toxics Information Clearinghouse Identification of Hazard Traits, Endpoints and other relevant Data regulation

[1] Initial Statement of Reasons Supporting Green Chemistry Identification of Hazard Traits, Endpoints and Other Relevant Data for Inclusion in the Toxics Information Clearinghouse at 3 of 121 (December 17, 2010) ("Initial Statement").

proposed by OEHAA goes beyond the SCPA proposed rule and defines specific hazard traits which are divided into four general categories (toxicological, environmental, exposure potential and physical).

The proposed regulation specifies general categories of endpoints for each toxicological and environmental hazard trait, as well as "other relevant data" for each toxicological and environmental hazard trait. Additionally, the regulation states how endpoint and other relevant data can be used as evidence in evaluating whether or not a chemical substance has a hazard trait, exposure potential or physical hazard trait. In particular, the SCPA proposed rule specifies the type of evidence required to determine whether a chemical (and ultimately a product) is prioritized and ultimately whether the chemical is required to be substituted in products.

However, in the proposed rule, OEHHA has significantly and substantially lessened the criteria and strength of the evidence needed to determine that a substance has a hazard trait. RMA believes this new framework will likely grind the regulatory system to a halt and may also significantly impact the legal system.

B. The Hazard Assessment Framework in the Proposed Rule Rejects Generally Accepted Scientific Principles, Causation, and The Existing U.S. Framework

1. The Provisions In The Proposed Rule

The hazard trait determination process in the proposed rule contains a series of key steps to determine whether a substance possesses a hazard trait. RMA believes that each of the steps in the hazard trait determination process lessen the quantity and quality of scientific evidence needed to determine whether a substance possesses a hazard trait.

2. Overview of RMA's issues with the Hazard Trait Determination Process

First, the proposed rule explicitly states that a hazard trait "can be demonstrated by … 'suggestive evidence,'"[2] as opposed to strong evidence or the weight of the evidence. Suggestive evidence is variously described as "positive evidence," that is not definitive of a

[2] Section 69402. Initial Statement, *supra* note 1, at 20 of 121. See Id. at 92-93 of 121.

causal association.[3] OEHHA clearly intends that suggestive evidence be used, even in the absence of strong evidence, e.g.:

> For example, if this regulation were to only describe "strong evidence" for hazard traits, an alternatives assessment might erroneously conclude that a chemical does not have a particular hazard trait even when there is evidence to suggest that it does. Overlooking suggestive evidence for a hazard trait would increase the likelihood of a business or regulatory decision that results in a regrettable substitution of one hazardous chemical for another in a product, thereby defeating one of the key purposes of the DTSC regulatory program. The description of "suggestive evidence" in this regulation will help ensure that such evidence is available in the Clearinghouse and is considered when DTSC, businesses and others weigh the advantages and drawbacks of using various chemicals as alternatives. …
>
> **Absence of data does not constitute absence of hazard, however, and the absence of strong or suggestive evidence does not translate to absence of the hazard trait** (bold face added).[4]

Second, the proposed rule defines suggestive evidence as including evidence: (a) from a "single experiment;" (b) where the design, conduct or interpretation of the studies may be questionable; (c) that "the agent increases the incidence only of benign neoplasms or lesions of uncertain neoplastic potential;" or (d) that is "restricted to studies that demonstrate only promoting activity in a narrow range of tissues or organs."[5]

Third, the "proposed regulation does not require a study be conducted in accordance with Good Laboratory Practice in order to be used in evaluating hazard traits.[6]

Fourth, the proposed rule seems to pick the easiest hazard trait determination criteria for designating a substance as a hazard from many existing hazard frameworks. The Initial

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[3] Id. at 22 of 121. Suggestive evidence is, by definition, evidence that is insufficient to prove causation. A positive "association" between an exposure (e.g., exposure to particulates) and a disease "is not necessarily proof that the exposure *caused* the disease." National Institute of Environmental Health Sciences, Questions and Answers, EMF in the Workplace, Electric and Magnetic Fields Associated with the Use of Electric Power at 10 of 40, (September 1996), *at* http://www.niehs.nih.gov/emfrapid/html/Q&A-Workplace.html ("NIEHS Q&As"). It is unlikely that any particular study, even a finding of a statistically significant increased risk in an exposed population in an epidemiological study, *proves* a causal relationship. According to US EPA, studies can only identify patterns or trends in disease occurrence over time or in different geographical locations but cannot ascertain the causal agent or degree of exposure.. EPA, Guidelines for Assessment of Carcinogen Risk at 2-5 (2005), available at http://www.epa.gov/raf/publications/pdfs/CANCER_GUIDELINES_FINAL_3-25-05.pdf ("EPA Cancer Guidelines").

[4] Initial Statement, *supra* note 1, at 21 of 121.

[5] Id. at 92 of 121.

[6] Id. at 18 of 121. This raises the specter of a substance being classified as hazardous based on bad laboratory practices.

Statement of Reasons states the "framework for each hazard trait … is loosely based on the framework used by the International Agency for Research on Cancer ["IARC"] for describing the available evidence on carcinogenicity."[7] The Initial Statement also cites an Environmental Protection Agency ("EPA") research Memorandum of Understanding used to develop screening tools as support for using *in vitro* data to identify toxicological hazards.[8]

Fifth, the proposed rule allows the use of "other data" (Subsection 69401.2(g)), which means that a substance may be classified as having a hazard trait because "data indicate[s] the potential for a hazard trait"; e.g., "an observation that a chemical" can cause placental insufficiency under certain circumstances and such an observation (e.g., placental insufficiency) may be "indirect evidence for decreased fetal weight."[9]

Sixth, the proposed rule allows DTSC to classify a substance as possessing a hazard trait merely due to a chemical or physical property. For example, the Initial Statement states that a substance may be classified a hazard because "a basic physico-chemical property is … associated with many hazard traits," such as the fact that many chemicals are strongly electrophilic (and thus, are "capable of binding to a number of large molecules in cells") and some strongly electrophilic chemicals have been demonstrated to cause a wide variety of adverse impacts.[10] Similarly, the Initial Statement cites EPA's Oncologic™ as an example model that "should be considered as suggestive of that a chemical substance may cause cancer."[11]

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[7] Id. at 6 of 121.

[8] Id. at 17 of 121, citing the July 19, 2010 Memorandum of Understanding on High Throughput Screening, Toxicity Pathway Profiling, and Biological Interpretation of Findings between the U.S. Department of Health and Human Services (HHS) National Institutes of Health (NIGH) National Institutes of Environmental Health Sciences (NIEHS)/National Toxicology Program (NTP) and the U.S. Department of Health and Human Services (HHS) National Institutes of Health (NIGH) National Human Genome Research Institute (NIGERIA) NIGH Chemical Genomics Center (NGOC) and the U.S. Environmental Protection Agency(EPA) Office of Research and Development and the U.S. Department of Health and Human Services (HHS) U.S. Food and Drug Administration (FDA), pages 1-2.

[9] Initial Statement, *supra* note 1, at 15 of 121.

[10] Id. at 15 – 16 of 121.
It should be noted that the language in the proposed rule applies to all hazard traits. The citation in the Initial Statement to the Globally Harmonized System, on the other hand, relates to labeling based on properties of a chemical or a product that may cause acute effects such as eye or skin irritation. For example, the citation to the REACH chemical classification, labeling, and packaging program applies to such acute adverse effects. Id. at 15 of 121. However, the proposed rule is more broadly written such that it might apply to the example provided in the text above.

[11] Id. at 29 of 121.

The proposed rule describes how these bases for hazard trait determination might apply for all eighteen hazard traits. Rather than comment on how these bases apply to all eighteen hazard traits, we have summarized the overall process and commented on how these procedures affect a few representative hazard traits. However, failure to comment on each trait is not agreement with the SCPA proposed rule approach.

3. The likely impact of the hazard trait determination process contained in the proposed rule

This approach contemplates that a substance might be given a hazard trait even if there was neither strong nor suggestive evidence. The plain meaning is that a hazard trait classification might be given even if there is a complete "absence of data" (see above). This radical approach is certain to result in a higher rate of false positives (i.e., a designation of a substance as a hazard when it is not one). The cumulative impact of these changes will not strengthen the scientific basis for the criteria in the proposed SCPA rule, but it will increase the likelihood of classifying a substance as a hazard that has little scientific evidence of hazard.

4. Reasons why this approach is inappropriate

The proposed rule's criteria and procedures to be used to determine whether a substance possesses a hazard trait warranting high priority consideration are (taken as a whole) contrary to generally accepted scientific principles, beyond the range of previously utilized regulatory practices, and are arbitrary and capricious. The OEHHA does not appear to have formally or informally considered the negative impacts of these changes.

First, these minimal criteria for determining a hazard significantly change the long-standing rules for designating a chemical as a hazard and, as a practical matter, render the determination of a hazard trait "virtually criteria-less."[12] Additionally, the proposed rule seems to cherry-pick the easiest hazard trait determination criteria from existing hazard frameworks without substituting a coherent framework.

Second, use of these criteria essentially means that a single experiment with methodological flaws showing an increase in benign tumors in test animals could result in the classification of a substance as a carcinogen. However, benign tumors, in and of themselves, do

[12] This intent is also demonstrated by the Initial Statement's admission that the proposed rule "is intended to promote the inclusion [in the regulatory clearinghouse] of information that is … insufficient for a finding of … suggestive." Id. at 20 of 121.

not demonstrate injury.[13] Similarly, one experiment demonstrating a statistically significant higher rate of disease in, for example, a rat study could be due to random variation, confounding factors, or methodological flaws. There are examples where preliminary epidemiological study findings have been found not to be statistically significant once a longer number of years of exposure were used in the analysis.

Third, this approach systematically eliminates (or ignores) causation,[14] which is, by definition, arbitrary and capricious. It is generally established that "association is not causation."[15] Nothing in the plain meaning of the proposed rule or its Initial Statement discusses the fact that there has been a long-standing reliance on causation in regulatory proceedings or the tradeoffs involved in making such a radical change in framework.

As acknowledged by EPA's Guidelines on the Assessment of Carcinogen Risk:

> Determining whether an observed association (risk) is causal rather than spurious involves consideration of a number of factors. Sir Bradford Hill (Hill, 1965) developed a set of guidelines for evaluating epidemiologic associations that can be used in conjunction with the discussion of causality such as the 2004 Surgeon General's report on smoking (CDC, 2004) and in other documents (e.g., Rothman and Greenland 1998; IPCS, 1999). The critical assessment of epidemiologic evidence is conceptually based upon consideration of salient aspects of the evidence of associations so as to reach fundamental judgments as to the likely causal significance of the observed associations. In so doing, it is appropriate to draw from those aspects initially presented in Hill's classic monograph (Hill, 1965) and widely used by the scientific community in conducting such evidence-based reviews. A number of these aspects are judged to be particularly salient in evaluating the body of evidence available in this review, including the aspects

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[13] "Observation of only benign neoplasia may or may not have significance for evaluation under these cancer guidelines" and "observation of a benign tumor response alone may have no significant health hazard implications when other sources of evidence show no suggestion of carcinogenicity " EPA, Guidelines for Assessment of Carcinogen Risk 2-2 (2005), available at http://www.epa.gov/raf/publications/pdfs/CANCER_GUIDELINES_FINAL_3-25-05.pdf.

[14] "Cause-and-effect relationships often are quite subtle, and carefully designed studies are needed to draw valid conclusions." *Reference Manual on Scientific Evidence* at 92 (2d Ed., Federal Judicial Center, 2000), available at http://www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/$file/sciman00.pdf ("Judicial Science Manual"). We do not assume that causation for the purpose of issuing regulations is identical to causation required by science or in personal injury litigation. The distinction between these two levels of proof has been the subject of numerous academic articles. We need not address for the purposes of these comments what is the appropriate level of causation for regulation because this proposed rule virtually eliminates causation and defines the level of causation necessary far below any existing regulation.

[15] Judicial Science Manual, supra, note 14, at 94, citing Experimentation in the Law: Report of the Federal Judicial Center Advisory Committee on Experimentation in the Law (Federal Judicial Center 1981).

> described by Hill as strength, experiment, consistency, plausibility, and coherence. Other aspects identified by Hill, including temporality and biological gradient, are also relevant and considered here (e.g., in characterizing lag structures and concentration-response relationships), but are more directly addressed in the design and analyses of the individual epidemiologic studies included in this assessment. As discussed below, these salient aspects are interrelated and considered throughout the evaluation of the epidemiologic evidence generally reflected in the integrative synthesis of the mode of action framework.[16]

The widely accepted principles of causation[17] are: (1) the temporal relationship;[18] (2) consistency of the effect across different studies;[19] (3) the magnitude of the theoretic incidence of disease in an exposed population compared to an unexposed or less exposed population (called relative risk) found in the studies (i.e., preferably there is a relative risk ratio[20] of greater

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[16] EPA Cancer Guidelines, *supra* note 13, at 2-11 to 2-12.

Some, but not all, of these criteria are similar to the factors required by trial courts to assess "evidential reliability" of the scientific method used to reach scientific conclusion offered in testimony, i.e.:

1. The theoretical underpinnings of the methods must yield testable predictions by means of which the theory could be falsified.
2. The methods should preferably be published in a peer-reviewed journal.
3. There should be a known rate of error that can be used in evaluating the results.
4. The methods should be generally accepted within the relevant scientific community. Judicial Science Manual, *supra* note 18, at 82, citing *Daubert v. Merrell Dow Pharms., Inc*., 509 U.S. 579 (1993). Note that the court in *Daubert* was not interpreting a statute, but developing rules for courts to use in assessing the scientific reliability of evidence in judicial proceedings.

[17] A.B. Hill, "The Environment and Diseases: Association and Causation", 58 *Proc. Royal Soc. Med, Sec. Occup. Med.* 295-300 (1965), available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1898525/pdf/procrsmed00196-0010.pdf. The A.B. Hill. criteria have long been recognized as the key criteria to assess causality, as demonstrated by EPA's citation to them in the EPA regulatory cancer guidelines. Although these principles were first developed in the context of evaluating the likelihood that exposure of a substance causes an adverse health effect, these principles evince a more generally accepted set of principles for evaluating the scientific validity generally.

[18] The adverse health effect has to occur within a biologically-reasonable time after initial exposure. Because some adverse health effects are believed to become manifest only after a latency period, the determination of temporal effect is not always straightforward. *See* EPA Cancer Guidelines, *supra* note 13, at 2-11 to 2-12.

[19] The strongest association occurs when the same effect in the same organ with the same biological mechanism is observed in several independent studies of a similar exposure in different populations. The association may be strong if it consistently occurs in different subgroups in the same study. Bias and confounding factors need to be taken into account in reviewing multiple studies.

[20] The relative risk is the ratio of the risk of disease or death among the exposed population to the risk in the unexposed population.

than 2.0);[21] (4) whether there is a biological gradient between the level of exposure and the magnitude of the effect (that is, does higher exposure result in a higher incidence of disease);[22] (5) the specificity of the association; (6) the biological plausibility that the exposure could cause the effect;[23] and (7) coherence.[24]

Scientific causation "can be answered only through … formulating a question that can be answered, designing a study that can answer it, collecting objectively verifiable evidence that will address the question, and drawing only those conclusions supported by the evidence."[25] However, "a theory can never be proved right by agreement with observation, but it can be proved wrong by disagreement with observation. Because of this asymmetry, science makes progress uniquely by proving that good ideas are wrong so that they can be replaced by even better ideas."[26] RMA recommends that it is inappropriate to abandon causation, particularly in

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[21] A statistically significant relative risk of 2.0 means that the more exposed population had twice the rate of disease than expected from an examination of a population that was not exposed or significantly less exposed. Thus, in those situations where, based on the weight of the evidence, the exposure caused the increased relative risk of 2.0, any one individual in the exposed population would have a 50% probability of having their disease caused by the exposure. J. Rosenbaum, *Lessons from Litigation over Silicone Breast Implants: A Call for Activism By Scientists*, Vol. 276, SCIENCE, No. 5318, Issue 6, p. 1524 (June 6, 1997), *at* http://www.sciencemag.org/content/vol276/issue5318/.

According to US EPA, "when a relative risk is less than 2, if confounders [i.e., other potential causes of the disease being studied]… are having an effect on the observed risk increases, it could be enough to account for the increased risk." US EPA, Health Assessment Document for Diesel Engine Exhaust at 7-138 (EPA/600/8-90/057F, May 2002), *at* http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060.

[22] Generally, the risk ratio should increase with increasing exposure or dose. Generally, the lack of a dose-response relationship, in and of itself, may not be sufficient evidence against a causal relationship.

[23] Biological plausibility is the factor that is both conceptually simple to understand, yet difficult to apply. The mechanism for many diseases is not yet fully understood. Generally, toxicologists evaluate data from animal studies, the toxicokinetics of the substance (i.e., how the chemical moves through the body and interacts with cells), the structure-activity relationship compared to other known carcinogens, and the data from short-term studies of the agent's influence on biological steps known or believed to occur.

[24] Evaluating coherence involves comparing the assumed cause-and-effect relationship with what is known about the history and biology of the disease, i.e., the entire body of knowledge about the agent. Some authors do not include coherence as a separate factor. The A.B. Hill. criteria (discussed above) have long been recognized as the key criteria to assess causality. *See also* International Agency for Research on Cancer, World Health Organization, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Nonionizing Radiation, Part 1: Static and Extremely Low-Frequency (ELF) Electric and Magnetic Fields* (Vol. 80) pp.16 (2002, Lyon, France) ("IARC ELF Monograph").

[25] Brief of Amicus Curiae The New England Journal of Medicine and Marcia Angell, M.D., In Support of Neither Petitioners Nor Respondents at 5, in General Electric Co. v. Joiner, 522 U.S. 136, 118 S. Ct. 512 (1997).

[26] Judicial Science Manual, *supra* note 14, at 70.

the context of a regulation that provides for legally mandated ban on the use of chemical in a product or all products.

Fourth, OEHHA does not even attempt to assess the trade-off between false positives and false negatives for each individual criterion, no less the cumulative impact. This proposed hazard determination framework radically alters the long-standing hazard assessment and risk assessment framework. Thus, a careful consideration of the positive and negative impacts of such a change is necessary.

The decision to regulate is based on balancing the calculated upper-end regulatory risk against the degree of uncertainty in the risk calculation, the feasibility of pollution controls, the societal impacts of such controls, and the costs of implementing the regulatory controls (i.e. the so called risk management decision).[27] As a matter of policy and law, it is now well established that "safe" is not necessarily the same as "risk-free," and mere exposure is not sufficient to support regulation unless there is a significant risk.[28] The proposed rule moves the regulatory risk assessment framework close to a mere exposure level.

Such a dramatic shift in policy risks undermines the credibility of the regulatory process. The costs of removing substances and products that present no meaningful risk can be significant.

Fifth, while some physical traits (e.g., acidity) may cause an adverse effect in most uses (e.g., irritation when sprayed in the eyes), the language in the proposed rule could be interpreted as allowing the designation of any hazard trait based on physical and chemical properties. Thus, for some chemicals, the proposed rule can be interpreted as allowing chemical/physical data to demonstrate that all chemicals containing that chemical and/or physical property causes an adverse health effect.

Similarly, the proposal to use the Oncologic™ model to determine whether a specific chemical or substance is a carcinogen is, at best, premature and its citation in the Initial Statement is likely to be misleading and should be removed. While much research has been done

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[27] The Presidential/Congressional Commission On Risk Assessment And Risk Management, Risk Assessment And Risk Management In Regulatory Decision-Making, Final Report, Vol. 2, at 2-5 (1997), also available at http://www.riskworld.com ("National Risk Commission Final Report"). The Initial Statement cherry-picks various National Academies of Science reports and other expert body recommendations. These quotes are selective and do not reflect a societal consensus on how best to reduce the risk of toxic chemicals in products.

[28] *Industrial Union Dep't. v. API,* 448 U.S. 607, 642 (1980); *Natural Resources Defense Council v. EPA,* 824 F.2d 1146, 1164-65 (D.C. Cir. 1987) (unanimous en banc decision) ("NRDC v. EPA").

in this area, there simply is not sufficient scientific certainty currently available for such a broad generalization.

In summary, the proposed rule allows mechanistic evidence "alone" to "provide strong evidence of carcinogenicity" or in some cases, to provide evidence suggestive of carcinogenic potential.[29] Therefore, the proposed rule allows classification of substance as having a hazard trait based merely on suggestive mechanistic evidence.

Sixth, although the Initial Statement of Reasons states the "framework for each hazard trait … is loosely based on the framework used by the International Agency for Research on Cancer [IARC] for describing the available evidence on carcinogenicity,"[30] the Initial Statement cites a myriad of governmental frameworks and nonregulatory documents to support one or another aspect of the hazard trait framework.[31]

Thus, the framework for decision-making appears to choose a series of criteria from different existing frameworks. As a result, the tradeoffs and balancing that resulted in each of these other hazard frameworks is ignored. In fact, it appears the SCPA hazard trait decision criteria are simply a list of the most draconian decision criterion rather than a balanced scheme designed to meet the needs of the SCPA.

Seventh, even assuming that the claim that OEHHA modeled its cancer classification framework on the one used by IARC is accurate, the selection of the IARC was not reasoned, is arbitrary, and is inconsistent with some of the examples provided in the Initial Statement. Nothing in the Initial Statement compares the IARC,[32] EPA,[33] NTP[34] or other frameworks for classifying chemicals as carcinogens or other hazard traits. More importantly, both the proposed rule and the Initial Statement fail to distinguish between the IARC mission of prioritizing

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[29] Initial Statement, *supra* note 1, at 28 of 121.

[30] Id. at 6 of 121.

[31] For example, the Initial Statement cites the EPA Cancer guidelines, the NTP,, the Globally Harmonized System, among other agency for definitions, such as "the term 'toxicological endpoint' or 'toxic endpoint' more narrowly. Id. at 16-17 of 121.

[32] IARC, Monographs on the Evaluation of Carcinogenic Risks to Humans (revised 2006), available at http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf.

[33] EPA Carcinogen Guidelines, *supra* note 13,..

[34] NTP, Report On Carcinogens, Eleventh Edition Introduction at 2, available at http://ntp.niehs.nih.gov/ntp/roc/eleventh/intro.pdf.

chemicals for further research and consideration by national regulatory agencies, and the purpose of the Safer Consumer Product Alternatives proposed rule to prioritize which chemicals and products should undergo rigorous review regarding the availability of less toxic alternative chemicals.

Eighth, the criteria do not distinguish between observations that a chemical "can" cause an effect versus a chemical is likely to cause an effect in actual use.

In summary, the proposed rule impermissibly allows one study, a statistical quirk or a poorly performed study protocol to impose significant economic costs with little or no risk reduction benefit. The level and strength of evidence has been unreasonably lowered by this proposed rule. The long-held causation principles support making a hazard trait determination based on the weight of the scientific evidence as a whole rather than an approach that selectively relies on only studies or other information that appears in isolation to suggest adverse effects. The framework articulated by this proposed hazard trait rule seems to reject basic scientific principles. A determination that a substance has a hazard trait that may justify restricting a products use or banning its sale altogether must only be made after evaluating the weight of all the scientific evidence in light of long established scientific principles.

C. The Definition of Respiratory Toxicity Is Overly Broad

1. The Provisions In The Proposed Rule

Contrary to the existing risk assessment framework in the United States, the proposed rule also greatly reduces the evidence needed to classify a particle or fiber as possessing a respiratory toxicity hazard trait. The proposed rule defines a respiratory toxicity hazard "as an adverse change in the structure or function of the respiratory tract following exposure to a chemical substance, including respiratory tract injury or decreased ability of the lungs to function in gas exchange."[35] Respiratory tract injury endpoints "include, but are not limited to those indicating: respiratory irritation; pathological changes to the airway or other lung structures; inflammation; fibrosis; hypersensitivity pneumonitis; airways hyperresponsiveness; altered lung function; asthma; airways remodeling; increased respiratory infections; altered composition of

[35] § 69403.15(a) Respiratory Toxicity.

bronchoalveolar lavage fluid."[36] Other relevant data "include but are not limited to: in vitro evidence for respiratory toxicity; particle size distribution inclusive of respirable particles; respirable fibers; long half-life in the lung; chemical reactivity; redox potential; structural or mechanistic similarity to other chemical substances with the respiratory toxicity hazard trait."[37]

However, "suggestive evidence" includes:

- "An authoritative organization identifies or discusses the chemical substance as possibly having the hazard trait."
- "A well-conducted scientific study indicates exposure to the chemical substance induces a toxicological endpoint or endpoints for the hazard trait."
- "Strong indications of the hazard trait from **structure activity relationships**, including but not limited to those from validated Quantitative Structure Activity Relationship models." (red highlighting added)
- "**Mechanistic evidence that is suggestive of the hazard trait, from cell-based, tissue-based or whole organism-based assays showing perturbations of known physiological, biochemical or other pathways involved in causing the hazard trai**t." (red highlighting added)[38]

DTSC interprets this as meaning that:

- [M]aterials that are fibrous in nature, and respirable, such as asbestos, can deposit in the lung and cause damage along the respiratory tract. Chemicals that are gases tend to impact the upper airway if they are water soluble, and impacts can extend down to the lower airway and parenchyma if they are less water soluble. In all cases, the extent of damage is dependent on the chemical concentration to which animals or people are exposed" (footnotes deleted);[39]
- "Particle size and fiber dimension influences where in the respiratory system a particle phase chemical will deposit and influences the toxicity of the chemical. If the chemical substance has a long half-life in the lung, then the probability of adverse health impacts from toxicity increases;"[40] and

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[36] § 69403.15(b) Respiratory Toxicity.

[37] § 69403.15(c) Respiratory Toxicity.

[38] § 69403.16(b) Evidence for Toxicological Hazard Traits. At p. 16-17 out of 24.

[39] Initial Statement, *supra* note 1, at 88 of 121.

[40] Id. at 91 of 121.

- "[A] series of *in vitro* assays demonstrating the potential for a compound to produce prolonged inflammation would be suggestive evidence for the respiratory hazard trait, among others."[41]

Also, according to DTSC:

> A chemical that has not been tested for respiratory toxicity but which has a high redox potential (ability to oxidize other molecules) may form destructive reactive oxygen species in the lung. Particle size and fiber dimension influences where in the respiratory system a particle phase chemical will deposit and influences the toxicity of the chemical. If the chemical substance has a long half-life in the lung, then the probability of adverse health impacts from toxicity increases.[42]

In summary, the criteria proposed to determine whether particles or fibers possess a hazard trait would be dramatically narrowed.

2. The Likely Impact

Thus, based on the proposed rules, mechanistic and other suggestive evidence (perhaps as little as the use of broad physical and chemical characteristics of particles or fibers) might be used to designate a wide range of particles and/or fibers as possessing a hazard trait and eventually to be used in a risk assessment.

3. The Reasons That the Proposed Rule Is Inappropriate

The use of criteria to determine whether particles or fibers possess a hazard trait that dramatically lessen the scientific evidence necessary is inappropriate and unwarranted for the following reasons.

First, there is no scientific support for broadly concluding that substances with similar physical and chemical properties (alone) prove that the toxicological effects will be the same. The Initial statement provides no scientific basis and no independent scientific body has made such a broad recommendation.

Second, basing a hazard trait determination solely on extrapolation from the rat inhalation bioassays where the level of animal exposure overwhelms the natural lung clearance defense to human health effects (i.e., lung overburdening studies), as provided in the proposed hazard trait

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[41] Id. at 93 of 121.

[42] Id. at 91 of 121.

rule) is not justified. The lung overburdening studies with other test animals (other than rats) do not result in lung overburdening or increases in tumors.[43]

The claim that the lung overburden rat studies of particle or fiber exposure can demonstrate whether such exposure causes cancer in humans is not biologically plausible. These lung overburdening rat studies use exposure levels that overload the lung clearance mechanism in rats. Given the different physiology of humans and rats, this mechanism is not relevant to human toxicity.[44] EPA,[45] the American Conference of Government and Industrial Hygienist

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[43] MRC IEH, Workshop on *Approaches to predicting toxicity for occupational exposure to dusts* (IEH, 1999).

[44] Comments of John Addison and Arthur M. Langer, On the NTP Report on Carcinogens Background Document for Asbestiform Talc and Non-asbestiform Talc, at 11 (November 30, 2000), available at: http://dir.niehs.nih.gov/dirtob/rocpubcom/10throc/2000nominations/talcasbestiform-nonasbestiform/jaddison-11-30-00.pdf ("Addison and Langer") and. Gűnter Oberdörster, DVM, Ph.D., Comments on NTP Proposed listing of Talc Asbestiform and Non-Asbestiform, As Reasonbly Anticipated to be a Human Carcinogen at 2-6 (November 20, 2000), available at http://dir.niehs.nih.gov/dirtob/rocpubcom/10throc/2000nominations/talcasbestiform-nonasbestiform/oberdorster-11-29-00.pdf, ("Oberdörster Comments").

[45] EPA, Toxic Chemical Release Reporting; Community Right-to-Know; Titanium Dioxide, 53 Fed. Reg. 23106, 23111 (1988), which concluded that titanium dioxide is not a carcinogen despite lung overburden rat study results. In 2002, EPA concluded that:

> The lung cancer response in rates from high-concentration exposures [to diesel exhaust] appear to be mediate by impairment of lung clearance mechanisms through particle overload, resulting in persistent chronic inflammation and subsequent pathologic and neoplastic changes in the lung. Overload conditions are not expected to occur in humans as a result of environmental and most occupational exposures to DE. Thus, the rat lung tumor response is not considered relevant to an evaluation of the potential human environmental exposure-related hazard. EPA, Health Assessment Document for Diesel Engine Exhaust at 7-139 (EPA/600/8-90/057F, May 2002).

Similarly, in 2003, EPA concluded in its assessment of the health impact of particulate matter that the

> relevance of lung overload to humans exposed to poorly soluble, nonfibrous particles remains unclear" and "is likely to be of little relevance for most 'real world' ambient exposures," although it may be of concern in interpreting some long-term experimental exposure data and, perhaps, also for occupational exposures." EPA Fourth External Review Draft of Air Quality Criteria for Particulate Matter (June 2003), Volume II at 6-59 (2003).

In addition, Vanessa Vu of EPA's Office of Pollution and Toxics concluded in a paper reviewed and approved by EPA for publication that:

> several insoluble biochemically inert particles (e.g., titanium dioxide, talc, test toner) have been shown to cause fibrogenic and/or carcinogenic effects in rats at high exposure concentrations. These effects have been attributed to particle overloading in the lungs of the animals due to excessive particle exposure … For these reasons, EPA/OPPT does not general consider any new particulate substance of low intrinsic toxicity to be of high concern. However, if these particles are produced in large amounts and/or if there is substantial human exposure to them, toxicological testing is generally requested for a full risk assessment.

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(ACGIH (2000)), the National Commission on Risk Assessment and Risk Management (1997),[46] a joint workshop on talc sponsored by the FDA and International Society of Regulatory Toxicology and Pharmacology;[47] IARC,[48] and other reputable scientific bodies and individual scientists[49] have concluded that the rat lung overburdening studies are not relevant to cancer in humans. Also, statistically significant increase in malignant and benign lung tumors in humans exposed to particles in lung overburdening conditions has not been observed in other test animals nor in humans for many particles. [50] The rat reaction to lung overburdening is species-specific.[51]

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V. Vu, Use of Hazard and Risk Information in Risk Management Decisions: Solid Particles and Fibers Under EPA's TSCA and EPCRA at 10, in Proceedings of MIT Toxicology Symposium (March 1995).

Cancer classification policy determinations by EPA and National Academy of Science panels support using mechanism of action, metabolism, and other chemical specific factors in making determinations of whether animal data should be extrapolated to humans (*e.g.*, EPA, Proposed and Interim Guidelines for Cancer Risk Assessment (NCEA-I-024, July 1995)(Renew Draft)).

[46] National Risk Commission Report, *supra* note 27, at 65.

[47] The Executive Summary of this joint FDA and International Society of Regulatory Toxicology and Pharmacology workshop on talc states: "In regard to the NTP talc bioassay in rodents, it [the unanimous expert panel] found that because of the extreme doses and the unrealistic particle sizes of the talc employed, because of the negative results in mice and male rats, because of the lack of tumor excess at the low doses, and because of the clear biochemical and cytological markers of excessive toxicity in female rats, the positive talc bioassay results in female F344/N rats are likely experimental artifact and non generic response of dust overload of lungs and not a reflection of a direct activity of talc." Workshop on Talc: Consumer Uses and Health Perspectives, cosponsored by International Society of Regulatory Toxicology and Pharmacology and the US Food and Drug Administration, Bethesda, MD, January 31- February 1, 1994, J. Reg. Tox. and Pharm. 21 211, at 215 (1995) ("Talc: Consumer Uses and Health Perspectives").

[48] Comments of Roger McClellan, DVM, DABT, DABVT, Critique of "Draft Report on Carcinogens Background Document for Talc; Asbestiform and Non-Asbestiform"at 6 (December 1, 2000), available at: http://dir.niehs.nih.gov/dirtob/rocpubcom/10throc/2000nominations/talcasbestiform-nonasbestiform/mcclellan-12-1-00.pdf ("McClellan Comments").

[49] Cogent summaries of the evidence that lung overburden studies in rats are not relevant to human exposure are provided in A. Watson and P. Valberg of Gradient Corporation, Particle-Induced Lung Tumors in Rats: Evidence for Species-Specificity in Mechanism, in Proceedings of MIT Toxicology Symposium (March 1995) ("Valberg Paper") and McClellan Comments, *supra* note 48, at 6.

[50] MRC IEH, Workshop on *Approaches to predicting toxicity for occupational exposure to dusts* (IEH, 1999); IARC Monographs On the Evaluation of Carcinogenic Risks to Humans, Vol. 68, Silica, Some Silicates, Coal Dust and Para-armid Fibrils 34 (1997) ("IARC Silica Monograph"); IARC Monographs On the Evaluation of Carcinogenic Risks to Humans, Vol. 65, Printing Processes and Printing Inks, Carbon Black and Some Nitro Compounds at 241-243 (1996) ("IARC Carbon Black Monograph"); Valberg Paper, *supra* note 49 and McClellan Comments, *supra* note 48, at 6.

Epidemiological data with humans exposed to very high levels of fine particulates (e.g., studies of exposure to high levels of carbon black, coal, talc, diesel exhaust, silica, among others) demonstrate that lung overburdening does not occur in humans and there are no increased rates of lung cancer in such workers. In 1997 IARC's Working Group for coal dust acknowledged the "increasing evidence supporting the hypothesis that the tumors represent a generic response of the rat lung."[52] Similarly, in 2006, the International Agency for Research on Cancer (IARC) "working group concluded that inhaled talc (i.e., an inert fiber) that does not contain asbestos or asbestiform fibers is not classifiable as to its carcinogenicity (i.e,, group 3)."[53]

As a practical matter, it is highly relevant that the populations with the highest exposures to many different particles, including some which are clearly more toxic than inert fibers and particles, demonstrate no lung overburdening or lung tumor responses similar to those found in the lung overburdening studies.[54] In fact, the practical implication of the fact that the worker exposure levels are not exceeded or even approached by exposure levels in the general population reinforces the scientific consensus that lung overburdening studies are not relevant to cancer hazards in humans.

The mode (or mechanism) of action and metabolism information available indicates that the lung overburdening of the body's natural defenses which is observed in rats exposed to fibers and particles does not and should not occur in humans. Rats are much more sensitive to the inhalation of high levels of particulates than a human.

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[51]P. Valberg, Gradient, Public Review Comments On U.S. EPA's "Health Assessment Document For Diesel Emissions Vols. I and II" (April 1995).

[52] IARC Monographs On the Evaluation of Carcinogenic Risks to Humans, Vol. 68, Silica, Some Silicates, Coal Dust and Para-armid Fibrils 34 (1997) ("IARC Monograph").

[53] IARC Monograph Supplement 17, available at http://monographs.iarc.fr/ENG/Monographs/suppl7/Suppl7-144.pdf; and Policy Watch: Carcinogenicity of carbon black, titanium dioxide, and talc. Robert Baan, Kurt Straif, Yann Grosse, Béatrice Secretan, Fatiha El Ghissassi, Vincent Cogliano, on behalf of the WHO International Agency for Research on Cancer Monograph Working Group, The Lancet Oncology, Volume 7, Issue 4, Pages 295 - 296, April 2006, available at http://www.thelancet.com/journals/lanonc/article/PIIS1470-2045(06)70651-9/fulltext.

[54] It is an impossible burden to demand that negative epidemiological studies and other studies definitively disprove the possibility of a hazard.

Third, the extreme criteria cited in the proposed rule for classifying particles and fibers as carcinogenic is inconsistent a number of recommendations of findings of expert panel.

Many expert groups who have reviewed the risk assessment process (including the bipartisan Presidential and Congressional Risk Commission ("National Risk Commission"))[55] and various Committees of the NAS[56] have recommended the use of more realistic methodologies in the risk assessment process so that the process does not unduly overestimate the risk from exposure to particles. In fact, the Chairman of the National Risk Commission specifically cited carbon black and the lack of relevance of lung overburdening studies to human toxicity in the press conference announcing the results of the commission's study. Similar recommendations have been made in the context of assessing the toxicity of individual substances, specifically:

- IARC reclassified glass wool (except for special purpose fibers) from "possibly carcinogenic to humans" (IARC 2B) to "not classifiable as to carcinogenicity to humans (Group 3) and the National Toxicological Program's ("NTP") Glass Wool Expert Panel and Board of Scientific Counselors and other expert groups supported this distinction because the weight of the scientific evidence demonstrates that more soluable fibers are less toxic and the scientific evidence specific to each glass wool fiber should determine whether that type of fiber should be classified as carcinogenic.[57] That is, rather than

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[55] National Risk Commission Report, *supra* note 27.

[56] Committee On Risk Assessment of Hazardous Air Pollutants, National Research Council, Science and Judgment in Risk Assessment (National Academy Press, 1994) ("NAS Science and Judgment Rep.").

[57] IARC Monographs On The Evaluation Of Carcinogenic Risks To Humans: *Man-made Vitreous Fibres*, Volume 81 at 339 (published 2002, working group held in 2001). The Working Group elected not to make an overall evaluation of the newly developed fibres designed to be less biopersistent such as the alkaline earth silicate or high alumina, low-silica wools. This decision was made in part because no human data were available, although such fibres that have been tested appear to have low carcinogenic potential in experimental animals.
Glass Wool Fibers Expert Panel Report Part B – Recommendation for Listing Status for Glass Wool Fibers and Scientific Justification for the Recommendation (June 2009), available at http://ntp.niehs.nih.gov/Ntp/roc/twelfth/2009/june/GWF_PartB.pdf. All but one of the lead reviewers from the Board of Scientific Counselors expressed the view that not all glass wool fibers cause cancer (i.e., Dr. Cattley, Dr. McDiarmid, Dr. Teeguarden, Dr. Zelikoff, Dr. Eastmond, and Dr. Faustman). NTP Summary Minutes of Board of Scientific Counselors June 21-22, 2010 Meeting on glass wool at 23-26, available at http://ntp.niehs.nih.gov/ntp/About_NTP/BSC/2010/June/Minutes20100622.pdf. NTP. 2010. DRAFT Report on Carcinogens Substance Profile for Glass Wool Fibers (Respirable) as a Class at 1 (2010) (Peer review — June 21-22, 2010 Board of Scientific Counselors Meeting) ("Glass Wool Substance Profile"), available at http://ntp.niehs.nih.gov/NTP/RoC/twelfth/2010/DrftSubProfiles/GWF20100604.pdf. The draft also acknowledges that "the carcinogenicity of individual glass wool fibers must be evaluated on a case-by-case basis until the properties that lead to development of cancer after inhalation exposure are more clearly defined." Id. at 1-2.

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- The National Academies of Science ("NAS") Committee recommended that the National Institute of Occupational Safety and Health ("NIOSH") [58] and EPA's Science Advisory Board for Asbestos[59] determine toxicity based on specific scientific evidence of toxicity

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European Union (Note Q of the Regulation (CE) n° 1272/2008), available at http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2008:353:0001:1355:EN:PDF. "All mineral wool insulation products manufactured by EUCEB members … are not classified as dangerous in accordance with the European Union regulations on classification, labelling and packaging of substances and mixtures" *See* available at http://www.euceb.org/.

German Dangerous Substances Act. *See* NTP, FINAL Report on Carcinogens Background Document for Glass Wool Fibers at 13-14 (September 9, 2009), available at http://ntp.niehs.nih.gov/ntp/roc/twelfth/2009/June/GWF_Final_Background%20Document.pdf. "[E]ven long-term exposure to glass wool does not cause cancer in humans or animals. Even where glass wool fibres were clearly shown to reach the lungs of lab animals, there was no increase in tumours." Health Canada, THE SAFETY OF MANMADE VITREOUS FIBRES at 1 (2006), available at http://www.hc-sc.gc.ca/hl-vs/alt_formats/pacrb-dgapcr/pdf/iyh-vsv/prod/vitre-eng.pdf.
Environment Canada classifies glass wool as not carcinogenic. Australian and New Zealand manufacturers "have assessed that their products are not classified as hazardous" according to the criteria in the National Occupational Health and Safety Commission.." The Insulation Council of Australia and New Zealand ("ICANZ"), Glass Wool & Rock Wool Health and Safety, ICANZ Fact , Sheet 1 (2005), available at http://www.icanz.org.au/pdf/FS1_Health_web_copy.pdf.

[58] NAS. 2009. *Review of the NIOSH roadmap for research on asbestos fibers and other elongate mineral particles. at 84-85* Washington, DC: The National Academies Press NAS recommended that NIOSH "should revise the Roadmap to describe a systematic tiered strategy for characterizing and testing the relative toxicities of elongate mineral particles and/or their mixtures … using [among other things] tiered panels of in vitro and in vivo assays of increasing complexity to identify and characterize biological responses and categorize the potential hazards." Id.

[59] Letter from Dr. Agnes Kane, Chair of Science Advisory Board Asbestos Committee to EPA Administrator Stephen Johnson, Re: Consultation on the U.S. Environmental Protection Agency's *Proposed Approach for Estimation of Bin-specific Cancer Potency Factors for Inhalation Exposure to Asbestos* at 2 (November 14, 2008), available at http://www.southbayriders.com/ccma/epa_2008c_prop_approach_asbestos_bin.pdf. The SAB found that "the scientific basis as laid out in the technical document in support of the proposed [bins-specific] method [of calculating risks of mineral fibers] is weak and inadequate" and is "woefully inadequate with respect to the representation of available information on epidemiology, toxicology, mechanism of action and susceptibility." That is, adequate epidemiology, toxicology, mechanism of action and susceptibility information is needed before characterizing mineral fibers as highly toxic;

EPA proposed a "bin-specific cancer potency factor approach" to calculate the risk from exposure to asbestiform fibers (based on the distribution of the length and width of fibers and perhaps types of the mineral constituents). EPA, Office of Solid Waste and Emergency Response, *Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos* (April 2008), available at < http://www.epa.gov/oswer/riskassessment/asbestos/pdfs/2008_prop_asbestos_approach.pdf> ("Asbestos Bin-Specific Cancer Potency Approach"). There is even less scientific evidence for other fibers.

As a result of these comments, EPA decided not to pursue the effort. Letter from Stephen Johnson, Administrator of EPA to the Science Advisory Board Asbestos Committee, re: *Proposed Approach for Estimation of Bin-specific Cancer Potency Factors for Inhalation Exposure to Asbestos* (December 29, 2008) , available at

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of the specific type of asbestos fiber and other mineral particles (using a systematic tiered testing of the relative toxicities of elongate mineral particles and/or their mixtures) rather than rely on broad overly protective characterization of fibers as a class.

In each case, an expert body concluded that the science was not sufficient to characterize a particle and/or fiber as a carcinogen based solely on chemical and physical properties.

Thus, there is no scientific support for broadly concluding that substances with similar physical and chemical properties (alone) will have the same toxicological effects. RMA recommends that the DTSC should abandon the virtually boundless criteria in the proposed rule for classifying any fiber or particle as a hazard and allow a case-by-case determination using generally accepted scientific principles.

D. The SCPA Rule Must Consider Dose-Response and Risk in the Hazard Trait Classification

DTSC specifically rejected considering dose-response relationships because the hazard trait framework "incorporates dose-response information in prioritizing chemicals of concern and in alternatives analysis."[60] This approach will allow the triggering regulatory action (i.e., the initiation of the prioritization and substitution assessment process), based on weak data. We believe this approach is bad policy.

First, contrary to the inference in the Initial Statement, the classification of a substance as possessing a hazard trait triggers regulatory action (potentially including banning the sale of products), not just a hazard identification classification which typically is used in material safety data sheets, worker hazard communication, and labeling. Second, a biological gradient between the level of exposure and the magnitude of the effect is embedded in the causation determination necessary to make a hazard trait classification (see discussion above). Third, if a substance only represents a hazard at levels of exposure that simply do not exist during product use (or even product misuse); it makes no sense to classify a substance as possessing a hazard trait. Fourth, DTSC will be forced to prioritize an enormous number of chemicals and products for a

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http://yosemite.epa.gov/sab/sabproduct.nsf/77CFF6439C00ABF3852575010077801F/$File/EPA-SAB-09-004+Response+12-29-2008.pdf.

[60] Initial Statement, *supra* note at 14 of 121.

substitution assessment based on minimal evidence, which wastes both governmental and private sector resources and ultimately does not significantly reduce risk.

The traditional risk assessment framework uses hazard assessment to screen chemicals so the chemicals with the highest potential for human risk require additional research and, if appropriate, eventual regulation. The proposed rule designates substances as a hazard based on a scintilla of evidence, thereby encompassing a larger number of substances as a priority. As a result, the costs of implementing the proposed rule are likely to be very high.

E. A Biological Change Should Not Be a Hazard Trait

OEHHA argues that defining "adverse effect" as "a biochemical change, … that negatively affects the performance of the whole organism or reduces an organisms ability to respond to environmental challenges" (i.e., the actual regulatory language in Section 69401.2 of the Proposed rule) is the same as the 2007 National Research Council report's definition that biological "perturbations are sufficiently large or when the host is unable to adapt …and this leads to toxicity and disease."[61] The Initial Statement also cites the American Society for Veterinary Clinical Pathology a definition of adverse that is limited to a biochemical change "that either singly and/or in combination adversely affects the performance of the organism as a whole or reduces the organism's ability to respond to an additional environmental challenge."[62]

The plain meaning of the Hazard Trait proposed regulatory language, however, is clearly much more open-ended than the NRC and the American Society for Veterinary Clinical Pathology definition (since in each case the perturbations must cause an adverse effect).

Finally, nothing in the regulations (or the Initial Statement) proscribes how DTSC will apply its discretion. If the intent was to limit adverse effects to perturbations that "would lead to toxicity and disease," then the definition in the proposed rule must be changed to reflect that intent.

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[61] Id. at 8 of 121.

[62] Id at 8 of 121, citing L. Boone, D. Meyer, P. Cusick, D. Ennulat, A. Provencher Bolliger, N. Everds, V. Meador, G. Elliott, D. Honor, D. Bounous, H. Jordan, for the Regulatory Affairs Committee of the American Society for Veterinary Clinical Pathology, Position Paper, Selection and Interpretation of clinical pathology indicators of hepatic injury in preclinical studies, Vet Clin Pathol. 2005;34:182–188.

F. Ecological Effects Should Not Include Any Effect on an Individual Organism

The proposed rule inappropriately equates adverse health effects on humans and ecological effects.[63] However, the ecological risk assessment work is much less well-developed than the risk assessment framework for human health risks.

The breadth of the change is further highlighted by DTSC's expansion of ecological adverse effects from population affects to include any effect on any individual organism.[64] Given the genetic diversity in any species, this expands adverse effects to any biological effect in any individual organism.

G. The Decision To Include All Microorganisms In the Definition of Wildlife Is Extreme and May Result in High Costs to Protect Microorganisms That Have Little Inherent Societal Benefit

The proposed rule defines wildlife to include all microorganisms.[65] While there may be some microorganisms which warrant inclusion within the scope of the proposed rule, the use of all microorganisms is excessive. RMA recommends that the final regulation should be limited to microorganisms of significance to protection of human health and ecological communities.

H. DTSC Cannot Rely On Authoritative Bodies Without Developing Its Own Administrative Record and Making Its Own Independent Scientific Determination

The proposed hazard traits rule defines "authoritative organization" broadly, including "other organizations that provide expert evaluations of chemical hazards."[66] The Initial Statement directs that EPA's, IARC's, and the NTP's "finding would take precedence over a third party's analysis use the same criteria since the agency is authoritative for its own determinations."[67]

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[63] The definition of environmental endpoints "parallels that for 'toxicological endpoint.'" Id at 10 of 121.

[64] Id at p. 8 of 121.

[65] Subsection 69401.2(j). *See* Initial Statement, *supra* note, at 19 of 121.

[66] Id at 9 of 121.

[67] Id at 25 of 121.

Deference by the courts to an agency's own findings is common, but even an agency's application of its own general applicable rules and criteria are challengeable, precisely because an agency may attempt to ignore or improperly apply its own rules and criteria in a specific case to ensure a favorable outcome. Thus, it is clearly wrong to claim that a California court interpreting a California rule should give absolute deference to EPA, IARC, the NTP or any other authoritative body.

There are serious issues raised by automatically adopting a hazard determination made by authoritative organizations because: (a) some of these authoritative organizations have no legal jurisdiction in California; (b) each authoritative body uses different classification criteria; and (c) these authoritative bodies (particularly those outside the United States) often do not follow the same rules of transparency and due process that apply in the United States. This makes it difficult, if not impossible, for companies (particularly small businesses) to exercise their due process right to comment on such authoritative organizations' findings and decisions. California cannot ignore the long-standing legal requirements of state rulemaking simply because the World Health Organization, European governments or other international bodies issue a hazard recommendation (often in a different legal context). The hazard assessment criteria process laid out in the proposed rule seems to be inconsistent with the broad principles articulated in many independent expert reviews. Neither the Initial Statement of Reasons or the proposed rule articulate a justification for the differences or a rationalization of these disparate criteria.

RMA recommends that California independently review any such determination, develop a proposed rule, subject that rule to public comment and actively issue a final rule that is consistent with California administrative law.

I. <u>What Happens When Authoritative Bodies Disagree</u>

Precisely because the various authorities utilize different classification criteria, follow different processes, and have different purposes underlying their decision making, authoritative bodies do and will continue to disagree. Nothing in the proposed rule addresses how DTSC should address such disagreements. At the very least, a disagreement must eliminate the reliance on such authoritative bodies.

This level of evidence is minimal and likely to result in misclassifications. Additionally, RMA recommends that this model must be subject to its own public comment period and a separate expert review for its use in a binding regulation.

J. The Proposed Rule Cannot Incorporate Future Changes in Existing Classification Criteria

The Initial Statement states that criteria for classification of a human reproductive toxicant is in "chapter 3.7 Reproductive Toxicity of the 2009 Third Edition of the Global Harmonized System" and "[a]s with other documents cited in this proposed regulation, the current version of the document should be used."[68] Such an approach is illegal because it delegates to the United Nations the legal authority to specify the criteria for human reproductive toxicants.

K. The Hazard Trait Determination Process Must Incorporate Peer Review In the Appropriate Circumstances

As drafted, the proposed rule provides little check or independent review of DTSC decisions, decisions which could ban the sale of products containing levels of a substance that contain a chemical that presents little or no hazard. Peer review is widely used at environmental agencies and involves solicitation of "advice on the reasonableness of judgments made from the scientific evidence," but not advice on policy.[69] Although the risk assessments performed in the overarching SCPA proposed rule process are subject to peer review, nothing in the hazard trait proposed rule provides for scientific peer review of chemicals that are reviewed to determine whether they present a hazard trait. Thus, RMA recommends that DTSC incorporate peer review in the hazard trait process.[70]

L. The Failure to Use the Hazard Assessment Process To Screen Chemicals Will Result in Chemicals With Weak Evidence of Risk Being Assigned Higher Risks Than Chemicals with Strong Evidence

Each individual change in the traditional risk/hazard assessment framework is dramatic, but the cumulative impact renders the proposed rule as useful as a screening tool. The proposed

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[68] Id at 34 of 121.

[69] Office of Management and Budget, Final Information Quality Bulletin for Peer Review, 70 Fed. Reg. 2,664, 2,669(January 14, 2005). "Many governments have in place a number of procedures, such as peer review, data quality and scientific guidance, which is designed to ensure the scientific integrity of work products produced by them for regulatory and public health purposes." Initial Statement, *supra* note 1, at 26 of 121.

[70] The fact that some of the hazard determinations by authoritative bodies involved peer review is not sufficient to provide peer review in the SCPA process. First, the nature of the peer review of these other bodies may not have been sufficient. Second, the use of the hazard determination of these other bodies may not have been the same as in the SCPA proposed rule (i.e., a determination whether a product can be sold at all).

rule will open the floodgate to chemicals and products subject to prioritization and substitute assessment. The fact that the screening bar has been lowered significantly means that chemicals with weak (or minimal) evidence of a hazard will proceed to a quantitative risk assessment. However, the weakness of the evidence used to conclude that a chemical has a hazard trait typically does not directly result in a less-stringent toxicity factor in the risk assessment. In fact, precisely because a chemical has less data and the data is weaker means that large uncertainty and safety factors are likely to be applied. Ironically, a chemical with very strong evidence of a risk may end up with lower cancer potency or a higher reference dose than a chemical with little evidence of risk.

The end result is likely to be that the risk prioritization performed using the framework articulated in the proposed rule may place a higher priority on the chemical least likely to present a significant risk.

M. Taken As A Whole The Proposed Rule Provides Little Objective, Science-Based Direction To the Staff of DTSC or the Courts

As illustrated above, the proposed rule adopts the least evidentiary burden at each stage of the hazard trait decision-making process. The cumulative impact of these shifts in the burden of proof result in a process that lacks objective, science-based decision-making criteria. In fact, the sum of all burden shifting aspects of the proposed rule make the rule as a whole less supportable than the sum of its parts. The basic foundation of due process is that there are clear rules that can be understood by the regulated community and can be independently reviewed by the courts. To the contrary, the criteria provided in the proposed rule provide no meaningful objective, science-based guidance. Rather, virtually any decision can be "justified" by citation to the broad criteria listed in the rule. As a result, this rule is incapable of being reviewed by a court and fails to meet minimum due process protections.

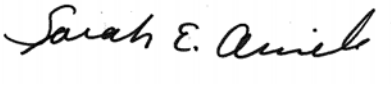
II. CONCLUSION

Regretfully, the framework provided in the proposed rule is likely to designate a large number of substances as having a hazard trait when in reality the substance have little or no risk when the product is in use. The framework is so radically different from the existing risk-reduction framework that it is likely to result in gridlock, undermine the public's faith in the regulatory process, and misdirect regulatory priorities. RMA strongly recommends that OEHHA

revise the majority of this proposed rule and present a hazard assessment framework that has practicable application. Major revisions to this proposed rule, followed by a repromulgation of the proposed rule, are necessary.

RMA again thanks the California Office of Environmental Health Hazard Assessment for the opportunity to comment on the proposed regulation. Please contact me at (202) 682-4836 if you have questions or require additional information.

Respectfully Submitted,



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