Responses to Major Comments on Technical Support Document

Public Health Goal
For
Trichloroethylene
In Drinking Water

Prepared by

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INTRODUCTION

The following are responses to major comments received by the Office of Environmental Health Hazard Assessment (OEHHA) on the proposed public health goal (PHG) technical support document for trichloroethylene, based on the first draft released for public comment on July 25, 2008. Changes were made in response to these comments, and were incorporated into the version of the document that was posted on the OEHHA Web site February 6, 2009 for a second comment period. No comments were received on the second draft; however, some minor additional changes were made to the PHG document for the final posting. Comments appear in quotation marks where they are directly quoted from the submission; paraphrased comments are in italics.

These comments and responses are provided in the spirit of the open dialogue among scientists that is part of the process under Health and Safety Code (HSC) Section 57003. Development of PHGs for regulated chemicals in drinking water is mandated under HSC 116365. For further information about the PHG process or to obtain copies of PHG documents, visit the OEHHA Web site at www.oehha.ca.gov. OEHHA may also be contacted at:

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RESPONSES TO MAJOR COMMENTS RECEIVED

Comments from the Aerospace Industries Association

Comment 1: "OEHHA's suggestion that ambient air and food are significant sources of exposure to TCE is not supported by any data or citations. While such sources may be theoretically possible, current restrictions on emissions of volatile chemicals like TCE from industrial sites reduce the likely contribution of ambient air as a significant pathway, and there is little evidence for significant contamination of food with TCE (ATSDR, 1998). OEHHA scientists have themselves noted that the '(d)efault of 0.2 tends to be overused, and may be over-protective' (Howd *et al*., 2004). Furthermore, OEHHA does not explain the basis for its statement that 'it is judged that the data are inadequate for a comprehensive evaluation.' If this is based on a review of a detailed literature search, OEHHA should describe that; if not, OEHHA should perform such a review to provide a valid basis for its choice of RSC."

We urge OEHHA to further evaluate information on sources of exposure of the general population to TCE to better define a realistic RSC value."

Response 1: The RSC value builds a measure of conservatism into the calculation of the public-health protective concentration in order to capture various possible exposure scenarios. We appreciate and consider that ambient air exposure may be reduced in the general and worker populations due to restrictions on emissions of TCE from industrial sites, but our literature search did not reveal adequate data to establish an alternative RSC. We agree that the default value of 0.2 for proportion of the total TCE exposure from drinking water could be an underestimate. However, it appeared to us that further efforts to calculate a more precise RSC are not justifiable because the PHG calculation is based on carcinogenicity, which does not incorporate the RSC. In general, conservative estimates and allowances for uncertainties are important to protect sensitive populations like infants, children, the elderly, and other possible sensitive or highly-exposed subpopulations.

Comment 2: "In deriving a 'public-health protective concentration' of TCE in water, OEHHA assumes an estimated equivalent water consumption of 7.1 L/day." *The equivalent water intake value appears to be an unrealistic value and significantly greater than the usual default water consumption value of 2 L/day used by EPA for deriving ambient water quality criteria and MCLs. Other estimates of TCE water consumption (references provided) indicate* "a more reasonable value for this term might b(e) around 4 or 5 Liter/day. We urge OEHHA to further evaluate all relevant information on equivalent water consumption for TCE to better define a more realistic value."

Response 2: All the relevant information was considered in determining the equivalent water consumption for TCE. As discussed in the PHG document, equivalent water consumption values that were considered ranged from 2 to 18.3 L/day. OEHHA believes that the multi-route tap water intake value of 7.1 L/day provides the most suitable

estimate for the calculation of the public health-protective concentration. Again, the calculation includes various uncertainties as well as the distributions of exposures in order to protect sensitive populations such as infants, children, the elderly, and other possible sensitive or highly exposed subpopulations.

Comment 3: "In deriving its proposed PHG, OEHHA selected a linear extrapolation default approach 'because the nonlinear MOA is insufficiently supported by the data.' Although OEHHA includes some reference to the recent NAS (2006) review of TCE, it does not appear to have adopted the NAS opinion regarding the mode of action of TCE in causing mouse liver tumors, and their implications for human health risk. NAS (2006) extensively discusses the available information on the carcinogenicity of TCE and concludes that the liver tumors seen in mice are most likely caused by a mode of action involving the PPAR-alpha receptor, and/or a tumor promotion mechanism, both of which would be expected to show a threshold, and may not even be relevant to humans exposed to low doses of TCE. … Even if OEHHA ultimately chooses to use the linear model, it would seem important to characterize the uncertainty in this choice…. We urge OEHHA to develop this type of discussion of scientific uncertainty, and to use it to document the high degree of caution associated with the choice of the linear model."

Response 3: The approach used by OEHHA is, we believe, the most appropriate approach given the current state of the science related to an understanding of the mechanisms of TCE toxicity. The NAS review provides an excellent assessment of TCE toxicity and recommendations for addressing the shortcomings. As part of the assessment, the NAS review discusses several studies on differences among species and the uncertainty of assumptions on the relationship between TCE and kidney and liver carcinogenesis in humans, based on the animal data. TCE was noted as being a complete kidney carcinogen in animals, where "(t)he committee ruled out the accumulation of $\alpha_{2\mu}$-globulin, peroxisome-proliferator activated receptor α (PPARα) agonism, and formic acid production as modes of action for the production of renal tumors in rodents." For liver tumors, the committee did conclude that the mode of action of TCE and its metabolites in liver carcinogenesis in animals is "principally as a liver peroxisome proliferator and agonist of PPARα rather than as a genotoxicant." However, the NAS review goes on to state that "species differences in susceptibility and phenotypic differences in tumors derived from trichloroethylene and its metabolites suggest that there are mechanistic differences in the way these chemicals cause tumors that cannot be fully explained by peroxisome proliferation."

OEHHA is concerned that recent studies on peroxisomal proliferators indicate that many toxic effects on liver occur in addition to and unrelated to peroxisomal proliferation. Given the evidence for a genotoxic mechanism of carcinogenicity in kidney as well as some uncertainty considering the liver tumors, OEHHA considers linear extrapolation from the liver tumor data as the better approach at this time. Discussion of the uncertainty in this approach is discussed in the Risk Characterization section of the document.