FINAL STATEMENT OF REASONS
TITLE 27, CALIFORNIA CODE OF REGULATIONS

SECTION 25705(b) SPECIFIC REGULATORY LEVELS POSING NO SIGNIFICANT RISK

NO SIGNIFICANT RISK LEVEL: DICHLOROACETIC ACID

This is the Final Statement of Reasons (FSOR) for the adoption of a No Significant Risk Level (NSRL) for dichloroacetic acid. On May 1, 1996, dichloroacetic acid was listed for purposes of Proposition 65[1] as a chemical known to the state to cause cancer. On May 22, 2020, the Office of Environmental Health Hazard Assessment (OEHHA) issued a Notice of Proposed Rulemaking to adopt a proposed amendment to Title 27, California Code of Regulations, section 25705(b)[2], Specific Regulatory Levels Posing No Significant Risk, identifying an NSRL of 17 micrograms per day (µg/day) for dichloroacetic acid. The Initial Statement of Reasons (ISOR) sets forth the grounds for the amendment to the regulation.

SUMMARY

In developing the NSRL for dichloroacetic acid, OEHHA relied on a study by DeAngelo et al. (1999)[3,4], a study by Bull et al. (2002)[5], Volume 106 in the series of International Agency for Research on Cancer (IARC) Monographs on the Evaluation of Carcinogenic Risks to Humans, entitled “Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents”[6], the National Toxicology Program (NTP) report entitled “Toxicology Studies of Bromodichloroacetic Acid (CAS No. 71133-14-7) in F344/N Rats and B6C3F1/N Mice and Toxicology and Carcinogenesis Studies of Bromodichloroacetic Acid in F344/NTac Rats and B6C3F1/N Mice (Drinking Water

[1] The Safe Drinking Water and Toxic Enforcement Act of 1986, codified at Health and Safety Code section 25249.5 *et. seq*., hereafter referred to as “Proposition 65” or “The Act”.
[2] All further regulatory references are to sections of Title 27 of the Cal. Code of Regs., unless otherwise indicated.
[3] DeAngelo AB, George MH, House DE (1999). Hepatocarcinogenicity in the male B6C3F1 mouse following a lifetime exposure to dichloroacetic acid in the drinking water: Dose-response determination and modes of action. J Toxicol Environ Health A 58(8):485-507.
[4] Individual animal survival and tumor data provided by Dr. DeAngelo, December 2007.
[5] Bull RJ, Orner GA, Cheng RS, Stillwell L, Stauber AJ, Sasser LB, Lingohr MK, Thrall BD (2002). Contribution of dichloroacetate and trichloroacetate to liver tumor induction in mice by trichloroethylene. Toxicol Appl Pharmacol 182(1):55-65.
[6] International Agency for Research on Cancer (IARC 2014). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 106, Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. IARC, World Health Organization, Lyon, France. Available from: https://publications.iarc.fr/130

Studies)"[7], and additional genotoxicity studies[8,9,10,11,12,13,14]. The NSRL for dichloroacetic acid is based upon the results of the most sensitive scientific study deemed to be of sufficient quality[15].

PEER REVIEW

OEHHA provided the Notice of Proposed Rulemaking and the Initial Statement of Reasons for the proposed NSRL for dichloroacetic acid to the members of the Carcinogen Identification Committee for their review and comment, as required by Section 25701(e). OEHHA received peer-review comments from committee members Jason Bush, Ph.D, Dana Loomis, Ph.D, MPH, Thomas Mack, MD, MPH, and Luoping Zhang, Ph.D.

RESPONSE TO PEER REVIEW COMMENTS

Comment 1: Drs. Loomis, Mack, and Zhang peer reviewed the materials, and indicated that they did not have any comments.

Response 1: OEHHA acknowledges the responses.

Comment 2: Dr. Bush indicated that he supports the rationale for the proposed NSRL for dichloroacetic acid, and concurs with the calculations and the proposed NSRL.

Response 2: OEHHA acknowledges the comments in support of the proposed NSRL.

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[7] National Toxicology Program (NTP 2015). Toxicology Studies of Bromodichloroacetic Acid (CAS No. 71133-14-7) in F344/N Rats and B6C3F1/N Mice and Toxicology and Carcinogenesis Studies of Bromodichloroacetic Acid in F344/NTac Rats and B6C3F1/N Mice (Drinking Water Studies). NTP Technical Report Series No. 583. US Department of Health and Human Services, NTP, Research Triangle Park, NC.

[8] Zhang SH, Miao DY, Tan L, Liu AL, Lu WQ (2016). Comparative cytotoxic and genotoxic potential of 13 drinking water disinfection by-products using a microplate-based cytotoxicity assay and a developed SOS/umu assay. Mutagenesis. 31(1):35-41.

[9] Hu Y, Tan L, Zhang SH, Zuo YT, Han X, Liu N, Lu WQ, Liu AL (2017). Detection of genotoxic effects of drinking water disinfection by-products using Vicia faba bioassay. Environ Sci Pollut Res Int. 2016 Oct 26.

[10] Varshney M, Chandra A, Chauhan LK, Goel SK (2013). Micronucleus induction by oxidative metabolites of trichloroethylene in cultured human peripheral blood lymphocytes: a comparative genotoxicity study. Environ Sci Pollut Res Int. 20 (12): 8709-16.

[11] Hassoun E, Cearfoss J, Mamada S, Al-Hassan N, Brown M, Heimberger K, Liu MC (2014). The effects of mixtures of dichloroacetate and trichloroacetate on induction of oxidative stress in livers of mice after subchronic exposure. J Toxicol Environ Health A. 77(6):313-23.

[12] Ono Y, Somiya I, Kawamura M (1991). The evaluation of genotoxicity using DNA repairing test for chemicals produced in chlorination and ozonation processes. Water Science and technology 23(1-3): 329-338.

[13] Stalter D, O'Malley E, von Gunten U, Escher BI. (2016). Fingerprinting the reactive toxicity pathways of 50 drinking water disinfection by-products. Water Res 91: 19-30.

[14] Hassoun EA, Dey S. (2008). Dichloroacetate- and trichloroacetate-induced phagocytic activation and production of oxidative stress in the hepatic tissues of mice after acute exposure. J Biochem Mol Toxicol 22(1): 27-34.

[15] Section 25703(a)(4)

PUBLIC COMMENTS

A public comment period was provided from May 22, 2020 to July 7, 2020. OEHHA received written public comments on the proposed rulemaking from the following organizations:

1. Southern California Water Coalition (SCWC)
2. American Chemistry Council's (ACC) Chlorine Chemistry Division (CCD)

RESPONSE TO PUBLIC COMMENTS

A summary of the relevant public comments received and OEHHA's responses are provided in this FSOR. Some of the comments submitted included observations or opinions regarding the benefits of chlorine-based disinfection processes and other assessments OEHHA might perform on dichloroacetic acid and other disinfection by-products. Such remarks do not constitute an objection to or recommendation specifically directed at the proposed action or the procedures followed in this rulemaking action. Accordingly, OEHHA is not required under the Administrative Procedure Act to respond to such comments in this FSOR. Because OEHHA is constrained by limitations upon its time and resources and is not obligated by law to respond to irrelevant comments[16], OEHHA does not provide responses to all of these remarks in this FSOR. However, the absence of responses to such remarks should not be construed to mean that OEHHA in any way agrees with them.

As explained in detail in the responses to comments, OEHHA declines to change the proposed NSRL based on the comments.

Comment 1 (SCWC, ACC): NSRLs should not be based on draft risk assessments still under development in other programs. CCD [ACC's Chlorine Chemistry Division] is troubled by OEHHA's decision to move ahead with NSRLs before the Office has considered the information submitted in response to the PHG [Public Health Goal] proposal and before the science that is the basis for both the PHGs and NSRLs has been subject to peer review. The NSRL should not be released until the process for the PHG for haloacetic acids (HAAs) has been completed. It is premature and inappropriate for OEHHA to use draft PHG risk assessments to support Proposition 65 NSRLs or any other regulatory decisions until those draft risk assessments are completed. SCWC is concerned that using the draft PHG risk assessments as the basis for enforceable NSRLs would undermine the PHG development process because the proposed NSRLs would create an institutional bias against meaningful changes to the draft PHG risk assessments.

Response 1: The NSRL does not rely on the draft Public Health Goal (PHG), which was developed in parallel with the NSRL. This process allows for adequate time for the

[16] California Government Code section 11346.9(a)(3)

NSRL and the PHG to undergo external peer review and encourages consistency between the two programs within OEHHA. The process for the dose-response assessment and development of the NSRL for dichloroacetic acid was conducted in collaboration with the OEHHA program that produces PHGs. Both programs critically evaluated the same key rodent carcinogenicity studies of dichloroacetic acid (DeAngelo et al. 1999[17,18], Bull et al. 2002[19]) and used the same data analysis principles, methods, and software to calculate the cancer potencies. After careful consideration by both programs, the male mouse study by DeAngelo et al. (1999) was chosen for assessing the carcinogenic effects of dichlooacetic acid, and thus, the human cancer slope factor derived from that study was used as the basis for both the NSRL and the PHG. An assessment by one OEHHA program does not preclude another OEHHA program from making changes to a draft document. The proposed levels for both programs are based on the best available science and have undergone rigorous scientific review.

No changes to the proposed regulation were made based on this comment.

Comment 2 (SCWC, ACC): There is no justification for proposing the NSRLs at this time. These chemicals were listed several years ago, yet OEHHA saw no need to develop the NSRL until now. Dichloroacetic acid (DCA) appears to be limited only to narrow consumer product applications. However, there is nothing in the ISOR indicating an increase in consumer product uses or other applications that would justify the development of an NSRL at this time.

Response 2: OEHHA develops NSRLs for chemicals listed as carcinogens under Proposition 65 as time and resources allow. There are no limits on the time between the date of listing and the development of an NSRL. In recent years, multiple HAAs have been added to the Proposition 65 list and OEHHA has developed NSRLs for each of the five HAAs listed (trichloroacetic acid, dibromoacetic acid, dichloroacetic acid, bromochloroacetic acid, and bromodichloroacetic acid) in order to provide compliance assistance for businesses and guidance for Proposition 65 enforcers.

No changes to the proposed regulation were made based on this comment.

Comment 3 (SCWC): These NSRLs present a potential public health threat because they prioritize reduction of exposure to disinfection by-products (DBPs) over drinking water disinfection. OEHHA should establish alternative Safe Harbor Levels pursuant to Section 25703(b) that allows for such exceptions to the default NSRL.

Response 3: OEHHA followed the guidance in Section 25703(b), which states that “the risk level which represents no significant risk shall be one which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime

[17] DeAngelo et al. (1999). Full citation provided in footnote 3.
[18] Individual animal survival and tumor data provided by Dr. DeAngelo, December 2007.
[19] Bull et al. (2002). Full citation provided in footnote 5.

exposure at the level in question, except where sound considerations of public health support an alternate risk level", and gives as one such example "where chlorine disinfection in compliance with all applicable state and federal safety standards is necessary to comply with sanitation requirements".

In developing the NSRL for this carcinogen, OEHHA conducted the evaluation necessary to identify a level that would meet the 1 in 100,000 standard. OEHHA recognizes the public health benefits of the use of chlorine disinfection, and at the same time notes that nothing in Proposition 65 prohibits or places limits on drinking water disinfection. In fact, the statute[20] expressly exempts all agencies of the federal, state, or local government, as well as entities operating public water systems, from the requirements of Proposition 65, including the warning requirement.

Nothing in the analysis for the NSRL prohibits a business from calculating an alternative risk level for this chemical, should the business determine that one is needed.

No changes to the proposed regulation were made based on this comment.

Comment 4 (ACC): The NSRL does not consider the long history of low-level exposure to these substances (i.e., HAAs) and several other disinfection byproducts (DBPs) considered to be liver carcinogens by OEHHA (chloroform, bromodichloromethane, and dibromochloromethane). This history reveals a lack of consistent evidence of an increased incidence of liver cancer resulting from exposure to DBPs in the multiple epidemiology studies that have been conducted.

Response 4: The NSRL for dichloroacetic acid was based on a study conducted in mice because it was deemed to be a sensitive study of sufficient quality, consistent with the requirements described in Section 25703. To our knowledge, no human epidemiological studies of sufficient quality and sensitivity have been published in the scientific literature that would be adequate for conducting a cancer dose-response assessment for dichloroacetic acid. Thus, the DeAngelo et al. (1999) study in male mice in which liver tumors were observed was chosen as the most sensitive study of sufficient quality. Regarding the lack of consistent evidence of an increased incidence of liver cancer in humans, tumor site concordance across species is neither required, nor predicted, for chemical carcinogens. It is a generally accepted principle that although there may be site concordance between humans and animal test species in specific cases, this is not true in general. For risk assessment purposes, site concordance is not assumed unless there is evidence to support this assumption[21]. In the absence of data to the contrary, the ability of an agent to induce tumors in animals is considered predictive of the potential for the agent to induce tumors in humans.

[20] Health and Safety Code section 25249.11(b)
[21] OEHHA (2009). Technical Support Document for Cancer Potency Factors. Available from http://oehha.ca.gov/air/crnr/technical-support-document-cancer-potency-factors-2009

No changes to the proposed regulation were made based on this comment.

Comment 5 (SCWC, ACC): Dichloroacetic acid appears to be weakly genotoxic and only at higher doses, which may indicate a threshold cancer mechanism. As noted by the US EPA, there is little basis for judging whether genotoxic effects are important in the carcinogenic response, and if so, whether the dose-response curve for genotoxic effects is linear or nonlinear.

Response 5: Although the US EPA IRIS document[22] states that the genotoxicity data for dichloroacetic acid are inconsistent and seem to indicate that dichloroacetic acid is a weak mutagen, and that it induces mutations predominantly at higher concentrations, the US EPA concludes the following:

> "Nevertheless, in the absence of causal data, EPA considers it prudent to assume that DCA might be genotoxic, at least under in vivo exposure levels that are associated with detectable increases in tumor incidence (particularly at the higher doses). Whether DCA is genotoxic at lower doses (which would suggest a linear dose-response curve for cancer risk) is not known."[23].

US EPA considered the possibility of a threshold but determined there are not enough data to draw conclusions. In fact, US EPA specifically states that the data are inadequate to support any conclusive mode of action for the carcinogenicity of dichloroacetic acid [24]. The IRIS document describes several possible modes of action, including genotoxicity, hepatocytotoxicity and regenerative hyperplasia, promotion of spontaneous mutation, and depression of apoptosis. The mechanistic data suggest a complex etiology for tumor development; "[t]he data on mechanism implicate more than one type of cellular change in the origin of tumors along with defects in intra- and inter-cellular communication pathways"[25]. US EPA goes on to state, "[b]ecause the mode of action by which DCA increases cancer risk is not understood, extrapolation to low dose was performed by assuming a no-threshold linear dose-response curve between the origin and the POD [point of departure]"[26]. Thus, US EPA supports the use of a linear model because there is not enough evidence to support the use of a threshold approach.

As described in the ISOR, both IARC and NTP, in evaluations conducted subsequent to that of the US EPA, stated there is evidence that dichloroacetic acid is genotoxic. Moreover, the ISOR describes additional genotoxicity studies identified by OEHHA,

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[22] United States Environmental Protection Agency (US EPA 2003). Toxicological Review of Dichloroacetic Acid (CAS NO. 79-43-6) In Support of Summary Information on the Integrated Risk Information System (IRIS). US EPA, Washington, DC. Document number EPA 635/R-03/007. Available from https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?&substance_nmbr=654.
[23] *Ibid*.
[24] *Ibid.*
[25] *Ibid.*
[26] *Ibid.*

several of which were positive over a range of doses. Thus, the genotoxicity data do not support a threshold mechanism, and the default linearized multistage model is a scientifically appropriate method for calculating an NSRL for dichloroacetic acid.

No changes to the proposed regulation were made based on this comment.

Comment 6 (ACC): ACC quotes US EPA (2003)[27], which states that issues regarding the mechanism and shape of the dose-response curve are highlighted by comparing the concentrations of dichloroacetic acid in water that are carcinogenic in animals with those that are commonly observed in chlorinated drinking water. Thus, concentration values are about 4-5 orders of magnitude lower in drinking water than were used in experimental animals. This difference is further magnified by the lower water intake per unit body weight of humans.

Response 6: This comment addresses issues related to the characterization of the cancer risk from drinking water exposures to dichloroacetic acid (i.e., risk characterization); it is not relevant to the subject of this regulatory action, which is the development of the NSRL for dichloroacetic acid. Information on the current or anticipated levels of exposure to human populations via chlorinated drinking water has no bearing on the calculation of the NSRL. As shown in the ISOR, the derivation of the NSRL for dichloroacetic acid is based on the analysis of cancer dose-response data from studies conducted in mice. The NSRL is the level of exposure, expressed in micrograms per day (µg/day), which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime exposure at that level. If levels in drinking water are below the NSRL, a Proposition 65 warning is not required.

No changes to the proposed regulation were made based on this comment.

Comment 7 (SCWC): Dichloroacetic acid has been used therapeutically in humans at doses as high as 25 mg/kg-day.

Response 7: The commenter is correct in stating that dichloroacetic acid is used therapeutically in humans. Dichloroacetic acid is used as a cauterizing agent and medical disinfectant, and dichloroacetic acid and its salts have been used in the treatment of congenital lactic acidosis and have been proposed for use in treating diabetes and cancer. Dichloroacetic acid and its salts are not commonly used due to side effects[28]. It is important to note that the approval of a drug for use as medication with a specific indication does not imply that it cannot cause long-term toxicological effects, such as cancer. In fact, there are a number of drugs that are carcinogenic but are also the best or only available option for treatment. For example, chloramphenicol sodium succinate is used as a broad-spectrum antibiotic for serious infections, and has been identified as a carcinogen by the Food and Drug Administration and is listed under

[27] US EPA (2003). Full citation provided in footnote 22.
[28] Summarized by IARC (2014), p. 355. Full citation provided in footnote 6.

Proposition 65[29]. There are a number of other chemicals used as medications that are listed as carcinogens under Proposition 65.

No changes to the proposed regulation were made based on this comment.

Comment 8 (ACC): The NSRL for dichloroacetic acid is based on reports of liver tumors in studies conducted in male mice. However, the evidence in female mice is less consistent, and studies in rats suggest lower sensitivity than in mice.

Response 8: As described in Section 25703, the NSRL should be based on the most sensitive study of sufficient quality. As stated in the ISOR, OEHHA reviewed the available data from the rodent carcinogenicity studies of dichloroacetic acid and determined that the studies in male mice by DeAngelo et al. (1999) and Bull et al. (2002) were sensitive studies of sufficient quality. Of these two studies, the one by DeAngelo et al (1999) was determined to be the most sensitive study, and thus, the NSRL for dichloroacetic acid was based on the DeAngelo et al (1999) study conducted in male mice. The fact that the available rodent carcinogenicity studies indicate that rats and female mice are less sensitive than male mice to dichloroacetic acid carcinogenicity does not detract from the strong findings of carcinogenicity observed in male mice.

No changes to the proposed regulation were made based on this comment.

Comment 9 (SCWC, ACC): The commenters believe that DeAngelo *et al.* is not an appropriate study for quantitative health risk assessment and deriving a cancer slope factor. Both commenters point out that the mice in the key study selected by OEHHA for the dichloroacetic acid risk assessment (DeAngelo et al. 1999) exhibited a high rate of spontaneous liver tumors and ACC mentions that there was significant mortality and body weight decreases at the two highest doses.

Response 9: The incidence of liver tumors in the control group of the $B6C3F_1$ mouse study by DeAngelo et al. (1999) does not diminish the significance of the findings in the dose groups. Hepatocellular adenomas and carcinomas are commonly observed in male $B6C3F_1$ mice. For example, NTP historical control data[30] reports incidences of 208/339 (61.4%) for adenomas and 95/339 (28%) for carcinomas in NTP drinking water studies conducted in male $B6C3F_1$ mice from 1984 to 1994[31]. Thus, an incidence of 20/70 (28.6%) hepatocellular adenoma or carcinoma combined in the control animals in the DeAngelo et al. (1999) male $B6C3F_1$ mouse study is not unusual.

[29] https://oehha.ca.gov/proposition-65/crnr/chemical-listed-effective-september-27-2013-known-state-california-cause-cancer

[30] NTP (1999). National Toxicology Program Historical Controls. US Department of Health and Human Services. Available from https://ntp.niehs.nih.gov/ntp/research/database_searches/historical_controls/path/m_orlwr.txt

[31] DeAngelo et al. (1999) was conducted from 1990 to 1992. Control data should be compared to historical control data that are gathered within 2 or 3 years one way or the other of the study under review.

Regardless of the incidence found in the control group, there was a significant increase in hepatocellular adenoma or carcinoma incidence in male mice in the study by DeAngelo et al. (1999) by pairwise comparison with controls and by exact trend test, as shown in Table 1 of the ISOR. Bull et al. (2002) also demonstrated significant increases in the incidences of hepatocellular adenoma or carcinoma by pairwise comparison with controls and by exact trend test. Thus, it has been shown through multiple studies that dichloroacetic acid induces liver tumors in mice. The DeAngelo et al. (1999) study was chosen as the basis for the NSRL because it was of longer duration and had more animals per treatment group than Bull et al. (2002).

The ISOR discusses the survival issues observed in the DeAngelo et al. (1999) study, noting that survival was significantly decreased in the two highest dose groups compared to controls, with a significant trend, and that the majority of early deaths were due to liver tumors. Thus, the survival issue should not be considered as a limitation of the study and is not a reason to consider the study inappropriate for dose-response assessment. Regarding the body weight decreases, DeAngelo et al. (1999) concluded that, based on the water consumption and body weight gain through 78 weeks of treatment, the drinking water concentration of 2 g/L dichloroacetic acid did not exceed the maximum tolerated dose.

Therefore, DeAngelo et al. (1999) was judged to be a sensitive study of sufficient quality. The study by Bull et al. (2002) was also considered for cancer dose-response assessment. The Bull et al. (2002) study was of shorter duration (52 weeks) and had fewer animals in each treatment group (20 mice/group), and was judged to be less robust than DeAngelo et al. (1999).

No changes to the proposed regulation were made based on this comment.

Comment 10 (ACC): The ISOR notes limitations for all of the cancer studies considered as candidates for deriving the proposed NSRL. In light of these limitations, OEHHA should use a geometric mean of the CSFs from the most relevant studies, rather than selecting the highest CSF among the male mouse studies.

Response 10: DeAngelo et al. (1999) and Bull et al. (2002) were chosen because they met the criterion in Section 25703 as being sensitive studies of sufficient quality. The main limitation of DeAngelo et al. (1999) was the reduced survival in treated animals. This was accounted for by using a multistage Weibull model. Thus, the DeAngelo et al. (1999) study did not have “major limitations” and provided an adequate dataset for dose-response assessment. As explained in the ISOR, DeAngelo et al. (1999) was judged to be more robust and to provide a better overall estimate of the cancer dose-response than Bull et al. (2002) because DeAngelo et al. (1999) had a longer study duration and more animals in each treatment group. Since Bull et al. (2002) terminated the study at 52 weeks, one year before the assumed rodent lifespan for carcinogenicity studies (104 weeks), the animal cancer slope factor would need to be adjusted by

assuming cancer risk increases with the third power of age. This extrapolation introduces additional uncertainty in the analysis, thus DeAngelo et al. (1999), which also had more animals in each treatment group, is preferred. It is more appropriate to choose a single well-conducted study than to combine it with a less appropriate study.

No changes to the proposed regulation were made based on this comment.

Alternatives Determination

In accordance with Government Code section 11346.9(a)(4), OEHHA has, throughout the adoption process of this regulation, considered available alternatives to determine whether any alternative would be more cost effective in carrying out the purpose for which the regulation was proposed, or would be as cost effective and less burdensome to affected private persons than the proposed action. No alternatives have been suggested. OEHHA has determined that no reasonable alternative would either be more effective in carrying out the purpose for which the action is proposed or would be as effective and less burdensome to affected private persons, or would be more cost-effective to affected private persons and equally effective in implementing the statutory policy or other provision of law than the proposed regulation.

For chemicals listed under the Act as known to cause cancer, the Act exempts discharges to sources of drinking water and exposures of people without provision of a warning if the exposure poses “no significant risk” of cancer (Health and Safety Code, section 25249.10(c)). The Act does not specify numerical levels of exposure that represent no significant risk of cancer.

The purpose of this regulation is to establish a No Significant Risk Level for dichloroacetic acid. At or below this level, the Act does not require a warning or prohibit discharges of the chemical to sources of drinking water. Thus, adopting this level will allow businesses subject to the Act to determine whether a given discharge to sources of drinking water or a given exposure to this chemical is subject to the warning requirement or discharge prohibition provisions of the Act (Health and Safety Code, section 25249.5 and 25249.6).

Although Section 25703 describes principles and assumptions for conducting risk assessments to derive No Significant Risk Levels, some businesses subject to the Act do not have the resources to perform these assessments. Yet each business with ten or more employees must determine whether its activities or products are subject to the discharge prohibition or warning requirements of the Act. Adopting an NSRL for this chemical provides an efficient way of determining if a business is in compliance with the Act.

Local Mandate Determination

OEHHA has determined this regulatory action will not pose a mandate on local agencies or school districts, nor does it require reimbursement by the State pursuant to Part 7 (commencing with Section 17500) of Division 4 of the Government Code. OEHHA has also determined that no nondiscretionary costs or savings to local agencies or school districts will result from this regulatory action. Proposition 65 provides an express exemption from the warning requirement and discharge prohibition for all state and local agencies. Thus, these regulations do not impose any mandate on local agencies or school districts.